The involvement of angiotensin II (Ang II) in maximal decidualization has been reported. We demonstrated that Ang II activates the Ca^{2+} / calcineurin (CN)/ NFAT pathway in rat endometrial stromal cells (ESCs). Ang II modulates gene expression, proliferation and metalloprotease activity in ESCs that could be involved in decidualization.
Objective: To analyze *in vitro* a) whether Ang II induces decidual reaction and uterine receptivity in ESCs, b) the involvement of calcineurin, and c) whether Ang II secreted by trophoblasts could mediate those effects.
Methods: ESCs from rat uterus were cultured with Ang II or estrogen plus medroxyprogesterone acetate (E2+MPA), for 24 h or 6 days. ESCs were pretreated with Cyclosporin A to assess the involvement of CN. mRNA expression of desmin (decidual prolactin related protein (dprp)), and insulin growth factor binding protein 1 (Igfbp1), markers of decidualization, was measured by RT-PCR. mRNA and protein expression of HB-EGF and IGF-1, markers of uterine receptivity, were measured by RT-PCR and western blot. The rat choriocarcinoma-derived cell line Rcho-1, which can be differentiated *in vitro* into invasive trophoblasts, was cultured in the presence or absence of E2 (10^{-8} M) or MPA (1 μM) for 48 h. ESCs were then cultured with the trophoblast conditioned medium (TrCM) for 48 h in the presence or absence of the Ang II receptor antagonist Losartan.
Results: Ang II induced ($p<0.05$) the expression of *desmin*, *dprp*, *igfbp1*, HB-EGF and IGF-1 in ESCs. Cyclosporin A inhibited ($p<0.05$) the effect of Ang II. MPA-TrCM, compared to control TrCM, induced *dprp* (1.7 fold) and HB-EGF (2.1 fold) expression in ESCs. Losartan inhibited TrCM induction ($p<0.05$).
Conclusions: These results suggest that 1) the induction of markers of decidualization and uterine receptivity by Ang II appears to require calcineurin activity, 2) the activation of the renin-angiotensin-system in trophoblasts may induce the decidual reaction in ESCs.

PA.36.
PROGESTERONE RECEPTORS AND PROGESTERONE CONCENTRATION IN SERUM AND PORCINE MATERNAL PLACENTAL EXTRACTS

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Pregnancy in the pig is characterized by rapid development and endocrinological changes involving the conceptus and the uterine environment. Porcine placenta is epitheliochorial, folded, non-decidual, non-invasive and diffuse. Progesterone receptor (PGR) is expressed as two isoforms, PGRA and PGRB, which have been shown to have different functional activities.
Objectives: To investigate a) progesterone concentration in serum and maternal placental extracts (HoPM), b) PGR, PGRA and PGRB expressions in porcine placenta of different gestational periods (16–17, 30–35 and 63–78 days of gestation).
Methods: The porcine females from slaughterhouses (n=12) of 16–17, 30–35 and 63–78 days of gestation were used. PGR, PGRA and PGRB were visualized by immunohistochemistry using monoclonal antibodies. Progesterone (P_4) concentration was measured by chemiluminescence in maternal placental extracts and serum.
Results: Progesterone (P_4) concentration in maternal placental extracts was $\overline{x} = 1.54$ ng/ml at 16-17 days, $\overline{x} = 1.02$ ng/ml at 30-35 days, and $\overline{x} = 1.38$ ng/ml at 63-78 days, whereas that in serum was $\overline{x} = 36.66$ ng/ml at 16-17 days, $\overline{x} = 24.16$ ng/ml at 30-35 days; and $\overline{x} = 27.9$ ng/ml 63-78 days. Total progesterone receptor was expressed at 16-17 days of gestation in uterine glands and maternal stroma, in coincidence with the expression of the PGRA. At 30-35 and 63-78 days of gestation PGR and PGRA were expressed in maternal stroma, while uterine glands and epithelium were negative. No expression of PGRB was observed in the periods of gestation and structures studied.
Conclusions: Progesterone levels remain constant through pregnancy in HoPM and serum with tissue expression of PGRA. The results suggest that progesterone acts via PGRA in the periods of swine gestation studied.

PA.37.
UPREGULATION OF ANTIVIRAL FACTORS EXPRESSION BY LIGANDS OF TOLL-LIKE RECEPTORS IN CORD BLOOD OF HIV-INFECTED MOTHERS

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Introduction/Objective: A high percentage of infants from mothers infected by HIV-1 infection are uninfected even in the absence of antiretroviral therapy. This finding emphasizes the importance of evaluating the immunological characteristics in the maternal-fetal interface, mainly related to the innate immune response. The purpose of this study was to determine the expression of antiviral restricting factors in HIV-infected pregnant mothers and in cord blood after stimulation with ligands of Toll-like receptors (TLR).
Methods and Results: After cesarean delivery of HIV-1-infected mothers, blood was collected from umbilical cords and mothers to obtain mononuclear cells (MNC). MNC were stimulated for 4 hours with the TLR4/LPS and TLR7/8/CL097 ligands. The mRNA levels of the antiviral factors APOBEC3G/A3G, TRIM-5α/T5α, SAMHD1, STING and IFN-α, and endogenous retrovirus (HERV-K) were evaluated. After activation, the TLR4 ligand was able to potentiate the response of control mothers to A3G, T5α, SAMDH1 and STING and for RNs to T5α. The mother-newborn binomial infected by HIV, showed a deficit of signaling after activation via TLRs, except via TLR7/8 which was able to activate the expression of STING in newborns of infected mothers. The activation with ligands of TLRs was not able to alter the expression of HERV-K. However, it is necessary to expand the sampling.
Conclusion: These data suggest that stimulation of the TLR7/8 (CL097) agonist may be a potent inducer of antiviral factor in cells of umbilical cord from infected mothers, and so a future candidate for vaccine formulations

PA.38.
ROLE OF PRENATAL EXPOSURE TO ENVIRONMENTAL POLLUTANTS AND MATERNAL OBESITY IN FETAL PROGRAMMING OF CHRONIC DISEASES IN MICE

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Intrauterine exposure to adverse environmental conditions can affect the health of the adult offspring, through still mostly unexplained mechanisms known as "fetal programming".

Objectives: a) To evaluate whether intrauterine exposure to a mixture of environmental pollutants known as endocrine disruptors (ED), alone or combined with maternal obesity, affect the adult offspring metabolism. b) To evaluate reticulum endoplasmic stress (ER stress) in the liver as a possible mechanism mediating those metabolic disturbances.
Methods: C57BL/6J pregnant mice with normal weight or obese (25% overweight by high fat diet feeding) were injected i.p. daily from gestational day 7 (E7) to E15 with vehicle (sesame oil) alone or containing a mixture of endocrine disruptors (mED) DEHP+DPP+BBP(300mg/Kg/day)+4-nonilfenol+4-ter-Octilfenol(50mg/Kg/day)] in a complete dose (1X) and in 1/2X and 1/10X dilutions. Adult offspring were analyzed for body weight, glucose tolerance [i.p. glucose tolerance tests (GTTs)] and cholesterol levels. ER stress was studied in livers from male offspring by detecting classic response sensors (phospho-IRE1α, phospho-eIF2α and ATF6) by western blot.
Results: Intrauterine exposure to mED at 1X and 1/2X doses was associated with gestational shortening, smaller litters and death of neonates.