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Supplemental Material to:

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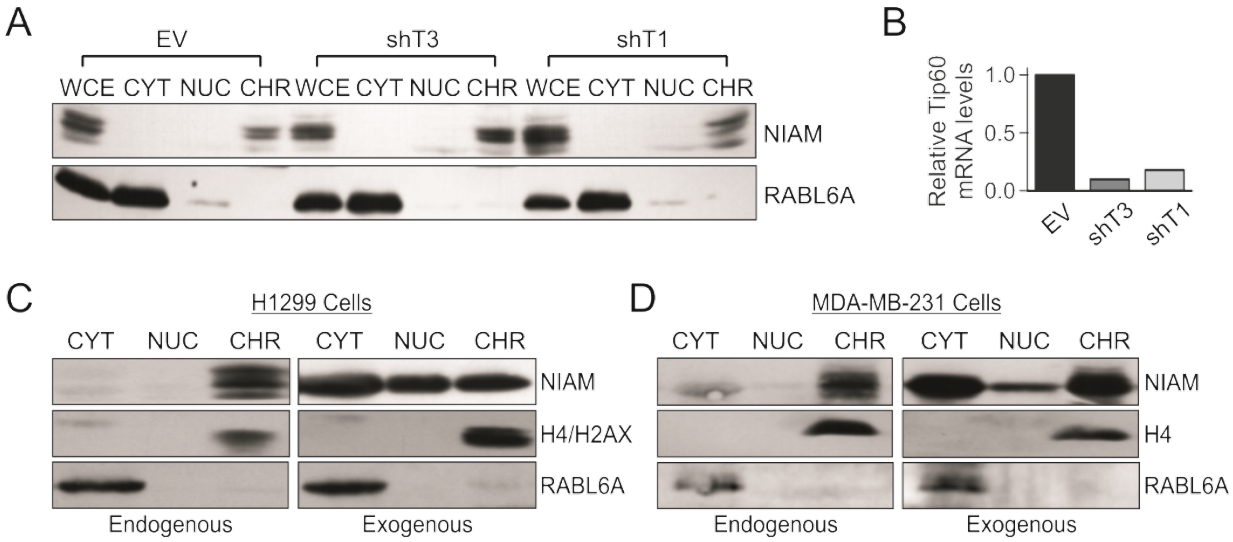
Nuclear interactor of ARF and Mdm2 regulates multiple pathways to activate p53

Cell Cycle 2014; 13(8)
http://dx.doi.org/10.4161/cc.28202

http://www.landesbioscience.com/journals/cc/article/28202



Supplemental Figure 1. The association of endogenous NIAM with chromatin does not require Tip60, Mdm2, or p53. (A) Localization of endogenous NIAM was determined in U2OS cells expressing empty vector (EV) or Tip60 knockdown constructs (shT1 and shT3). Chromatin (CHR), nucleoplasmic (NUC), and cytoplasmic (CYT) fractions were isolated by biochemical fractionation and protein localization in equivalently loaded fractions was examined by western blotting for NIAM and RABL6A (cytosolic control). WCE, whole cell extracts. **(B)** qRT-PCR demonstrates effective knockdown of Tip60 in cells from (A) expressing shT1 and shT3 relative to EV control. **(C and D)** Endogenous and exogenous NIAM localization was determined as in (A) by biochemical fractionation in (C) H1299 cells (p53-null) and (D) MDA-MB-231 cells (mutant p53). NIAM localization is shown relative to histone 4 or H2AX (chromatin-bound) and RABL6A (cytoplasmic protein) controls. Whereas endogenous NIAM was almost exclusively chromatin bound, exogenous NIAM redistributed throughout the cells.