

pregnancies ($1.3 \pm 0.3 \times 10^8$). Incubation of cells with $MgSO_4$ reduced the release of exosomes in cells from normal pregnancies (2 mmol/L: $40 \pm 15\%$, 3 mmol/L: $25 \pm 11\%$), and preeclampsia (2 mmol/L: 28 and 110%, 3 mmol/L: 54 and 128%). Incubation of hPMECs from normal pregnancies with $MgSO_4$ increased $A_{2B}AR$ (~1.7 fold), iNOS (~1.4 fold), and eNOS (~1.5 fold) protein abundance. In cells from preeclampsia $MgSO_4$ increased $A_{2A}AR$ (~1.6 fold), $A_{2B}AR$ (~1.8 fold), and iNOS (~1.6 fold).

Conclusion: Extracellular Mg^{2+} cause differential modulation on protein expression in the foetoplacental microvascular endothelium from normal and LOPE pregnancies. Since Mg^{2+} reduced exosomes release, these nanovesicles may be involved in this phenomenon in the foetoplacental vasculature.

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CHANGES IN THE CARBOHYDRATE EXPRESSION IN CATTLE AND BUFFALOES INFECTED WITH THE ABORTIGENIC PROTOZOAN *NEOSPORA CANINUM*

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Carbohydrate pattern in the placenta was studied in different species showing a great variation between species. The sacharydes of the surface glycoconjugates are important to the adhesion between conceptus and uterus. Besides, reproductive diseases as tritrichomonosis and campylobacteriosis generate changes in this pattern in cattle.

Objective: The aim of this study was to characterize the saccharide in placenta of cattle and buffaloes after experimental infection with the abortigenic protozoan *Neospora caninum* (*N. caninum*) at early gestation by lectin histochemistry.

Methods: Heifers and buffaloes with 70 days of pregnancy were inoculated with 1×10^8 tachyzoites of *N. caninum* (inoculated groups; bovine n = 9, buffaloes n = 2) and PBS (control group; bovine n = 2, buffaloes n = 2). Nc-1 (n: 3), Nc-6 Argentina (n: 3) and Nc-Spain 7 (n: 3) strains were used in heifers. In buffaloes only Nc-1 strain was used. Placenta and uterus samples were collected at time of necropsy (28 days after inoculation). These tissues were analysed by histopathology analysis, immunohistochemistry (IHC) for *N. caninum*, lectin histochemistry (LHC), PCR and microsatellite genotyping.

Results: Histopathology analyses showed differences among groups, and no compatible lesions were found in control groups. IHC and PCR test were positives to *N. caninum* in the inoculated groups and negatives in control groups. Microsatellite genotyping assay determined that every group had just the specific strain inoculated. LHC results were different between species. Besides, inoculated groups change the carbohydrate expression.

Conclusion: *N. caninum* inoculation changes the pattern of saccharides present in the placenta and uterus tissues both in cattle and in buffaloes. This variation could generate alterations in pregnancy in infected animals.

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MATERNAL SUPRAPHYSIOLOGICAL DYSLIPIDEMIA IN PREGNANCY WORSE VASCULAR RESPONSE OF UMBILICAL VEIN RINGS FROM GESTATIONAL DIABETES MELLITUS.

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Pregnancy associates with increased maternal level of lipids (i.e., maternal physiological dyslipidaemia, MPD). However, when lipid level reaches over the physiological values, maternal supraphysiological dyslipidaemia (MSPD) is recognized. The consequences of MSPD in the foetus include foetoplacental endothelial dysfunction and increased foetal atherosclerosis. Gestational diabetes mellitus (GDM) associates with MSPD and endothelial dysfunction in umbilical veins, the latter caused mainly by maternal hyperglycaemia and hyperinsulinemia. However, the consequences of MSPD in GDM pregnancies in the foetal vasculature have been neglected.

Objectives: To determine whether MSPD alters endothelial-dependent umbilical vein reactivity in GDM pregnancies.

Methods: Placentas were collected at Hospital Clínico UC-CHRISTUS (Santiago de Chile). Umbilical veins rings were isolated from four study groups: GDM or normal pregnancies, with or without MSPD. Vein rings were mounted in a wire myograph and the response to insulin (0.1-1000 nmol/L, 5 min) or calcitonin-gene related peptide (CGRP, 0.01-100 nmol/L, 5 min) in KCl-precontracted vessels (32 mmol/L) was measured. Nitric oxide (NO)-dependent dilation was estimated in the absence or presence of the NO synthase inhibitor N^G -nitro-L-arginine methyl-ester (L-NAME, 100 μ mol/L, 20 min).

Results: GDM showed lower ($P < 0.05$, n = 5-10) NO-dependent maximal relaxation (R_{max}) in response to insulin (GDM: $17 \pm 2\%$, normal: $43 \pm 6\%$) and CGRP (GDM: $15 \pm 3\%$, normal: $30 \pm 3\%$). CGRP-dilation was further reduced in vein rings from GDM with MSPD (MSPD: $12 \pm 1\%$) and in normal pregnancies with MSPD (MSPD: $14 \pm 1\%$), but not in response to insulin.

Conclusion: MSPD contributes to GDM-associated endothelial dysfunction by worsening the vascular dilation of umbilical vein rings in this pathology.

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MODULATION OF THE PLACENTAL HDL AND LDL CHOLESTEROL UPTAKE BY THE MATERNAL LIPIDS LEVEL IN HUMAN TROPHOBLAST

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In normal pregnancies, a physiological increase of the maternal plasma cholesterol from trimester 1 (T1) to T2 and T3 satisfies the foetal growth