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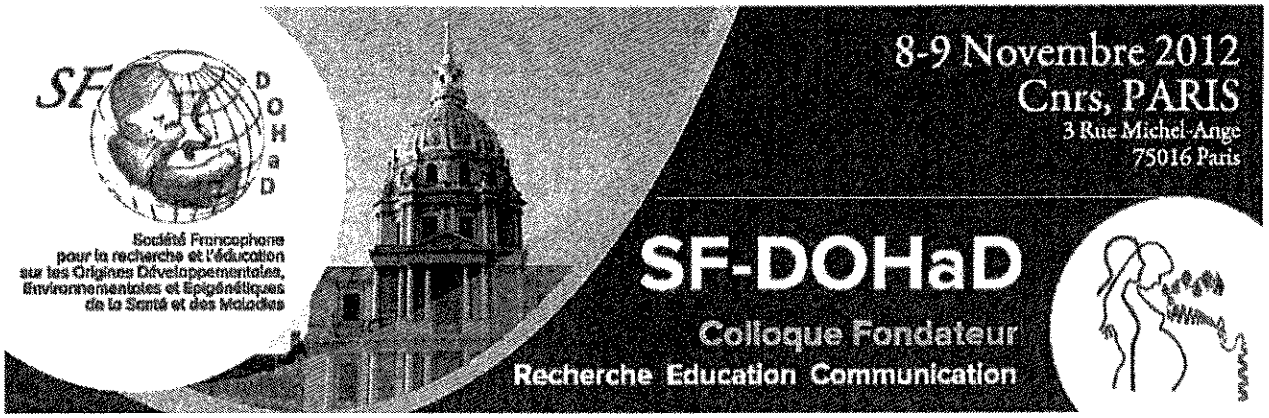

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now recognized as one of the factors contributing to the developmental programming of chronic diseases in later life.[2,3] To date, there is no relevant animal model that displays impaired glucose tolerance without diabetes during gestation to investigate the mechanisms underlying fetal overgrowth in pregnancy complicated with MGH. We have developed a rat model and investigated the effects of maternal dysglycemia on fetal growth and placental gene expression. Female rats were injected with nicotinamide and streptozotocin (N-STZ) 1 week before mating. N-STZ treatment induced impaired glucose tolerance in late gestation, resulting in metabolic disorders and fetal overgrowth in more than 20% of newborns. Placental mass was also increased in N-STZ macrosomic pups compared with normotrophes, and associated with a rise in the labyrinthine zone. Gene and protein expression of lipoprotein lipase was increased in N-STZ placentas from macrosomic pups. We reported that expression of insulin receptor and glucose transporters genes was down-regulated in macrosomic placentas, whereas the expression of amino acid transporters was not modified. For the first time, we showed that insulin-like growth factors and nutrient transporter genes were also differentially expressed in the placentas from normal pregnancies when the number of fetuses within the litter varies. The N-STZ model offers the potential for further studies into the effects of MGH on placental function that will allow better understanding of the mechanisms underlying fetal overgrowth. We propose that the regulation of placental gene expression constitutes a mechanism of physiological adaptation that is taking part during late gestation to optimize fetal growth and assure the viability at birth when the number or the size of fetuses is inappropriate.

Key words: animal, fetal growth, gestational diabetes, placenta

Statement of interest: Authors report no conflict of interest.

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POSTER N°37

Characterization of the placental development in the intrauterine growth-retarded piglet

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In pig production, the selection of hyperprolific animals has lead to an increased rate of intrauterine growth-retarded piglets in litters. Intrauterine growth retardation (IUGR) is an important economic issue inducing a higher mortality rate, a lower growth capacity, a higher muscular lipid content in carcasses and less tender meat. The maternal and fetal environments interact through the placenta to maintain nutrient supply of the fetus. The aim of this work was to explore placental adaptive mechanisms throughout pregnancy in IUGR piglets. Possible changes in placental morphometry and gene expression of the IUGR piglet were prospected, using a natural IUGR pig model. Placental samples from 18 pairs of control (normal birth weight) and IUGR fetuses at gestation days (gd) 45, 71 and 112 were analyzed by stereology. The expression of 10 candidate genes potentially associated with placental development and IUGR was examined by RT-qPCR. The DNA methylation of the insulin-like growth factor 2 (IGF2) imprinting gene was evaluated by pyrosequencing. Glycemia and fructosemia were measured in IUGR and control fetuses at gd 71 and 112. No morphometric abnormality was found in the IUGR placenta. An increased expression of the IGF2 gene, however, was observed in the IUGR chorionic tissue at gd 71 ($\times 1.48$, $P < 0.05$). No difference was shown, however, in the DNA methylation levels of the IGF2 gene. Fructosemia was significantly reduced in IUGR fetuses at gd 71, but not at gd 112, whereas glycemia remained normal at both stages. IGF2 affects the placental growth and the placental permeability by modulating the nutrient transport levels. This increase could be a compensatory mechanism from the placenta to meet the fetal nutrient demand and maintain fetal growth at mid-gestation. Indeed, there was a tendency for the expression of glucose transporter GLUT3 to be reduced at gd 71.

Key words: critical periods, fetal growth, large animals, placenta, pregnancy

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POSTER N°45

Maternal food-restriction leads to a drastic downregulation of H4K16 acetylation in IUGR rat placentas

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