Voltage-dependent anion channels are a key factor of male fertility

2- Woo-Sung Kwon, Yoo-Jin Park, El-Sayed A. Mohamed, Myung-Geol Pang.

Abstract:

Abstract OBJECTIVE: To examine how voltage-dependent anion channels (VDACs) regulate sperm function in capacitation conditions. DESIGN: Experimental prospective study. SETTING: Academic research laboratory. ANIMAL(S): Male ICR and female B6D2F1/CrljOri mice (8-12 weeks old). INTERVENTION(S): Female mice were superovulated with 5 IU of pregnant mare serum gonadotropin given IP and 5 IU of hCG given IP 48 hours later. Oocytes were applied to assess fertilization and embryo development. MAIN OUTCOME MEASURE(S): Immunofluorescence assay, computer-assisted sperm analysis, hypo-osmotic swelling test, combined Hoechst 33258/chlortetracycline fluorescence assessment of capacitation status, measurement of [Ca(2+)](i) and [pH](i), Western blotting, and IVF. RESULT(S): VDAC2 was localized on the acrosomal region and principal piece, while VDAC3 was localized on the acrosomal region and midpiece. Blocking VDAC with DIDS (500 μM) significantly decreased motility, viability, acrosome reaction, capacitation, tyrosine phosphorylation, fertilization, and embryo development regardless of Ca(2+). However, the most severe decreases were observed in the presence (+) of DIDS and absence (-) of Ca(2+), respectively. A significant decrease in [Ca(2+)](i) concentration was observed in (-) DIDS, while [pH](i) was significantly increased in (-) DIDS regardless of Ca(2+). However, a significantly elevated [pH](i) was observed in (+) Ca(2+). CONCLUSION(S): Abnormal regulation of VDACs negatively affected sperm function. Thus, VDACs may be key regulators of the fertilization ability of spermatozoa.

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