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Abortive Spontaneous Egg Activation: A Limiting Factor For Reproductive Outcome in Mammals

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Editorial

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EDITORIAL

Most of the mammalian species possess asynchronous ovary that exhibit different stages of follicles. During final stages of folliculogenesis, mammalian ovary ensures the generation of competent oocytes required for fertilization and embryonic development. To achieve this goal, large number of incompetent or defective germ cells (>99%) are eliminated from ovary through follicular atresia via apoptosis. At the time of puberty, limited number of germ cells are available in the ovary (<1%), which are culminated into oogonia and then primary oocytes [1]. These primary oocytes are arrested at diplotene stage until pituitary gonadotropin surge at the time of puberty [2]. Gonadotropins trigger meiotic resumption from diplotene arrest and these oocytes progresses to metaphase-II (M-II) stage just prior to ovulation [2,3].

In mammals, limited numbers of eggs are ovulated throughout the entire reproductive life span. These ovulated eggs are arrested at M-II stage of meiotic cell cycle possessing first polar body (PB-I) and normal morphology [4]. However, ovulated eggs do not wait for fertilizing spermatozoa and undergo meiotic exit from M-II arrest and initiation of second polar body (PB-II) extrusion, which never gets completed. This pathological condition is called as abortive spontaneous egg activation (SEA) [5-9]. Analyses of meiotic status of these eggs indicate that the chromosomes are scattered in the cytoplasm and eggs are arrested at metaphase-III (M-III) like stage without forming pronuclei [7,8]. Postovulatory egg aging is one of the causative factors for abortive SEA in several mammalian species [8].

Postovulatory aging may generate reactive oxygen species (ROS), which could result in oxidative stress (OS) in aged eggs. The increased OS can modulate mitochondria membrane potential as well as cytosolic free calcium (Ca^{2+}) level [10]. The increased OS as well as cytosolic free Ca^{2+} level triggers maturation promoting factor (MPF) destabilization.

The destabilized MPF triggers abortive SEA that deteriorates egg quality by inducing apoptosis [11]. These eggs are not suitable for fertilization as the chromosomes are scattered throughout the egg cytoplasm and programmed for OS-mediated apoptosis. Hence, the reproductive outcome *in vivo* as well as *in vitro* under various assisted reproductive technology (ART) programs including somatic cell nuclear transfer during animal cloning is severely affected. We propose that these factors could be involved in limiting ART outcome even after having optimal *in vitro* fertilization conditions in the laboratory.

Recent studies from our laboratory suggest that egg aging *in vivo* also results in abortive SEA [12]. This is an alarming condition for the mammals, since abortive SEA *in vivo* could result in poor reproductive outcome in several mammalian species. We speculate that the reduced reproductive potential of several mammalian species, which are at the verge of extinction, might be due to abortive SEA. Therefore, it is important to minimize the factors that trigger abortive SEA so that the reproduction ability

of mammals could be protected. This would be one of the important steps to increase the number of endangered mammalian species under animal conservation program in the country.

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