The study on regulatory mechanisms of Myt1 through its N-terminal domain in the cell cycle of Xenopus laevis

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論　文　内　容　の　要　旨

Immature animal oocytes are naturally arrested at the first meiotic prophase (Pro-I), which corresponds to the G2 phase of the cell cycle. In *Xenopus* oocytes, Myt1 kinase phosphorylates and inactivates cyclin-dependent kinase 1 (Cdk1) at Pro-I, thereby preventing oocytes from entering meiosis I (MI) prematurely. Previous studies have shown that, upon resuming MI, Cdk1 and p90rsk, which is a downstream kinase of the Mos-MAPK pathway, in turn phosphorylate the C-terminal region of Myt1, to suppress its activity, thereby ensuring high Cdk1 activity during M phase. However, the roles of the N-terminal region of Myt1 during meiosis and mitosis remain to be elucidated. In the present study, I show that the N-terminal region of Myt1 participates in the regulation of Myt1 activity in the *Xenopus* cell cycle. In particular, I found that a short, conserved sequence in the N-terminal region, termed here as the PAYF motif, is required for the normal activity of Myt1 in oocytes. Furthermore, multiple phosphorylations by Cdk1 at the Myt1 N-terminal region were found to be involved in the negative regulation of Myt1. In particular, phosphorylations at Thr11 and Thr16 of Myt1, which are adjacent to the PAYF motif, were found to be important for the inactivation of Myt1 in the M phase of the cell cycle. These results suggest that in addition to the regulation of Myt1 activity via the C-terminal region, the N-terminal region of Myt1 also plays an important role in the regulation of Myt1 activity.