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Placental and Fetal Endocrine Systems Depend on the Cause of Low Birth Weight

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Introduction: Low birth weight is caused by a huge number of different conditions ranging from constitutional shortness in a healthy newborn to severe fetal and maternal disease interfering with the growth of the fetus. In many studies examining the effect of low birth weight on later morbidity these different causes are not accounted for. It is therefore the objective of the present study to examine the impact of different causes of low birth weight in rats and humans.

Animal studies: Two rat models used for experimental research of IUGR were examined: The isocaloric low protein model and the bilateral uterine artery ligation model. The offspring was delivered at day 22 of gestation and placenta and liver were immediately snap frozen. Using real time PCR, the gene expression of leptin and IGF-I was examined in the two tissues. In placental tissue, there was an inverse pattern of leptin and IGF-I gene expression with an upregulation in the low protein model and a downregulation in the ligation model. In liver, IGF-I expression was decreased in the ligation but not in the low protein model.

Human studies: In a multicenter study placental tissue and umbilical blood of neonates with IUGR (birth weight < 10^{th} percentile and pathological placental Doppler velocimetry), SGA (birth weight < 10^{th} percentile, no pathological Doppler), and controls were obtained. Umbilical leptin concentration was similar in all groups. However, the concentration of the soluble leptin receptor, decreasing bioavailability of leptin, was significantly increased in IUGR infants only. For IGF-I, there was a decrease of cord blood concentration from control neonates via SGA to IUGR infants, while IGFBP-1 was not altered. Placental expression of leptin was increased in IUGR compared to AGA and SGA.

Conclusions: Studies in rats and humans indicate that the underlying cause of low birth weight appears to be essential for the availability of leptin and IGF-I. Since both hormones have been closely related to perinatal programming, it may be concluded that different causes of low birth weight lead to a distinct programming of the leptin and IGF-I pathways. As a consequence, various phenotypes of adult onset disease will have to be expected in low birth weight infants.

*** Nüsken E, Tzschoppe A, Dötsch J, Nüsken KD, Fetal programming of endocrine function in IUGR offspring depends on the cause of low birth weight: evidence from animal models and the human FIPS-study. In: Plagemann A, editor. Perinatal programming – The State of the Art. Berlin: Walter de Gruyter, 2011.**