Effect of Maternal Age on the Oocyte Cortical Actin Layer and Associated Endoplasmic Reticulum Clusters

Ashley Teate[1]; Huizhen Wang[2]; Lane K. Christenson[1]; William H. Kinsey[1]

1. Department of Cell Biology and Physiology, University of Kansas Medical Center, Kansas City Kansas, United States of America
2. Department of Histology, University of Kansas Medical Center, Kansas City Kansas, United States of America

Mature oocytes in many mammalian systems produce specialized endoplasmic reticulum 'clusters' unique to this cell type that are thought to play a critical element in the Ca2+ oscillations generated at fertilization. Endoplasmic reticulum clusters may also be important in human fertility, as dysmorphisms in smooth endoplasmic reticulum clusters are associated with lower oocyte viability. Here we compare structural differences in ER clusters and their interaction with the cortical actin layer in oocytes from young mice (5-6 weeks; n=60) and aged (55-58 weeks, n=57) C57BL/6 mice obtained from Jackson Laboratory or the National Institute for Aging. Mice were superovulated and oocytes were fixed in situ in the oviducts, isolated, and then labeled with anti-human calreticulin, Alexa488 goat anti-rabbit, and Alexa568 phalloidin, prior to confocal microscopy. The endoplasmic reticulum clusters in oocytes from young females were consistently localized in close proximity to the 'fenestrae' of the cortical actin layer, excluding the microvillus-free zone over the spindle. In contrast, oocyte endoplasmic reticulum clusters from aged females frequently exhibited disassociation from the actin layer (15%) and structural abnormalities including the presence of vesicles (44%) and enlarged, distorted shapes (6%). Additionally, the 'actin' fenestrae were commonly reduced or absent in the oocytes from aged females, especially when endoplasmic reticulum clusters were not associated with the cortex (29%). These results share many similarities with observational reports in human oocytes from patients of advanced maternal age, suggesting that a systematic study of the mouse system could be used to identify possible functional defects associated with these abnormal structures.