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JOURNAL ARTICLE

Cholesterol inhibitory effects on human sperm-induced acrosome reaction

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Progesterone (P4) is known to induce an acrosome reaction in mammalian sperm in vitro, whereas cholesterol is a major inhibitor of acrosome reaction. This study had three objectives: to study the in vitro effects of exogenous cholesterol on acrosome reactions in human sperm, to study the mechanism by which cholesterol affects P4-induced acrosome reaction and those induced by dibutyryl cyclic adenosine monophosphate (db-cAMP), and to study the status of the P4 surface receptor during capacitation and acrosome reaction and its relationship with cholesterol and different acrosome reaction inducers. Acrosome reaction was induced with exposure to 10 microg/mL of P4 for 30 minutes and 1 mM of db-cAMP for 30 minutes in motile sperm either in the presence or absence of 0.1-1 microg/mL of cholesterol for 30 minutes. The effects of a 30-minute exposure to 1 microg/mL of beta-sitosterol, a cholesterol plant analogue, as well as the effects of cholesterol on P4-induced acrosome reactions were compared. Fluorescein isothiocyanate-labeled albumin-progesterone conjugate (P4-FITC-BSA) was used as the probe in order to quantify the percentage of sperm in which the P, surface receptor was exposed. The results of this study indicate that cholesterol inhibited P4-induced acrosome reactions when added to the sperm during capacitation (long incubation) and when it was added with P4 during the induction of acrosome reactions (short incubation). Similarly, acrosome reaction that was induced by db-cAMP was also inhibited by cholesterol. Fifty percent of P4-induced acrosome reaction was inhibited by a cholesterol concentration of 0.2 microg/mL. Cholesterol's inhibition of induced acrosome reaction was independent of P4 concentration. Beta-sitosterol inhibited P4-induced acrosome reaction in a dose-dependent manner that was identical to that of cholesterol. We observed that increases in the P4 surface receptor exposure were time-dependent and receptors migrated toward the equatorial segment during the first 2 hours of capacitation. We also found that db-cAMP induced the appearance of the P4 surface receptor in the sperm plasma membrane and that cholesterol inhibited it. The results of this study suggest that cholesterol inhibits acrosome reaction in a noncompetitive manner by modifying the structure of the sperm plasma membrane, which prevents exposure of the P4 surface receptor for P4 binding.

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