小白鼠貯精囊自體抗原去精子獲能分子機制之研究

Decapacitation Mechanism of Sperm Induced by Mouse Seminal Vesicle Autoantigen

中文摘要

精子獲能是一連串生化反應，使精子具超泳動性，能進行頂體反應而擁有與卵受精的能力。精子從雄性睪丸分化及合成到進入雌性生殖道的旅程中，存在一些獲能因子以活化精子，同時也存在一些去獲能因子使精子不被提早活化，避免精子在與卵受精前就已能量消耗殆盡。目前關於精子獲能機制已被廣泛研究，然而針對去獲能因子機制的討論卻很少。本實驗室近幾年研究發現一貯精囊自體抗原(SVA)， 爲一 19 kDa 的醣蛋白，可與精子細胞膜上的帶有 choline 之 PC、SPM 磷脂質結合，扮演去獲能因子的角色，但其作用之分子機制則尚未釐清。本論文進一步探討 SVA 去精子獲能的機制，我們發現 SVA 減低精子鈣離子濃度、cAMP 濃度、細胞內 pH 值和抑制 protein tyrosine phosphory-lation 的表現，而能抑制 BSA / PAF 所引起的精子獲能，由這些實驗結果提示 SVA 的去獲能機制可能是經由 cAMP 的訊號傳遞路徑。另一方面，SVA 可以在不促使精子凋亡之下，抑制精子游動力並使精子粒線體膜電位下降，顯示粒線體活性也扮演著 SVA 去精子獲能過程中很重要的角色。除此之外，我們亦利用 DSS-linking 的方法，確定一在精子細胞膜上可能扮演 SVA 受體的蛋白，而此 SVA recptor 的特性則尚待釐清。

英文摘要

Sperm capacitation involves a complex biological molecular event to acquire the capacity for acrosome reaction and fertilization. During the transit of sperm from male testis to female oviduct, capacitation should take place at right time and right place in the reproductive tract after ejaculation. Factors promoted or inhibited sperm activity should interplay to prevent the gamete prematuriza- tion. Recently, the molecular events associated with capacitation are well documented, but less progress has been made to study the decapacitation effect. Here, we demonstrated a seminal vesicle autoantigen (SVA), a novel 19kDa phospholipids-binding protein, which serves as a decapacitation factor to suppress the mouse sperm capacitation induced by BSA / PAF, by reducing intracellular pH value, calcium ion concentration, and cAMP concentration. These data implicates the involvement of cAMP-dependent pathway in the decapacitation effect caused by SVA. Moreover, we found that the SVA effected the descending of mitochondria membrane potential as detected by Mitotracker- red feeding. The relevance of this observation to the SVA ability in the suppression of sperm motility awaits further study.