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JOURNAL ARTICLE

Rat tumor Leydig cells as a test system for the study of Sertoli cell factors that stimulate steroidogenesis

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Transplantable rat Leydig cell tumor H-540 was used to study the interactions between Leydig and Sertoli cells. These tumor cells maintain their steroidogenic capacity when cultured. Their responsiveness to a number of agonists that affect normal Leydig cells is markedly changed. Steroidogenesis can no longer be stimulated by luteinizing hormone (LH), but the cells remain responsive to dibutyrylcyclic adenosine monophosphate (dbcAMP) and cholera toxin. Cultured cells mainly produce C21-steroids, but the ability to produce androgens can be restored by pretreatment with dbcAMP. Coculture with Sertoli cells increases steroidogenesis in H-540 cells, and this effect is enhanced by follicle-stimulating hormone (FSH). Experiments with a two-chamber culture system show that these effects are mediated by one or more diffusible factors, some of which may be short-acting. SCF, a Sertoli cell-derived factor that stimulates normal Leydig cells, is not the mediator in this system since it is unable to stimulate steroidogenesis in Leydig tumor cells. Immunoneutralization experiments show that insulin-like growth factor I (IGF-I) is a permissive factor required to maintain steroidogenesis in Leydig tumor monocultures and in cocultures with Sertoli cells, but addition of IGF-I does not mimic the stimulatory effect of coculture. It was concluded that factors other than SCF and IGF-I must be involved in the stimulatory effects of coculture, and that H-540 cells may be a useful tool for the study of these factors.