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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 +/- 16% (mean +/- SE), and phosphatidylinositol labeling 258 +/- 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 phosphatidylinosital 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.