PLK1 belongs to the extended family of serine/threonine kinases controlling the cell cycle. It is well known for its role in the control of mitosis and contributes also to the regulation of meiotic division.

On a basis of Live Cell Imaging (LCI) experiments we can describe the phenotype of the oocytes with PLK1 inhibited by small molecular inhibitor BI2536. PLK1 inhibition leads to delayed nuclear envelope breakdown (NEBD) and chromatin condensation (CC) and also causes desynchronization of NEBD and CC; in contrast to control oocytes, PLK1 inhibited oocytes break down their nuclear envelope with chromatin almost fully condensed. Also duration of these two early nuclear events is prolonged in oocytes with inhibited PLK1. In contrast to somatic cells, PLK1 inhibition in mouse oocytes does not prevent assembly of spindle with two distinct poles but affects the final spindle volume. Similar to somatic cells, mouse oocytes with PLK1 inhibited from the beginning of the meiotic maturation stay arrested in metaphase I but in the case of mouse oocytes, this block is not dependent on Spindle Assembly Checkpoint (SAC) persisting activity. When mouse oocytes are synchronized on metaphase I/anaphase I transition by proteasome inhibition and then PLK1 kinase activity is inhibited, about 2/3 of the oocytes stay arrested with chromatin in metaphase figure and the rest undergo anaphase accompanied by many missegregations. In both these groups of PLK1 inhibited oocytes, an inhibitor of separase - securin is degraded to the level similar like in the control oocytes though the securin destruction is significantly slower after PLK1 inhibition. Moreover, an attempt for the first polar body extrusion follows the error-prone anaphase in PLK1 inhibited mouse oocytes. But cytokinesis is impaired after PLK1 inhibition; the first polar body is never fully separated and finally it is absorbed back to the oocyte.

All these results show that PLK1 controls multiple aspects of meiosis I in mouse oocytes.