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JOURNAL ARTICLE

Mechanism of stress-induced attenuation of the testicular response to gonadotropin: possible involvement of testicular opioids, a pertussis toxin-sensitive G-protein, and phosphodiesterase

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This study examined the potential role of testicular opioids, a pertussis toxin (PT)-sensitive G-protein, and phosphodiesterase in mediating the inhibitory effect of immobilization stress on testicular steroidogenesis in adult rats. The experiments were initiated with enriched preparations of Leydig cells, but the stress effect was not sustained in vitro either as a result of the disruption of the morphology of the testis and/or the time required for Leydig cell isolation. Consequently, testicular fragments from control and stressed (3-hour immobilization) rats were used in these experiments. When fragments from stressed rats were incubated for 2 hours in the absence and presence of human chorionic gonadotropin (hCG) (0.1,1, or 10 mIU), testosterone (T) production in response to 1 and 10 mIU hCG was lower ($P < 0.05$ and 0.01, respectively) than that from control fragments. Basal T secretion did not differ between stressed and control fragments. Naloxone (1, 10, or 100 mu M), did not alter basal or hCG-stimulated T secretion from control fragments, but it normalized the T response to hCG from stressed fragments. Control fragments also showed a reduced T response ($P < 0.05$) to hCG in the presence of beta-endorphin (beta-E; 36 nM). Incubation of control fragments with PT (30 ng) did not alter basal or hCG-stimulated T production. However, PT normalized ($P < 0.01$) hCG-stimulated T secretion from stressed fragments. Methylisobutylxanthine (MIX; 0.125 mM) elevated ($P < 0.01$) hCG-stimulated T production from control fragments, but hCG-stimulated T secretion from stressed fragments remained subnormal in the presence of the phosphodiesterase inhibitor. The data suggest that acute immobilization stress inhibits gonadotropin-induced T production in adult male rats via a mechanism involving testicular opioids and a PT sensitive G-protein. We found no evidence to suggest that a stress induced increase in the activity of phosphodiesterase was involved in this mechanism.

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