Cellular mechanisms of estrogen-and dopamine-induced control of glandular kallikrein in the anterior pituitary of the rat

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Abstract

Glandular kallikrein (GK, a trypsin-like serine protease) exhibits estrogen induction and dopamine repression in rat pituitary lactotrophs. Steroid induction may reflect primary actions to increase selectively the synthesis of specific proteins, or may be part of broad cellular responses secondary to steroid-induced phenotype transitions. This study examined the cellular mechanisms underlying estrogen and dopaminergic control of lactotroph GK using a quantified immunocytochemical approach. Pituitaries from ovariectomized rats exhibited little GK staining. Estradiol treatment for 10 days produced dose-dependent increases in pituitary mass, the percentage of lactotrophs (indicating lactotroph proliferation) and the percentage of GK-positive cells. Also, GK staining intensity was dependent upon estradiol dose, increasing 4-fold between 5 μg and 50 μg/48 h. Dopamine receptor blockade with haloperidol (2.5 mg/kg/24 h) elicited weak GK immunostaining in 46% of the lactotrophs in the absence of estradiol, and markedly potentiated GK staining intensity elicited with low but not high doses of estradiol. The results suggest that GK induction is a primary estrogen effect, and is not secondary to a phenotype transition: the induction is enhanced by estrogen-induced lactotroph proliferation. Dopaminergic systems strongly inhibit GK induction by low estradiol levels. This dopaminergic modulation may shift the induction of lactotroph GK to physiological events associated with high estradiol levels or low dopaminergic tone.