# A short periconceptional exposure to maternal type-1 diabetes is sufficient to disrupt the feto-placental phenotype in a rabbit model

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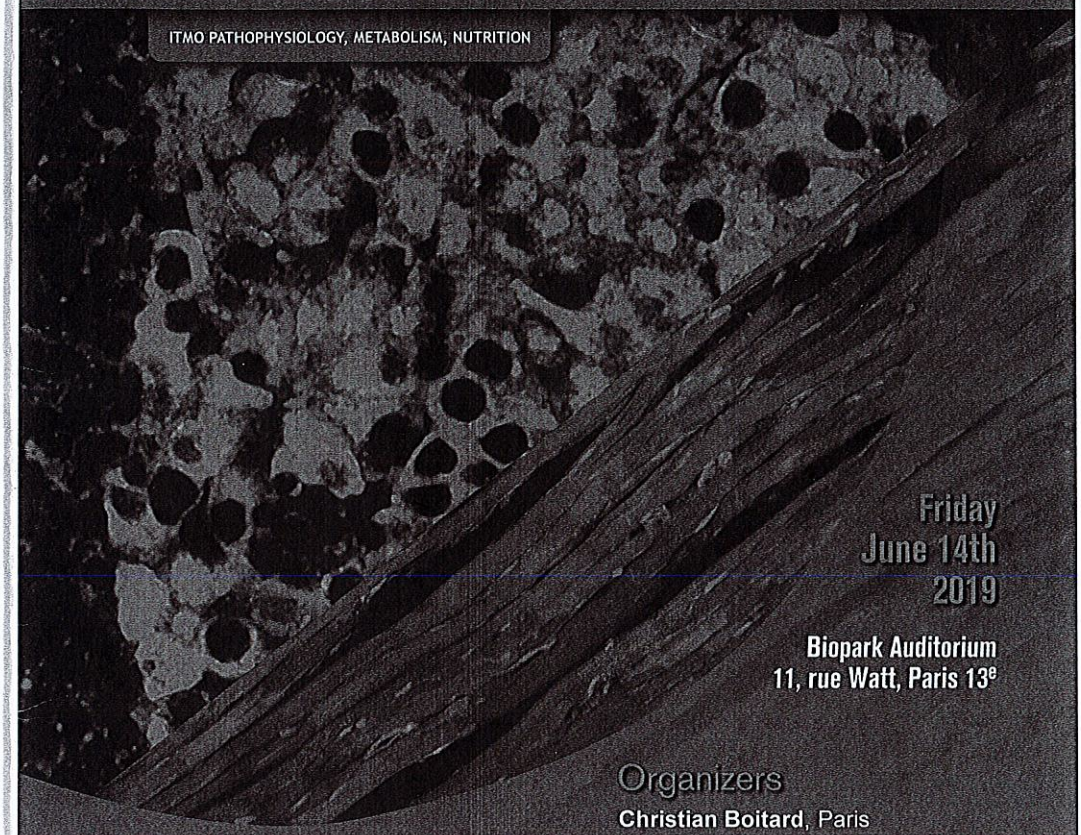
ITMO PATHOPHYSIOLOGY, METABOLISM, NUTRITION



Friday
June 14th
2019

**Biopark Auditorium**
**11, rue Watt, Paris 13e**

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# TRANSLATIONAL RESEARCH ON DIABETIC CARDIOMYOPATHY

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Translational research on Diabetic Cardiomyopathy – June 14th, 2019

# A short periconceptional exposure to maternal type-1 diabetes is sufficient to disrupt the feto-placental phenotype in a rabbit model

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Tight metabolic control of type-1 diabetes is essential during gestation, but it could be crucial during the periconception period. Feto-placental consequences of maternal type-1 diabetes around the time of conception need to be explored.
Using a rabbit model, type-1 diabetes was induced by alloxan 7 days before mating. Glycemia was maintained at 15-20mmol/L with exogenous insulin injections to prevent ketoacidosis. At 4 days post-conception (dpc), embryos were collected from diabetic (D) or normoglycemic control (C) dams, respectively, and transferred into non-diabetic recipients. At 28dpc, D- and C-feto-placental units were collected for biometry, placental analyses and lipid profiles.
D-fetuses were growth-retarded, hyperglycemic and dyslipidemic compared to C-fetuses. The efficiency of D-placentas was associated with an increased gene expression related to nutrient supply and lipid metabolism whereas volume density of fetal vessels decreased. Fetal plasma, placental and fetal liver membranes had specific fatty acid signatures depending on embryonic origin. Tissues from D-fetuses contained more omega-6 polyunsaturated fatty acids. The concentrations of docosahexaenoic acid decreased while linoleic acid increased in the heart of D-fetuses.
This study demonstrates that a short exposure to maternal type-1 diabetes in the periconception window had programmed, adversely and durably, the fetal phenotype, through placental structural and molecular adaptations.