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Lack of Voltage-Dependent Calcium Channel Opening During the Calcium Influx Induced by Progesterone in Human Sperm. Effect of Calcium Channel Deactivation and Inactivation

ALBERTO MARTÍN GUZMÁN-GRENFELL AND MARCO T. GONZÁLEZ-MARTÍNEZ

From the Departamento de Farmacología, Facultad de Medicina, Universidad Nacional Autónoma de México, Ciudad Universitaria, México, D. F., México.

Correspondence to: Dr Marco T. González-Martínez, Departamento de Farmacología, Facultad de Medicina, Universidad Nacional Autónoma de México, Ciudad Universitaria, CP 04510, Apartado Postal 70-297, México, D. F., México (e-mail: tuliog@servidor.unam.mx).

Progesterone induces calcium influx and acrosomal exocytosis in human sperm.

Pharmacologic evidence suggests that voltage-dependent calcium channels

(VDCCs) are involved. In this study, membrane potential (V_m) and intracellular calcium concentration ($[Ca^{2+}]_i$) were monitored simultaneously to assess the effect of VDCC gating on the calcium influx triggered by progesterone. Holding the V_m to values that maintained VDCCs in a deactivated (-71 mV) closed state inhibited the calcium influx induced by progesterone by approximately 40%. At this V_m , the acrosomal reaction induced by progesterone, but not by A23187, was inhibited. However, when the V_m was held at -15 mV (which maintains VDCCs in an inactivated closed state), the progesterone-induced calcium influx was stimulated. Furthermore, the progesterone and voltage-dependent calcium influxes were additive. These findings indicate that progesterone does not produce VDCC gating in human sperm.

Key words: Intracellular calcium, mammalian sperm, membrane potential, membrane potential-sensitive dyes.

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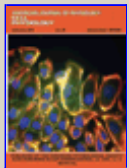


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