Suppress effect of seminal vesicle autoantigen on platelet-activating factor-induced mouse sperm capacitation

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摘要

Abstract

Mammalian sperm gain the ability to fertilize an egg successfully by the capacitation process. An unregulated capacitation process causes sperm to undergo a spontaneous acrosome reaction (AR) and resulting in loss of their fertilization activity. Thus, functional sperm activation is tightly regulated by a capacitation and suppression (decapacitation) mechanism. Factors, such as platelet-activating factor (PAF) present in both sperm and the female genital tract, are able to stimulate sperm capacitation. Seminal plasma is thought to have the ability to suppress sperm capacitation; however, the regulatory mechanisms of seminal plasma protein on sperm capacitation are not well understood. Recently, we demonstrated that seminal vesicle autoantigen (SVA), a major seminal vesicle secretory protein, is able to suppress mouse sperm capacitation. To further study the suppression spectra of SVA on sperm capacitation, we investigated the effect of SVA on PAF-induced mouse sperm capacitation-related signals. Here, we demonstrate that SVA decreases the [Ca(2+)](i) to suppress the PAF's effects on [Ca(2+)](i), the cAMP level, protein tyrosine phosphorylation, and capacitation. The inhibition of PAF-induced protein tyrosine phosphorylation and capacitation by SVA can be reversed by cAMP agonists. Characterization of the interactions of SVA with PAF by TLC overlay and tryptophan fluorescence spectrum analyses indicates that SVA is capable of binding PAF with an apparent dissociation constant K(d) > 50 microM. Together with these results, we demonstrate that SVA deceases [Ca(2+)](i) and cross-talks with PAF-induced intracellular signals to regulate mouse sperm capacitation.