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Weber Beringui Feitosa, KeumSil Hwang, Patricia L. Morris

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Temporal and SUMO-specific SUMOylation contribute to the dynamics of Polo-like kinase 1 (PLK1) and spindle integrity during mouse oocyte meiosis

Weber Beringui Feitosa[1], KeumSil Hwang[1] and Patricia L. Morris[1,2]

[1]Center for Biomedical Research, Population Council, [2]The Rockefeller University

1230 York Avenue, New York, NY 10065, USA

Corresponding author: Patricia L. Morris; p-morris@popcbr.rockefeller.edu

ABSTRACT

During mammalian meiosis, Polo-like kinase 1 (PLK1) is essential during cell cycle progression. In oocyte maturation, PLK1 expression is well characterized but timing of posttranslational modifications regulating its activity and subcellular localization are less clear. Small ubiquitin-related modifier (SUMO) posttranslational modifier proteins have been detected in mammalian gametes but their precise function during gametogenesis is largely unknown. In the present paper we report for mouse oocytes that both PLK1 and phosphorylated PLK1 undergo SUMOylation in meiosis II (MII) oocytes using immunocytochemistry, immunoprecipitation and *in vitro* SUMOylation assays. At MII, PLK1 is phosphorylated at threonine-210 and serine-137. MII oocyte PLK1 and phosphorylated PLK1 undergo SUMOylation by SUMO-1, -2 and -3 as shown by individual *in vitro* assays. Using these assays, forms of phosphorylated PLK1 normalized to PLK1 increased significantly and correlated with SUMOylated PLK1 levels. During meiotic progression and maturation, SUMO-1-SUMOylation of PLK1 is involved in spindle formation whereas SUMO-2/3-SUMOylation may regulate PLK1 activity at kinetochore-spindle attachment sites. Microtubule integrity is required for PLK1 localization with SUMO-1 but not with SUMO-2/3. Inhibition of SUMOylation disrupts proper meiotic bipolar spindle organization and spindle-kinetochore attachment. The data show that both temporal and SUMO-specific-SUMOylation play important roles in orchestrating functional dynamics of PLK1 during mouse oocyte meiosis, including subcellular compartmentalization.

Keywords: Cell cycle, SUMOylation; Meiosis; Kinetochore; Centrosome, Spindle, MTOC