Guanethidine-mediated destruction of ovarian sympathetic nerves disrupts ovarian development and function in rats

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Immunosympathectomy produced by treatment of newborn rats with antibodies to nerve growth factor (NGF) delays ovarian development and disrupts estrous cyclicity. While these alterations have been ascribed to loss of sympathetic neurons innervating the ovary, the treatment also causes partial loss of ovarian sensory innervation. The present experiments were undertaken to determine if selective interference with ovarian noradrenergic/sympathetic action would result in alterations of ovarian development similar to those caused by NGF antibodies (NGF Ab). We have used two approaches to disrupt catecholamine action on ovarian cells: 1) inhibition of /3-adrenoreceptors by local delivery of receptor blockers to the ovaries of juvenile rats; and 2) elimination of the sympathetic innervation by long term postnatal treatment with guanethidine (GD), an adrenergic neuron blocking agent. When GD is administered chronically it produces an autoimmune-mediated destruction of peripheral sympathetic ner