Foot notes

Fig.1. Schematic of canonical PRC1 component and mechanism of chromatin repression. (a) Canonical PRC1 consists of 4 components: PCGF2/4, CBX, HPH and RING. Non-canonical PRC1 is composed of RING proteins, PCGF1/3/5/6, RYBP/YAF2, and other co-factors. (b) PCGF and RING form a heterodimer and function as an E3 ubiquitin ligase. H2AK119Ub causes chromatin compaction leading target gene silencing.

Fig.2. Relationship between clinical outcome and *RING1* expression on messenger RNA (mRNA) level. (a) Overall and event-free Kaplan-Meier curves for 498 of NB patients with low versus high *RING1* mRNA expression (GE62564). (b) Overall and event-free Kaplan-Meier curves for 709 of NB patients with low versus high *RING1* mRNA expression (E-MTAB-1781).

Fig3. Over expression of mRing1A on NGP. Results were representative of three independent experiments. (a) Expression of RING1A and RING1B after mRing1A over expression was confirmed by Western blot analysis. (b) Cell viability was investigated by flat colony assay on the 19th day after transfection.

Fig.4. Structure of pLKO-Tet-On vector and mechanism of inducible knockdown. (a) pLKO-Tet-On vector has 2 restriction enzymes, EcoR I and Age I, into which any shRNA can be inserted. (b) In the absence of tetracycline (TET)/doxycycline (DOX), shRNA expression is repressed by constitutively-expressed TetR protein. Upon the addition of TET or DOX to the growth media, shRNA expression is triggered resulting in a target gene knockdown.

Fig.5. RING1A knockdown by inducible sh*RING1* #1, #2 in NGP. (a) Expression of RING1A and RING1B after RING1A knock down was confirmed by Western blot analysis. 100 ng/ml of doxycycline (DOX) was added every other day to induce RING1A knockdown. Signals were scanned and analyzed by ImageJ software. RING signals were normalized by GAPDH signals and indicated as bar graphs. (b) Cell viability was investigated by counting the number of cells on 6 and 9 days after seeding $1x10^4$ cells/5ml each on the first day. 100 ng/ml of DOX or equal amount of dimethyl sulfoxide (DMSO) was added every other day to induce RING1A knockdown or as a control. Results were representative of three independent experiments.

Fig.6. Amino acid sequences of mouse Ring1A (mRing1A) and human RING1A (hRING1A). Sequences are obtained from NCBI, National Center for Biotechnology Information with accession number NP_033092.3 (mRing1A) and NP_002922.2 (hRING1A).