

Ovarian steroidal response to gonadotropins and β -Adrenergic stimulation is enhanced in polycystic ovary syndrome: Role of sympathetic innervation

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Experimental induction of a polycystic ovarian syndrome (PCOS) in rodents by the administration of a single dose of estradiol valerate (EV) results in activation of the peripheral sympathetic neurons that innervate the ovary. This activation is evidenced by an increased capacity of ovarian nerve terminals to incorporate and release norepinephrine (NE), an increase in ovarian NE content, and a decrease in ovarian β -adrenergic receptor number in the ovarian compartments receiving catecholaminergic innervation. The present experiments were undertaken to examine the functional consequences of this enhanced sympathetic outflow to the ovary. The steroidal responses of the gland to β -adrenergic receptor stimulation and hCG were examined in vitro 60 days after EV administration, i.e. at the time when follicular cysts are well established. EV-treated rats exhibited a remarkable increase in ovarian progesterone and androgen responses to isoproterenol, a β -adrenergic receptor agonist, with no change