Methods: BeWo cells were cultured under standard conditions**.** BeWo cells were transiently transfected with different reporter vectors and expression vectors for Sp1 or ERα.
Results: We observed that Sp1 overexpression increased basal leptin promoter activity ($p < 0.0070$). This effect was enhanced by E_2 ($p < 0.01$). On the other hand, Sp1 increased leptin promoter activity of the reporter, which contains the promoter region of the leptin gene between -1951 and -1847 bp ($p < 0.05$), but not when the Sp1 element is mutated in this region. Sp1 effect was ERα-dependent as it had no activity in cells that had been knocked down with an ERα siRNA. We observed that there is a joint interaction between Sp1 and ERα regulating the expression of placental leptin.
Conclusions: All these findings suggest that leptin expression is tightly regulated and improve the understanding of the mechanisms whereby E_2 regulates leptin expression involving Sp1 transcription factor.

PA.16.
ADIPONECTIN RECEPTOR 1 EXPRESSION IN HUMAN UMBILICAL ARTERY ENDOTHELIAL CELLS (HUAEC) FROM LARGE FETUSES (LGA) OF OBESE WOMEN IS RELATED TO eNOS ACTIVATION

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Objectives: We aimed to determine whether Adiponectin Receptor 1 (AdipoR1) expression is related to eNOS activation in HUAEC. Additionally we studied the differential expression of AdipoR1 and eNOS activation in large for gestational age (LGA) fetuses of obese pregnant women compared to appropriate-for-gestational-age (AGA) fetuses of normal weight pregnant women.
Methods: Primary cultures of HUAEC were obtained from the umbilical cord of term single pregnancies of AGA babies from normal weight women (A/N) and LGA babies from obese women (L/O). AdipoR1 and eNOS mRNA was measured by qPCR (Sybr Green). AdipoR1 and eNOS protein expression was measured by Western blot and total and phospho-eNOS (p-eNOS) by ELISA.
Results: HUAEC expressed the mRNA and protein for AdipoR1. In basal conditions, mRNA and protein expression of AdipoR1 and eNOS were increased in HUAEC from the L/O compared to the A/N group. P-eNOS and the p-eNOS/eNOS ratio were decreased in the L/O group.
Conclusions: AdipoR1 is overexpressed in HUAEC from L/O and a negative association to eNOS activation could be associated with further vascular compromise. The participation of the classical AdipoR1 signaling pathway is currently being studied.
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PA.17.
MELATONIN INCREASES OFFSPRING SURVIVAL IN A MURINE MODEL OF PRETERM LABOR INDUCED BY LPS

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Preterm birth (PTB) is the leading cause of neonatal mortality and promotes delayed physical and cognitive development in children. Intra-amniotic infections are one of the main causes of PTB.
In a model of inflammation-associated PTB (induced by LPS), melatonin was administered on gestational day 14, preventing PTB in 50% of the cases and conferring fetal protection.
Objectives: a) To determine the consequences of melatonin and LPS treatment on the fetal brain. b) To evaluate whether maternal treatment with melatonin+LPS affects weight, physical landmarks in newborn mice and behavior in adulthood.
Methods: Histological studies were performed and IL-1β mRNA expression was determined in fetal brains on day 15 of pregnancy. At birth, pups were weighed and observed for physical landmarks. As adults, open-field, elevated plus maze and passive avoidance behavioral tests were also assessed.
Results: LPS induced IL-1β release and triggered neurovascular unit injury and cell damage in the fetal brain parenchyma. Melatonin blocked LPS-induced IL-1β expression, reduced cell infiltration and prevented the deleterious effects in brain parenchyma.
No differences were observed in body weight or physical landmarks: pinna detachment, incisor eruption, and eye opening. Maternal treatment with melatonin+LPS did not affect the offspring's horizontal and vertical locomotor activity following exposure to an open field test when compared to control or melatonin-treated mice. In repeated exposure to open field, all treated mice showed the same habituation memory. No differences were found in the anxiety-like behavior evaluated in the elevated plus maze. There were no effects of melatonin+LPS on associative memory in the passive avoidance test when compared to control or melatonin-treated mice.
Conclusions: We concluded that exposure to LPS is extremely detrimental to the fetal brain, but that a treatment with melatonin prevents brain injury. These results suggest a potential therapeutic use of melatonin as a tocolytic agent in order to prevent PTB and increase offspring survival.

PA.18.
INTRAUTERINE GROWTH RESTRICTED RATS EXERCISED AT PREGNANCY: MATERNAL-FETAL REPERCUSSIONS

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Background: There is evidence that the nature of fetal programming is such that it is involved in many disease phenotypes, including those of successive generations. Laboratory animal models have been developed as an attempt to understand the pathophysiological mechanisms involved in an unfavorable intrauterine environment. We hypothesized that the swimming program may lead to an adequate maternal environment, improving embryofetal development.
Objective: To evaluate the effect of swimming in pregnant rats born with intrauterine growth restriction (IUGR) and their offspring.
Methods: IUGR rats were obtained using streptozotocin-induced severe diabetic (SD) rats. The nondiabetic and SD pregnant rats generated offspring with appropriate (APA) and small (IUGR) weight for pregnancy age, respectively. At adult life, the APA group was maintained sedentary (non-exercised) and classified as control group and IUGR rats were distributed into two subgroups: non-exercised (IUGR) and exercised (IUGRex).
Results: The rate of mated rats in the IUGR group was reduced compared to the control group. During pregnancy, the IUGR rats presented hyperinsulinemia, impaired reproductive outcomes, decreased body weight, hypertriglyceridemia and hyperlactacidemia. The IUGRex rats presented reduced insulin and triglyceride levels. There was a reduced percentage of appropriate weight for pregnancy age (APA) fetuses in the IUGR and IUGRex groups in relation to the control group, and an increase in the proportion of small weight for pregnancy age (SPA) fetuses in the IUGRex rats compared with the control group.
Conclusion: Swimming improved lipid metabolism and increased insulin sensitivity. However, the offspring showed retarded growth, reinforcing the need to stimulate the exercise practice in women under supervision with different professional expertise to promote appropriate gestational conditions and to improve perinatal outcomes.
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