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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

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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 (Vm) 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 Vm to values that maintained VDCCs in a deactivated (-71 mV) closed state inhibited the calcium influx induced by progesterone by approximately 40%. At this Vm, the acrosomal reaction induced by progesterone, but not by A23187, was inhibited. However, when the Vm 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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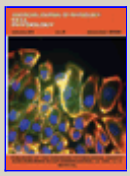

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