Maternal protein restriction during lactation induces early and lasting plasma metabolomic and hepatic lipidomic signatures of the offspring in a rodent programming model

Titre	Maternal protein restriction during lactation induces early and lasting plasma metabolomic and hepatic lipidomic signatures of the offspring in a rodent programming model
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Résumé en anglais	Perinatal undernutrition affects not only fetal and neonatal growth but also adult health outcome, as suggested by the metabolic imprinting concept. However, the exact mechanisms underlying offspring metabolic adaptations are not yet fully understood. Specifically, it remains unclear whether the gestation or the lactation is the more vulnerable period to modify offspring metabolic flexibility. We investigated in a rodent model of intrauterine growth restriction (IUGR) induced by maternal protein restriction (R) during gestation which time window of maternal undernutrition (gestation, lactation or gestation-lactation) has more impact on the male offspring metabolomics phenotype. Plasma metabolome and hepatic lipidome of offspring were characterized through suckling period and at adulthood using liquid chromatography-high-resolution mass spectrometry. Multivariate analysis of these fingerprints highlighted a persistent metabolomics signature in rats suckled by R dams, with a clear-cut discrimination from offspring fed by control (C) dams. Pups submitted to a nutritional switch at birth presented a metabolomics signature clearly distinct from that of pups nursed by dams maintained on a consistent perinatal diet. Control rats suckled by R dams presented transiently higher branched-chain amino acid (BCAA) oxidation during lactation besides increased fatty acid (FA) β-oxidation, associated with preserved insulin sensitivity and lesser fat accretion that persisted throughout their life. In contrast, IUGR rats displayed permanently impaired β-oxidation, associated to increased glucose or BCAA oxidation at adulthood, depending on the fact that pups experienced slow postnatal or catch-up growth, as suckled by R or C dams, respectively. Taken together, these findings provide evidence for a significant contribution of the lactation period in metabolic programming.
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