247 Effects of Adenosine on Human Decidual Prolactin (PRL) Production
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It is well known that human endometrial stromal cells can synthesize and secrete prolactin (PRL) after decidualization which seems to be essential for the maintenance of pregnancy. Although the decidualization is influenced mainly by progestogens, the regulatory mechanism(s) of decidual PRL production remains to be elucidated. In this communication, hence, we demonstrate that adenosine can regulate the decidual PRL production through the activation of protein kinase A.

Materials and Methods: Decidual cells for culture were prepared by the digestion of first trimester endometrial tissues with collagenase type I and deoxyribonuclease I, followed by Percoll gradient centrifugation. The concentrations of PRL and adenosine cyclic 3':5'-monophosphate (cAMP) in culture media were measured by enzymeimmunoassay and radioimmunoassay, respectively. The protein phosphorylation was analysed by two dimensional polyacrylamide gel electrophoreisis and autoradiography.

Results: adenosine dosedependently enhanced human decidual PRL production and cAMP production with the minimum effective dose of 1 μ M. Specific phosphorylation of 20 kDa nutral proteins was induced by the treatment of the culture cells with adenosine.

The results, therefore, suggest that adenosine plays an important biological role in the regulation of decidual PRL production by means of activation of protein kinase A.

248 Impaired suckling-induced prolactin secretion in streptozotocin-induced diabetic rats.
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To elucidate the reason for low milk yield in diabetic mothers, we examined prolactin (PRL) secretion by some stimulations in streptozotocin (STZ)-induced diabetic rats postpartum.

Pregnant Wistar rats were given citrate buffer (control group) or STZ only (DM group) or with insulin (insulin group). Growth of pups was significantly lower in the DM group than in the control group, but similar in the insulin group and the control group, suggesting the reduced milk volume in the DM group. Suckling-induced PRL secretion was significantly ($p<0.01$) lower in the DM group (123.2±27.7ng/ml) than in the control group (1086.0±181.5ng/ml), and intermediate in the insulin group (587.5±95.3ng/ml). TRH-induced PRL secretion was significantly ($p<0.05$) lower in the DM group than in the control group, but the same in the insulin group and control groups.

These results suggest that reduced milk secretion in diabetic mothers is partially due to impaired suckling-induced PRL secretion from the anterior pituitary and that insulin plays an important role in the mechanism of PRL secretion during suckling.

249 Tumor necrosis factor-α increases release of arachidonate and prolactin from rat anterior pituitary cells.
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We investigated the effect of tumor necrosis factor-α (TNF-α) on the release of arachidonate from dispersed anterior pituitary cells. Primary cultures of anterior pituitary cells from rats were preincubated with [^{3}H]arachidonate to label their phospholipid-containing components. TNF-α significantly increased the release of both PRL and [^{3}H]arachidonate release in a time- and dose-dependent manner. Other cytokines such as interleukin (IL)-1α, IL-1β and γ-interferon had no effect on [^{3}H]arachidonate release. To define the role of calcium, cells were incubated with low calcium medium, which decreased arachidonate release in response to TNF-α. BW755C, an inhibitor of the conversion of arachidonate to its metabolites, decreased TNF-α-induced PRL release, while indomethacin, a prostaglandin synthesis inhibitor, had no effect on TNF-α-induced PRL release. These data indicate that arachidonate metabolites may be involved in the process of TNF-α-induced PRL release.