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IN BRIEF

MITOSIS

PP1-mediated dephosphorylation of phosphoproteins at mitotic exit is controlled by inhibitor-1 and PP1 phosphorylation

Wu, J. Q. *et al. Nature Cell Biol.* 26 Apr 2009 (doi:10.1038/ncb1871)

Proteins that are phosphorylated by CDC2 must be dephosphorylated for cells to exit mitosis. Wu *et al.* now reveal that protein phosphatase 1 (PP1) dephosphorylates mitotic phosphoproteins in vertebrates. PP1 is inhibited during mitosis by CDC2-mediated phosphorylation and by inhibitor 1 binding. Inhibitor 1–PP1 binding is promoted by the phosphorylation of inhibitor 1. During mitotic exit, CDC2 inactivation allows PP1 to autodephosphorylate and to subsequently dephosphorylate and inactivate inhibitor 1. PP1 is then free to dephosphorylate mitotic phosphoproteins and promote mitotic exit.

GENE EXPRESSION

SATB1 defines the developmental context for gene silencing by *Xist* in lymphoma and embryonic cells

Agrelo, R. *et al. Dev. Cell* **16**, 507–516 (2009)

The non-coding RNA *Xist* silences gene expression during X chromosome inactivation. This chromosome-wide gene silencing is initially reversible, and genes can be reactivated in embryonic stem (ES) cells when *Xist* expression is abolished. It has now been shown that special AT-rich sequence-binding protein 1 (SATB1) is expressed in ES cells exclusively when *Xist* initiates chromosome-wide silencing. SATB1 overexpression also enables *Xist*-mediated gene silencing in mouse embryonic fibroblasts — cells in which *Xist* usually has no effect. Thus, SATB1 is a silencing factor that contributes to the initiation of X inactivation.

PROTEIN DEGRADATION

S5a promotes protein degradation by blocking synthesis of nondegradable forked ubiquitin chains

Kim, H. T. *et al. EMBO J.* 23 Apr 2009 (doi:10.1038/emboj.2009.115)

RING-finger ubiquitin E3 ligases form forked ubiquitin chains in the presence of the E2 enzyme UBCH5. Proteins that are conjugated to forked ubiquitin chains resist degradation by the 26S proteasome. Kim *et al.* show that S5a — a ubiquitin-interacting motif protein — dramatically stimulates the degradation of proteins that are ubiquitylated in the presence of a RING-finger E3 ligase and UBCH5. S5a does this by preventing the formation of forked ubiquitin chains, which bind poorly to the 26S proteasome, thereby ensuring the synthesis of efficiently degraded non-forked ubiquitin chains.

POLYCOMB PROTEINS

Bmi1 regulates mitochondrial function and the DNA damage response pathway

Liu, J. *et al. Nature* 29 Apr 2009 (doi:10.1038/nature08040)

Mice that are deficient for the transcriptional repressor BMI1 of the Polycomb family have several abnormalities, including a defect in thymocyte maturation. This study shows that cells from *Bmi1*$^{-/-}$ mice have a mitochondrial deficiency, which results in an increase in cellular reactive oxygen species (ROS). The increase in ROS triggers the DNA damage repair (DDR) pathway by activating CHK2. Treatment with antioxidants or deletion of CHK2 restores many wild-type properties to *Bmi1*$^{-/-}$ thymocytes. So, BMI1 regulates mitochondrial function and the DDR pathway.