WAKING UP THE EGG. HOW THE SPERM REGULATES EXIT OUT OF THE MEIOTIC CELL CYCLE

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A series of calcium spikes are induced in the mammalian egg cytoplasm at fertilisation. These calcium spikes, which last for several hours, are the necessary and sufficient signal that stimulates the egg to escape from arrest at metaphase of the second meiotic division. Metaphase arrest is achieved by preventing the destruction of cyclin B1, the regulatory component of Maturation (M-Phase) Promoting Factor, and securin, which prevents segregation of sister chromatids. Both these proteins are destroyed by tagging with ubiquitin, using an E3 ligase the Anaphase-Promoting Complex (APC). Ubiquitination tags them for proteolysis by the 26S proteasome. Work from my lab has demonstrated that the sperm calcium signal works through activating the APC, not the 26S proteasome. Although we do not know which APC component is affected by calcium, this activation appears specific to a metaphase-arrested cell cycle state. More recently we have found that the APC is differently regulated at specific points during exit from meiosis II. Before extrusion of the second polar body it is the APC activator cdc20 that regulates APC activity. However, following extrusion of the second polar body cdh1 appears the major regulator. It is probable, therefore, that the calcium spiking affects the activity of both APC^{cdc20} and APC^{cdh1}. This swap in APC activator at the time of second polar body extrusion has not been reported in eggs of other species, in fact non-mammalian eggs all lack cdh1. Since APC^{cdc20} and APC^{cdh1} have different substrate specificities, the function of APC^{cdh1} in mammalian eggs warrants further investigation.

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