13. EOSINOPHIL-MEDIATED NON-GENOMIC PARAMETERS OF ESTROGEN STIMULATION: A SEPARATE GROUP OF RESPONSES MEDIATED BY AN INDEPENDENT MECHANISM
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The present report describes evidence suggesting that eosinophil leukocytes mediate a separate group of responses to estrogens (uterine edema, increase in vascular permeability, release of histamine, etc.), independently from genomic activation. Conditions suppressing genomic activation do not interfere with this group of responses. Conditions interfering with eosinophil migration to the uterus (young age blood eosinopenia, eosinopenic hormones, agents blocking cell mobility) selectively interfere with this group of responses. It was proposed that estrogen cytosol-nuclear receptors are involved in estrogen-induced genomic activation, and that estrogen receptors from eosinophil leukocytes are involved in the migration of these cells to the uterus, where they would mediate the above responses. Conditions increasing (theophylline) or decreasing (thyroid hormones, insulin) estrogen binding by eosinophils selectively modify estrogen-induced uterine eosinophilia and the responses to estrogen proposed to be mediated by eosinophils in the uterus.

14. ESTROGEN-RESPONSIVE CREATINE KINASE IN IMMATURE RAT OVARY
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The immature rat ovary contains estrogen receptors and responds to estrogen administration by increase in weight, formation of corpora lutea and increased sensitivity to gonadotropins. The major component of the estrogen-induced protein in the uterus of the rat has been identified (Reiss, N. and Kaye, A.M. 1981. J. Biol. Chem. 256, 5741-5749), as the brain-type isozyme of creatine kinase (CK-BB); it was therefore of interest to determine if CK-BB is rapidly induced in the rat ovary by estrogen. Rats (25-27 days-old) were killed 1 h after i.p. injection of estradiol-17β(5μg) or 1% ethanol vehicle; 4-8 ovaries were incubated with 100μCi ^{35}S-methionine per ml of Dulbecco's modified Eagle's medium lacking methionine, at 37° C, for 90 min, in a 95% O_2, 5% CO_2 atmosphere. Fluorograms of the labelled cytosol proteins showed an increased rate of incorporation into a protein with the same migration as CK-BB. Daily injection of estradiol caused an increase in CK specific activity (38% after 4 days). The CK isozyme composition of immature rat ovarian cytosol was found to be exclusively CK-BB as determined by fractionation on a DEAE-cellulose column and a coupled spectrophotometric assay. These findings parallel results of experiments on the rat uterus and suggest that estrogen may induce a rapid increase in CK-BB synthesis in several estrogen responsive organs which result in a longer term increase in CK specific activity.

15. EFFECT OF NAFOXIDINE ON EARLY AND LATE RESPONSES OF IMMATURE RAT UTERUS TO ESTROGEN TREATMENT.
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We compared the time-course effects of a treatment with high and low doses of estradiol-17 β (E_2) (30 or 0.1 µg/100g b.w.) or of the partial agonist/antagonist, nafoxidine (10 or 100 µg/100g b.w.) on genomic (protein, RNA content) and non-genomic (early increase in wet weight, eosinophil migration) uterine responses. Nafoxidine was shown to act as a weaker agonist for the latter than for genomic responses. A strong parallelism was found in all instances between stromal eosinophilia and wet weight reponse. Pretreatment with nafoxidine for 48 hr abolished subsequent E_2 action on late genomic responses, not on the early wet weight responses, nor on eosinophil migration (in fact amplified). This therefore dissociates genomic from wet weight responses and lends further support to a role for eosinophils in estrogen-induced water imbibition.