The apelinergic system is implicated in fetoplacental development and is a target in developmental nutritional programming

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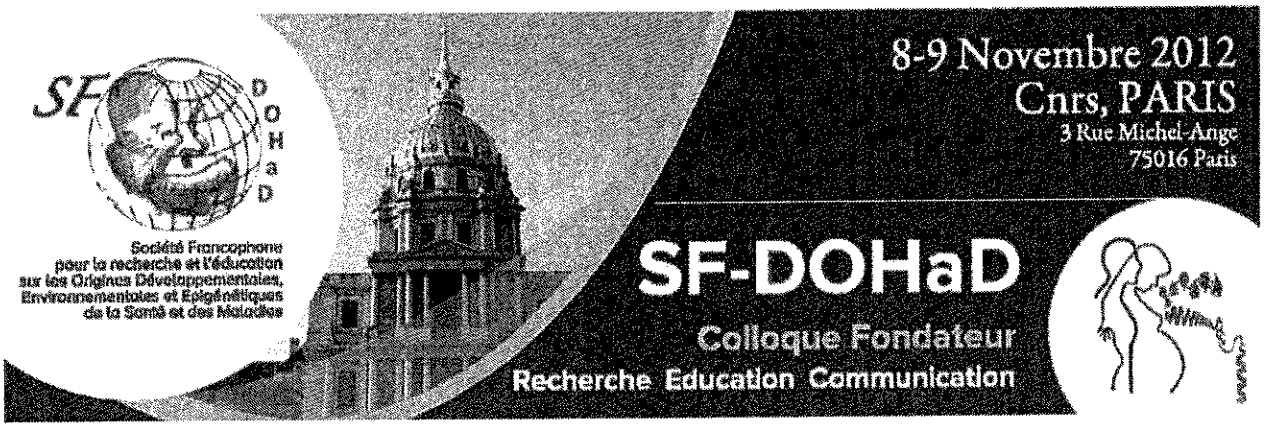

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(41 + 11 *v.* 61 + 13%/day, $P < 0.001$). Fetal muscle protein FSR was enhanced by CIT (56 + 4%/day), not with ARG or NEAA (45 + 7 and 50 + 19%/day, NS). We conclude that: (1) citrulline enhances fetal growth in a model of IUGR; and (2) such effect may be mediated by enhanced fetal muscle protein synthesis.

Key words: fetal growth, maternal diet, metabolism, placenta, stable isotopes

Statement of interest: The Authors declare no conflict of interest.

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POSTER N°35

The apelinergic system is implicated in fetoplacental development and is a target in developmental nutritional programming

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Apelin (APL) and its receptor APJ are expressed in numerous tissues in mammals.[1] APL is also present in the blood where its level is related to the nutritional status and is correlated to insulin plasma level.[2] Recent studies pointed out an emerging role of APL in cardiovascular regulations, energy metabolism and glucose homeostasis.[1,3] We investigated the gene expression profile of this system and circulating APL levels during the gestation in rat. Plasma APL level is reduced in mothers at day 7 of pregnancy (E7) and then augmented until E21. In fetuses, APL concentration decreases from E17 to E21 and is comparable, at term, to maternal level. High levels of APJ and APL mRNAs were found at the feto placental interface (i.e. the mesometrial triangle and the placenta) and exogenous maternal administration of APL increases both placental glucose uptake and transplacental transport of glucose. In addition, high APJ/APL mRNAs were detected in numerous fetal tissues at term. Maternal food-restriction (FR) modulates drastically APJ/APL mRNAs levels at the feto placental interface and reduces circulating APL levels in both mothers and in growth-restricted fetuses. In adult male rat offspring from FR mothers, an increase basal plasma APL level was found suggesting a prenatal nutritional programming of this new endocrine system. Altogether, our findings propose that the apelinergic system is implicated in the feto placental unit development and may be targeted by prenatal nutritional disturbances resulting to the programming of metabolic diseases.

Key words: developmental programming, fetal growth, pregnancy

Statement of interest: The authors declare no conflicts of interest.

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POSTER N°39

Nutritional epigenetics: considering maternal dietary and fetal programming

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Growing evidence suggests that epigenetic mechanisms of gene regulation, such as DNA methylation and chromatin modification, are influenced by the nutritional factors and play an important role in the fetal basis of adult disease susceptibility. Epigenetics refer to functionally relevant modifications to the genome that do not involve a change in the nucleotide sequence. The epigenetic marks get added to genes or chromosomal proteins during fetal life. These epigenetic marks become fixed after early life and may become a problem when environment or diet changes later in life. This is thought to be one of the underlying mechanisms of fetal programming and its consequences on disease risks in the adult are now well accepted by the scientific community. It is also probable that the first few years after birth are more likely to be influenced by dietary components nutrition. To address these essential issues, we investigate in the laboratory the influence of dietary components on epigenetic processes, including DNA methylation, histone modification and miRNA gene expression. In our studies, the potential *in vitro* effect of some dietary components, dietary quality (nutrients deficiency and the presence of contaminants) on DNA methylation and chromatin accessibility were carried out by using different epigenetic analysis methods and approaches.[1–4] Results of studies have shown that folate deficiency and fumonisin B1 decreased global genomic DNA