Abstract

Proper spindle assembly during meiosis of mammalian oocytes ensures accurate chromosome segregation, thus preventing embryonic aneuploidy. Mammalian embryos, especially human embryos, show an increased frequency of aneuploidy, which can lead to spontaneous abortion or developmental defects. The spindle assembly of mammalian oocytes lacking centrioles is controlled by acentriolar microtubule-organizing centers and chromatin. Ran-GTP signalling, localization PRC1 protein at kinetochores, and Aurora kinase A (AURKA), among others, play important roles in this process. In this thesis, we focused on studying of the possible cooperation of PRC1 with Ran-GTP signalling and AURKA. Using confocal microscopy, we looked at PRC1 localization at kinetochores of mouse oocytes after inhibition of Ran-GTP signalling and in *Aurka*-deficient oocytes. Then, we compared their PRC1 intensity on kinetochores with control oocytes. Our results show that both Ran-GTP and AURKA regulate PRC1 localization on kinetochores in mouse oocytes. Using microscopy of live mouse oocytes, we show that overexpression of PRC1 can partially rescue the Ran-GTP inhibition phenotype regarding spindle bipolarization. We further showed that increased Ran-GTP activity can partially rescue the effect of *Prc1* knockdown in the case of spindle elongation. Our results also indicate that PRC1 overexpression in *Aurka*-deficient oocytes negatively regulates the timing of microtubule nucleation and positively regulates spindle bipolarization and chromosome alignment.

Key words

Aurora kinase A, chromosomes, meiosis, meiotic spindle, oocyte, protein regulator of cytokinesis 1 (PRC1), Ran-GTP