Abstract

Mammalian oocytes, especially human oocytes, are prone to defective chromosome segregation and are prone to aneuploidies, most commonly in the first meiotic division. Due to these defects, aneuploid embryos are formed after fertilization, resulting in numerous birth defects or infertility. The spindle formation in the first meiotic division in mammalian ooctes is a critical moment for preserving genome integrity. In mammalian oocytes, the spindle assembly is regulated via acentriolar microtubule-organizing centers and chromatine itself, through the activity of Aurora kinases and Ran-GTP gradient. Apart from the Aurora kinase family, other kinases regulate spindle formation, like Polo-like kinase 1 (PLK1). This thesis focused on potential cooperation between Aurora kinases and PLK1 or Ran-GTP. Our results show that the levels of phosphorylated Aurora kinase A (AURKA), but not levels of phosphorylated Aurora kinase B or Aurora kinase C, decrease after pharmacological inhibiton of PLK1. Levels of phosphorylated Aurora kinases do not change after pharmacological inhibition of Ran-GTP signaling. Using live-cell microscopy we showed that overexpression of PLK1 can rescue the phenotype observed in *Aurka* depleted oocytes. Next, we found that at least one of the signal pathways at chromosomes is necessary for proper spindle formation and microtubule nucleation. Our data suggest that during mouse oocyte meiosis, PLK1 regulates AURKA activity and that the Ran-GTP signaling pathway likely does not regulate the activity of any Aurora kinase.

Key words

Meiosis, oocyte, spindle, microtubule-organizing center, potein kinases, Aurora kinases, Polo-like kinase 1, Ran-GTP