ID No.	K2007
Project Title	Analysis of plethora of ASXL1 functions under physiological and pathological conditions
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Report

1. We have identified a transcription factor HHEX by insertional mutagenesis and found that HHEX collaborate with ASXL1-MT in enhancing leukemogenesis via de-ubiquitination of H2AK119Ub (Takeda et al. Blood in press).

2. We have found that ASXL1-MT stabilizes and activates BAP1, which stabilizes phosphorylated Akt, resulting in cell cycle progression of hematopoietic stem cells (HSCs), which would induce replication stress and activation of mitochondria of HSCs (Fujino et al. manuscript in revise).

3. We have found that major phosphorylation site of ASXL1 by CDK1/2 is S503 of ASXL1. However, SA and SD mutants of S503 showed no phenotype. We have now found that ASXL1-MT-6A mutant, where all potential phosphorylation sites are changed to alanine which cannot be phosphorylated, presented phenotypes in growth and differentiation of hematopoietic cells.