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Journal of Andrology, Vol 18, Issue 5 528-534, Copyright © 1997 by The American Society of Andrology

JOURNAL ARTICLE

The cellular mechanisms of corticotropin-releasing hormone (CRH)-stimulated steroidogenesis in mouse Leydig cells are similar to those for LH

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Previous reports have demonstrated that corticotropin-releasing hormone (CRH) treatment of primary cultures of mouse Leydig cells and MA-10 mouse Leydig tumor cells results in a dose-dependent stimulation of steroidogenesis, probably by acting through the cAMP/protein kinase A second messenger pathway. Based on this observation, the mechanism of CRH-stimulated steroidogenesis is now further investigated and compared to trophic hormone stimulation. Both cell types were treated with human chorionic gonadotropin (hCG) or CRH in the absence and presence of the following agents: the translation inhibitor cycloheximide, the transcription inhibitor actinomycin D, the protonophore carbonyl cyanide m-chlorophenylhydrozone (mCCCP), which disrupts the mitochondrial electrochemical gradient or the phorbol ester, phorbol-12-myristate 13-acetate (PMA), a stimulator of protein kinase C. Cortico-releasing hormone-stimulated steroidogenesis was completely blocked by cycloheximide in both cell types, indicating that CRH-stimulated steroidogenesis in mouse Leydig cells requires ongoing protein synthesis. Actinomycin D had profound inhibitory effects on CRH-stimulated steroidogenesis in MA-10 cells, and this inhibition was greater than that seen in mouse primary Leydig cells. mCCCP severely inhibited CRH-stimulated steroid production in both cell types, indicating that an electrochemical gradient across the inner mitochondrial membrane is required for CRH-stimulated steroidogenesis. In addition, PMA inhibited hCG- and CRH-stimulated steroidogenesis in MA-10 cells and CRH-stimulated steroidogenesis in primary Leydig cells, suggesting that activation of the protein kinase C pathway can influence protein kinase A stimulated steroidogenesis. Results of these studies suggest that the mouse Leydig cell steroidogenic response to CRH shares many similarities to that of the LH response.

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