Signaling Pathways during Postovulatory Egg Aging in Rat

Shail K. Chaube

Cell Physiology Laboratory, Biochemistry Unit, Department of Zoology,
Banaras Hindu University, Varanasi-221 005, India
E-mail: shailchaubey@gmail.com

Abstract—*Postovulatory aging is associated with several morphological, cellular and molecular changes that deteriorate egg quality either by inducing abortive spontaneous egg activation (SEA) or by egg apoptosis. The reduced egg quality results in poor fertilization rate, embryo quality and reproductive outcome. Although postovulatory aging-induced abortive SEA has been reported in several mammalian species, the molecular mechanism(s) underlying this process remains to be elucidated. The postovulatory aging-induced morphological and cellular changes are characterized by partial cortical granules exocytosis, zona pellucida hardening, exit from M-II arrest and initiation of extrusion of second polar body in aged eggs. The molecular changes include reduction of adenosine 3'-5'- cyclic monophosphate (cAMP) level, increase of reactive oxygen species (ROS) and thereby cytosolic free calcium (Ca^{2+}) level. Increased levels of cAMP and/or ROS trigger accumulation of Thr-14/Tyr-15 phosphorylated cyclin-dependent kinase 1 (Cdk1) on one hand and degradation of cyclin B1 through ubiquitin-mediated proteolysis on the other hand to destabilize maturation promoting factor (MPF). The destabilized MPF triggers postovulatory aging-induced abortive SEA and limits various assisted reproductive technologies (ARTs) outcome in several mammalian species. Use of certain drugs that can either increase cAMP or reduced ROS level would prevent postovulatory aging-induced deterioration in egg quality so that the more number of good quality eggs can be made available to improve ART outcome in mammals including human.*

Keywords*: Postovulatory aging, Abortive SEA, Signal molecules, MPF, ART, Mammals.*