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IS-110 Significant increase in maternal plasma leptin concentration during induced labor : A possible contribution of pro-inflammatory cytokines to placental leptin secretion

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[Objective] Human placenta secretes leptin into maternal circulation and cuases maternal hyperleptinemia during pregnancy. The aims of this study are to study the effect of labor and pro-inflammatory cytokines on placental leptin secretion. [Methods] Under the patient's informed consent, we measured plasma leptin concentrations in pregnant women before and under induced labor (n = 6) and in postpartum women (n = 6). We also studied the effects of IL-1α, TNF-α, oxytocin or prostoglandin F2α (PGF2α) on leptin secretion from human term placental tissue and BeWo cells (a trophoblastic cell line). [Results] Plasma leptin concentration during labor (58.9 ± 9.2ng/ml, mean ± SEM) was significantly higher than that before labor induction (37.5 ± 5.8ng/ml, $P < 0.05$), but decreased to 14.2 ± 3.2ng/ml within 3—6 days. IL-1α (1, 2, 3ng/ml) and TNF-α (1, 10, 30ng/ml) dose-dependently stimulated leptin secretion from placental tissue in 24 hours culture (138 ± 17, 179 ± 17, 181 ± 13%increase, n = 4) and (130 ± 4, 148 ± 23, 163 ± 28%increase, n = 4), respectively. Similar augmentation by the same treatment was observed in BeWo cells. By contrast, oxytocin (1, 10μM) and PGF2α (1, 5, 10μM) did not affect leptin secretion from term placental tissue and BeWo cells. [Conclusion] These results suggest that pro-inflammatory cytokines may be involved in the increase in maternal plasma leptin concentration during labor by stimulating placental leptin secretion.

IS-111 Influence of Low Molecular Plasminogen Activator Activity on Onset of Labor and Hemostatic System

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[Objective] Both Bradykinin and Prostaglandin act on the muscles of uterus causing contraction. Fibrin plays an important role in maintaining the integrity of utero-placental circulation. Thereforeit is necessary to investigate the mechanism of the fibrinolytic parameters related to the hemostatic system during pregnancy and at the onset of labor. [Methods] With Informed consent, (1)44cases of normal pregnant women and 25cases of nonpregnant womens, Plasminogen Activator (U-PA activity) was measured in Zymography, and fractions were detected using Image-Analyzer. (2) Euglobulin-Lysis-Time (ELT) and Prekallikrein (S-2302) were tested as parameters of fibrinolytic andthe Kinin-Kallikrein system. [Results] (1). Urinary Plasminogen Activator activity of Low molecular weight (LMW) during the latter half of pregnancy was 0.32 ± 0.13 (%), but it decreased to 0.19 ± 0.03 (%) at the onset of labor ($p < 0.05$). (2) ELT during the latter half of pregnancy was prolonged (840.0 ± 26.4 min), at the onset of labor it shortened (356.6 ± 54.3min, $p < 0.05$). (3) Prekallikrein is decreased to 90.6 ± 16.8%after labor (latter half of pregnancy196.8 ± 33.6%). [Conclusion] The action of kallikrein upon LMW Plasminogen Activator causes it to change into plasmin at the onset of labor. They have an intimate relationship and effect the contraction of the uterus andthe hemostatic system.

IS-112 Induction of the hyaluronic acid-binding protein, tumor necrosis factor-stimulated gene-6, in cervical smooth muscle cells by tumor necrosis factor-alpha and prostaglandin E2

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[Objective] The role of hyaluronic acid (HA)-binding proteins in cervical ripening has not been explored. We examined the ability of PGE2 to induce expression of the HA-binding protein, tumor necrosis factor-stimulated gene (TSG) − 6, in human cervical smooth muscle cells (hCSMC) and compared the PGE2 response to that of TNF-a. [Methods] hCSMC were stimulated with PGE2 or TNF-a to compare the expression of TSG-6. A 1.3-kb of the TSG-6 proximal promoter region was cloned and transfected into hCSMC. The cells were stimulated with PGE2 or TNF-a to determine the effect on TSG-6 promoter activity. [Results] TNF-a stimulated TSG-6 mRNA in a dose and time dependent manner (maximal response : 10 ng/ml after 6 hours). PGE2 stimulated TSG-6 mRNA, but the magnitude of response was less than that produced by TNF-a, and it was maximal after 24 hours. TNF-a and PGE2 stimulated secretion of TSG-6 as detected by Western blotting. The effects of PGE 2 on secretion of TSG-6 were delayed compared to TNF-a. PGE2 increased TSG-6 promoter activity 1.75-fold. Paradoxically, TNF-a reduced TSG-6 promoter activity by 50%. [Conclusion] hCSMC express TSG-6 ; TSG-6 expression in hCSMC is regulated by PGE2 as well as TNF-a ; responses of hCSMC to TNF-a and PGE2 are distinct in terms of magnitude and the time course ; PGE2 and TNF-a exert different effects on the TSG-6 proximal promoter.

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