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JOURNAL ARTICLE

Mechanism of action of gonadotropin-releasing hormone-stimulated Leydig cell steroidogenesis. II. Gonadotropin-releasing hormone stimulates phospholipid labeling

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To investigate mechanisms responsible for gonadotropin-releasing hormone (GnRH)-stimulated Leydig cell steroidogenesis, the effects of GnRH agonist [des-Gly¹⁰, (D-Ala⁶) GnRH] on phospholipid turnover were studied. GnRH agonist in concentrations of 10^{-9} to 10^{-7} M increased phosphatidic acid labeling $292 \pm 16\%$ (mean \pm SE), and phosphatidylinositol labeling $258 \pm 13.2\%$. GnRH agonist-stimulated phospholipid labeling was detectable as early as 2 minutes. GnRH antagonist completely blocked GnRH agonist-induced testosterone formation and phosphatidic acid and phosphatidylinositol labeling. Nifedipine in concentrations of 1 and 10 micrograms/ml inhibited GnRH agonist-stimulated testosterone formation but had no effect on ³²P incorporation into phospholipids. Our results suggest that GnRH agonist-stimulated Leydig cell steroidogenesis is calcium dependent and correlated with increased phospholipid turnover.

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