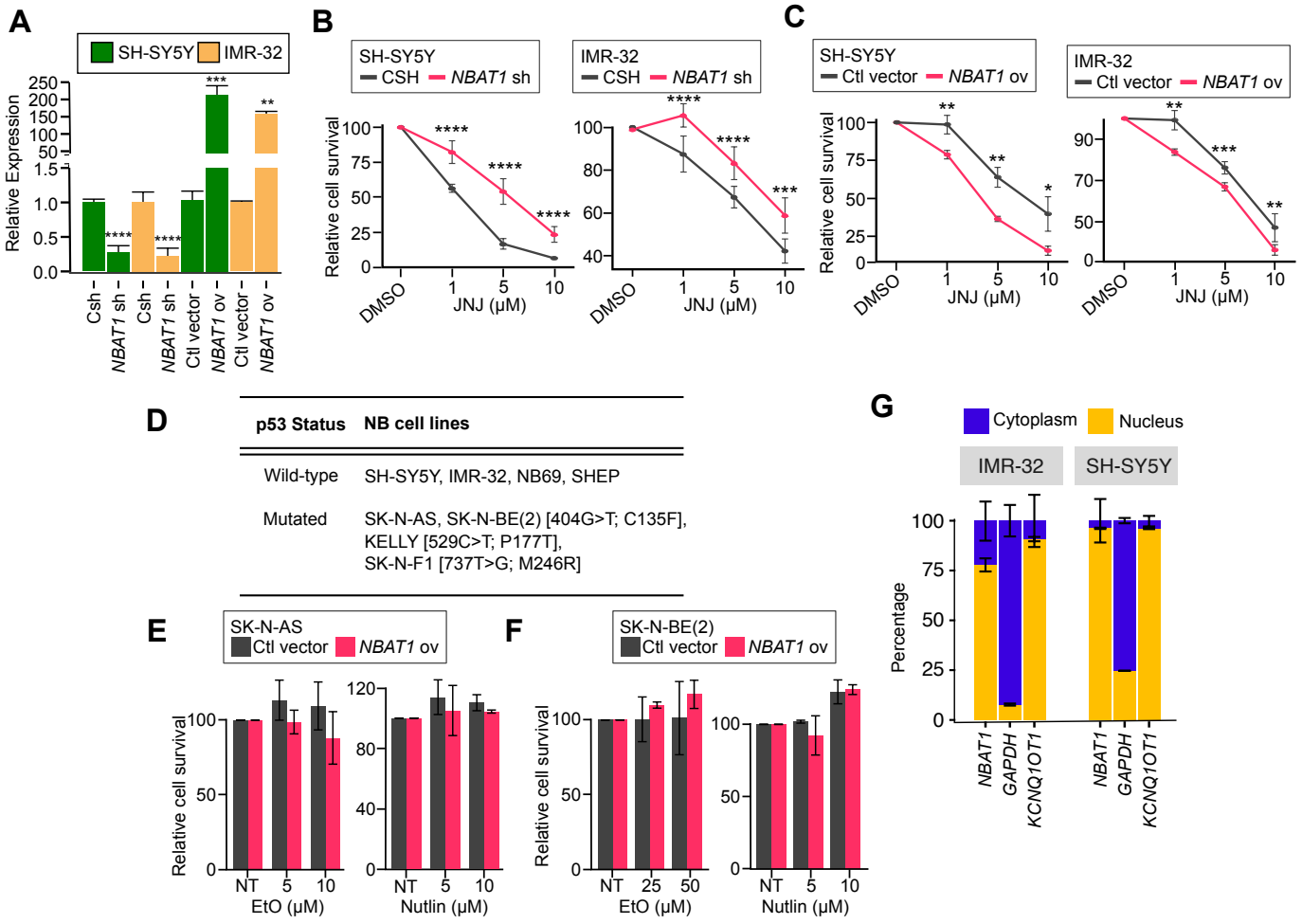
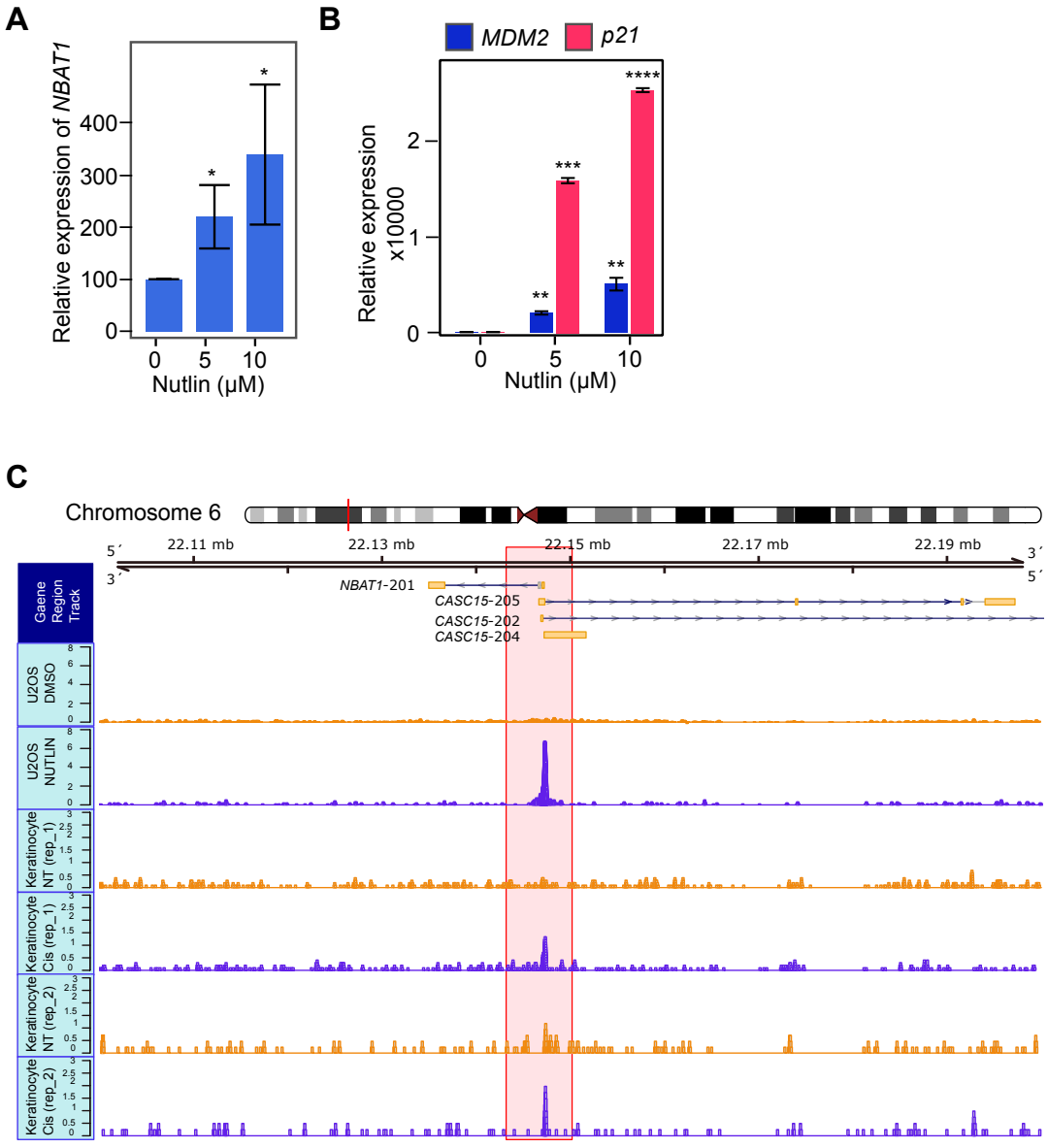
Supplementary Fig. S1



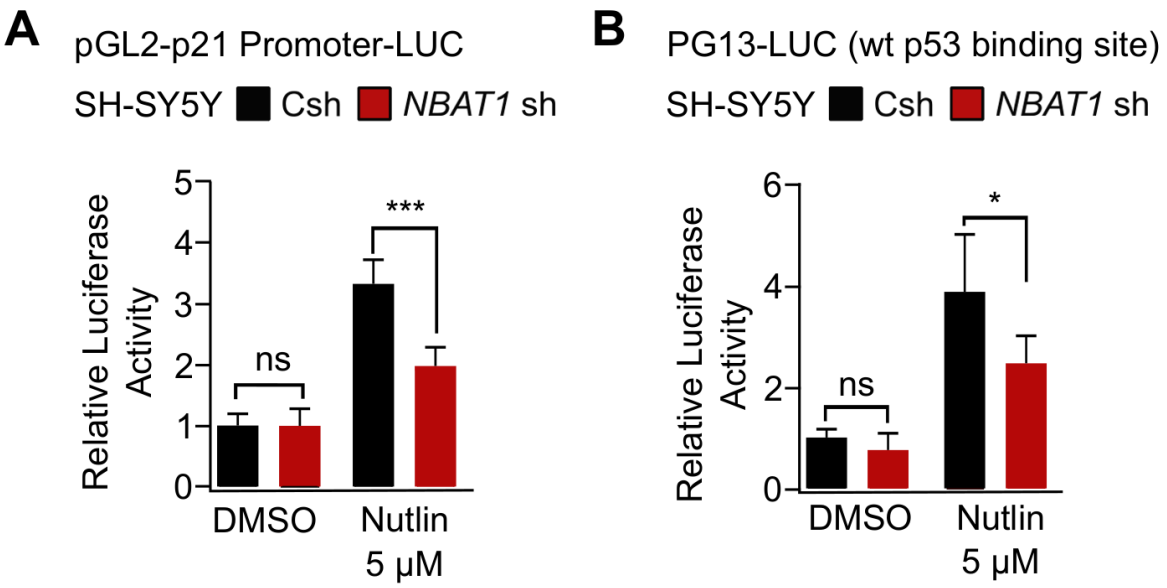
Supplementary Fig. S1. A) Relative expression of *NBAT1* in SH-SY5Y and IMR-32 cell lines with stable *NBAT1* knockdown (*NBAT1* sh) or *NBAT1* overexpression (*NBAT1* ov). **B)** Relative cell survival was measured using MTT of cell lines SH-SY5Y and IMR-32, stably transduced with control shRNA (Csh) or *NBAT1* shRNA (*NBAT1* sh), and treated with indicated concentration of JNJ or vehicle control DMSO. **C)** Relative cell survival of SH-SY5Y and IMR-32 cell lines, stably transfected with control vector or *NBAT1* expression vector followed by treatment with indicated concentrations of JNJ or vehicle control DMSO. **D)** *p53* mutational status in different NB cell lines **E-F)** Relative cell survival was measured using MTT of cell lines SK-N-AS and SK-N-BE(2) cells treated with the indicated concentrations of etoposide (Eto), nutlin-3a (Nutlin) or vehicle control DMSO. **G)** RT-qPCR showing sub-cellular distribution of *NBAT1* in the cytoplasmic and nuclear fractions in IMR-32 and SH-SY5Y cells. *KCNQ1OT1* was used as a nuclear control and *GAPDH* was used as a cytoplasmic control.

Supplementary Fig. S2



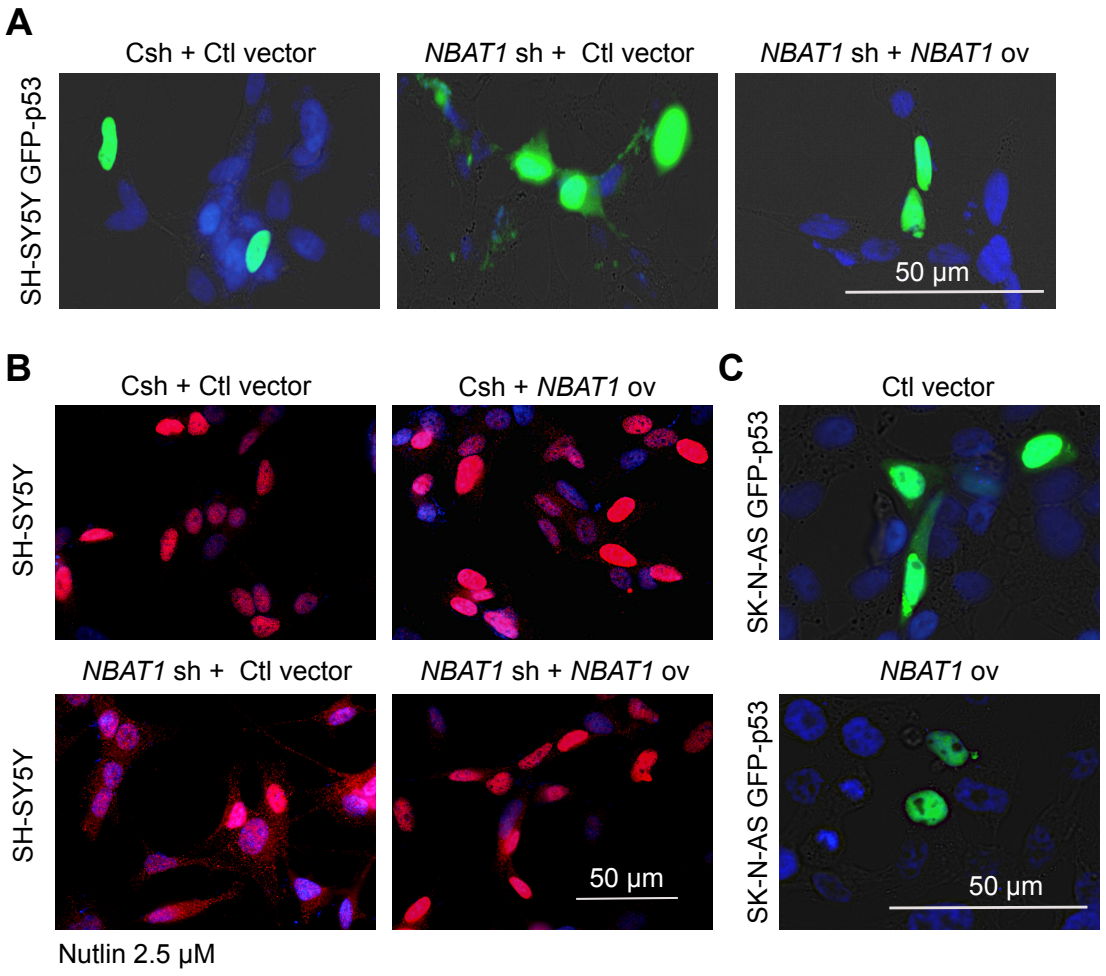
Supplementary Fig. S2. A-B) Relative expression of *NBAT1* and *p53* target genes (*p21* and *MDM2*) in Nutlin or DMSO (vehicle) treated SH-SY5Y cells. **C)** p53 ChIP-seq read distribution over the *NBAT1* promoter in the publicly available ChIP-seq data from U2OS cells with DMSO (control) and Nutlin treatments, and Keratinocyte cells with no treatment (NT) or with cisplatin (Cis) treatment.

Supplementary Fig. S3



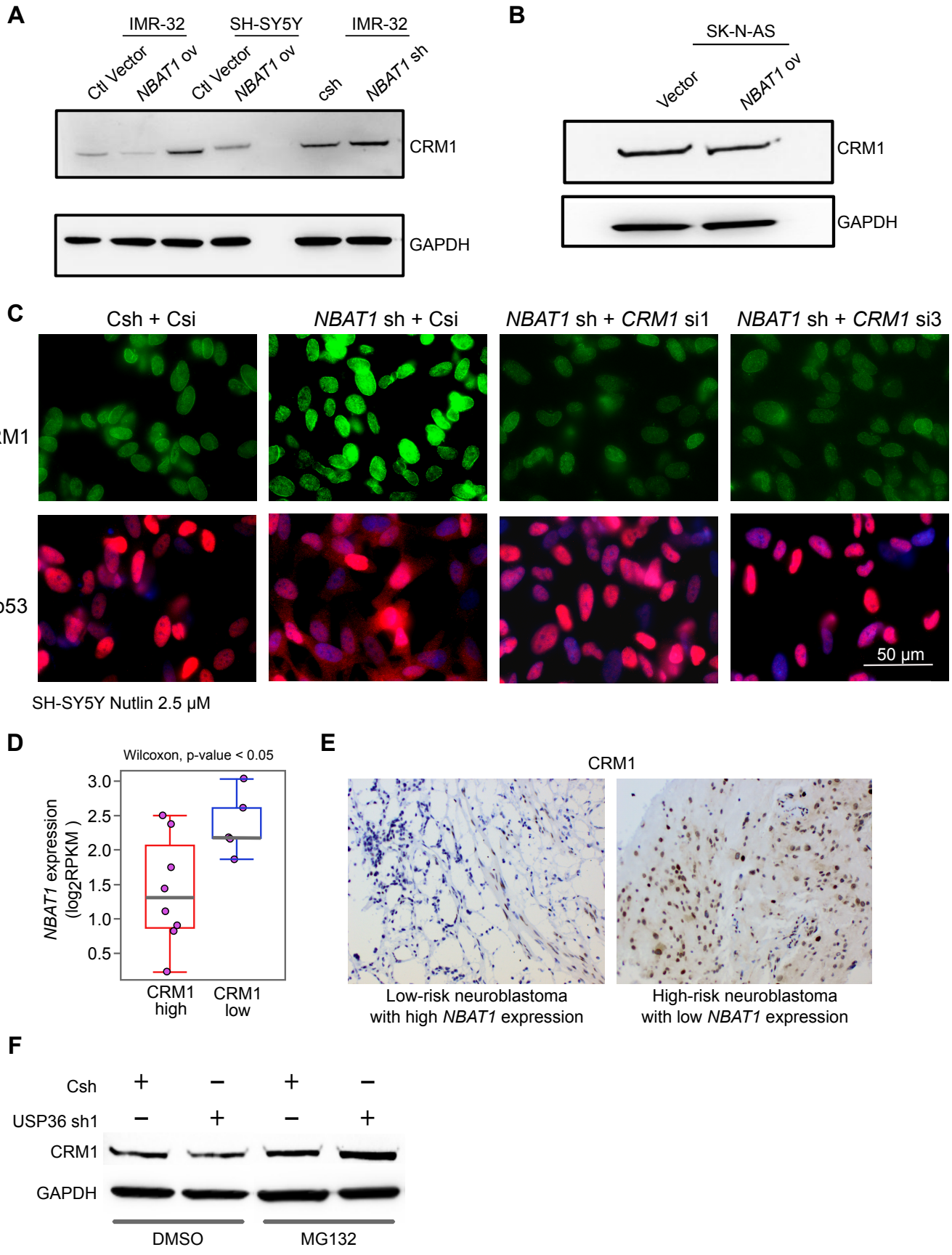
Supplementary Fig. S3. Luciferase reporter assay showing compromised p53 activity in the *NBAT1* depleted cells. A-B) *p21* promoter (pGL2-p21 Promoter-LUC) or a synthetic promoter, with wild-type p53 binding sites (wt p53-LUC), containing luciferase constructs were transfected into Csh and *NBAT1* sh cells. 24 hr post-transfection, the cells were treated with either Nutlin or DMSO and luciferase activity was measured 24 hr after treatment.

Supplementary Fig. S4



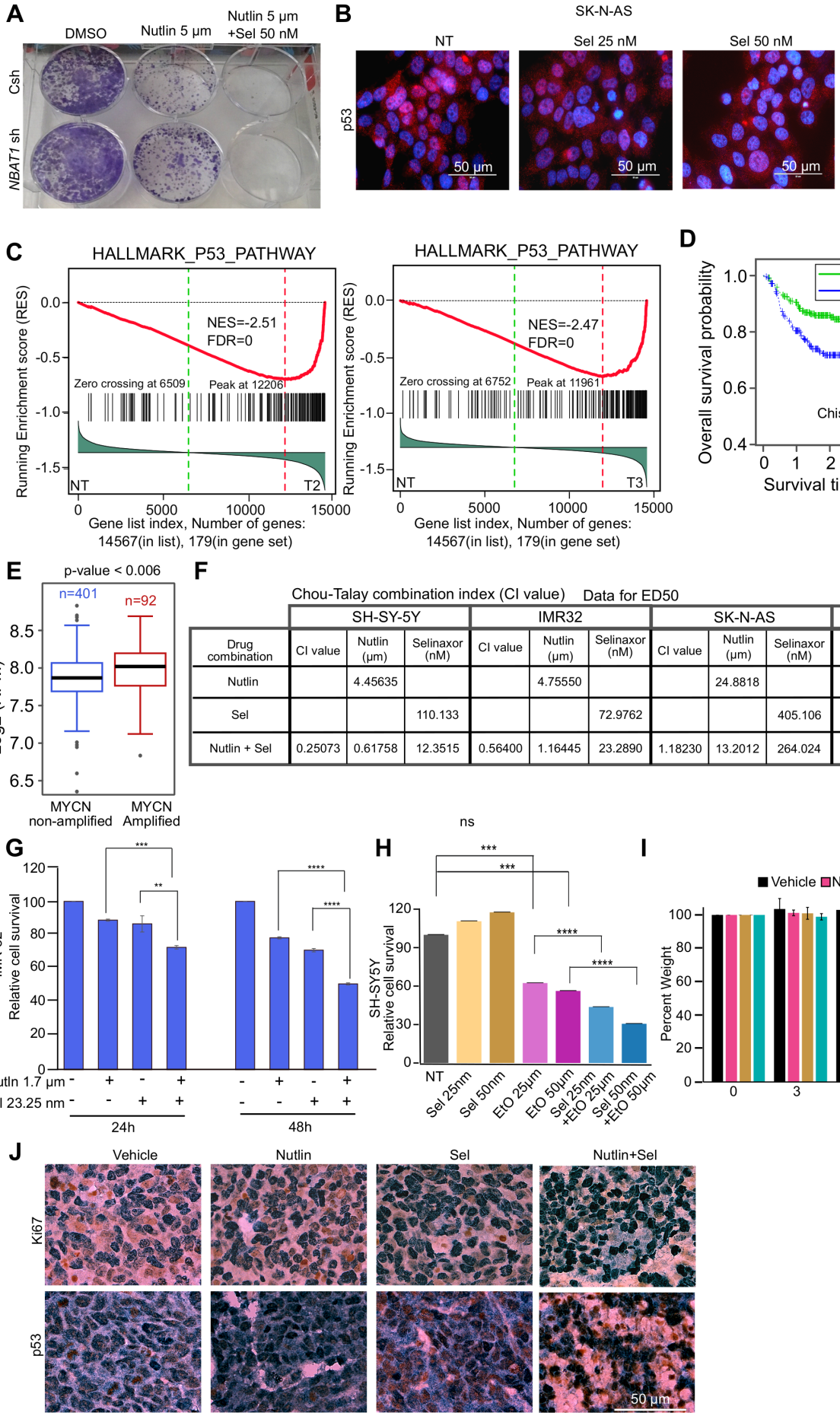
Supplementary Fig. S4. A) Cellular localization of the GFP tagged p53 (GFP-p53) in Csh and *NBAT1* sh cells. Ectopic expression of *NBAT1* (*NBAT1*ov) in *NBAT1* sh cells restores the nuclear localization of GFP-p53. Csh and *NBAT1* sh cells transfected with control vector serve as a control. **B)** p53 immunofluorescence (IF) images of Csh and *NBAT1* sh SH-SY5Y cells, ectopically expressing *NBAT1*, and treated with Nutlin (2.5 µM). *NBAT1* overexpression in *NBAT1* sh cells restores the nuclear localization of endogenous p53. Csh and *NBAT1* sh cells transfected with vector control serve as a control. **C)** Cellular localization of the GFP tagged p53 (GFP-p53) in SK-N-AS cells transfected with either *NBAT1* plasmid (*NBAT1*ov) or with vector control.

Supplementary Fig. S5



Supplementary Fig. S5. A-B) Immunoblots showing CRM1 protein levels in cells (SH-SY5Y and IMR-32) harboring Csh, *NBAT1* sh, *NBAT1*ov or control vector (A). Immunoblot on the right side shows CRM1 levels in SK-N-AS cells with *NBAT1*ov or control vector (B). GAPDH was used as a loading control in both (A) and (B). **C)** CRM1 and p53 IF on Csh and *NBAT1* sh SH-SY5Y cells treated with Nutlin (2.5 μM). *NBAT1* sh cells were transiently transfected with two different *CRM1* siRNAs which restores the nuclear localization of the endogenous p53. Csh and *NBAT1* sh cells transfected with control siRNA (Csi) serve as control. *CRM1* knockdown was visualized by IF in the *CRM1* knockdown cells. **D)** Box plot showing the expression of *NBAT1*, quantified using RNA-seq, in high CRM1 (N=8) and low CRM1 (N=4) NB tumor groups, stratified according to CRM1 immunostaining. **E)** Representative immunohistochemistry images showing CRM1 staining in NB tumors. **F)** Immunoblot showing level of CRM1 protein in Csh and *USP36*sh cells treated with either DMSO or MG132. GAPDH was used as a loading control.

Supplementary Fig. S6



Supplementary Fig. S6. A) Colony forming assay performed using Csh and *NBAT1* sh SH-SY5Y cells treated with DMSO, Nutlin or Nutlin and Selinexor (Sel) combination. DMSO was used as a control. **B)** p53 immunostaining in SK-N-AS cells after treatment with Sel with the indicated concentrations. **C)** GSEA analysis of RNA-seq data from IMR-32 cells treated with Nutlin and Sel combination or Sel alone. Left panel: GSEA enrichment plot showing the *p53* pathway on treatment with Nutlin and Sel with significant enrichment score of -2.51. Right panel: GSEA enrichment plot showing the enrichment of *p53* pathway on treatment Sel with significant enrichment score of -2.47. X-axis shows the ranked gene list in *p53* pathway and Y-axis show the enrichment score. **D-E)** *CRM1* expression in *MYCN* amplified (MYCN, n=92) and non-amplified (Non-MYCN, n= 401) NB tumors (n=493) (D). Kaplan-Meier (KM) plots showing overall survival in NBs (n=493) between the low- and high-expression groups of *CRM1*. *CRM1* expression was separated based on median expression level of *CRM1*in RNA-seq data. **F)** Chou-Talalay combination index for SH-SY5Y, IMR-32, SK-N-AS and SK-N-BE(2) cells at ED50, treated with Nutlin, Sel, or Nutlin in combination with Sel. Chou-Talalay index for the combination treatment was 0.25 and 0.56 for SH-SY5Y and IMR-32 cells, respectively, indicating the synergy between Nutlin and Sel treatments. Chou-Talalay index was 1.18 and 8.89 for SK-N-AS and SK-N-BE2 cells, respectively, indicating lack of synergy between Nutlin and Sel in these cells. **G)** Relative cell survival in IMR-32 cells was measured by MTT assay following treatment with Nutlin, Sel, or Nutlin in combination with Sel using the ED50 concentration of the individual drug or in combination, calculated using Chou-Talalay combination index as described in (F). **H)** Relative cell survival was measured using MTT in SH-SY5Y cells treated with the indicated concentrations of Sel, etoposide (Eto) or combination of both. **I)** Bar graph shows the mean percent weight loss of NSG mice used for four treatment group across the treatment days as indicated in Figure 6I. Mean weight loss is plotted with error bars representing SD. **J)** Immunohistochemistry (IHC) images showing Ki67 and p53 staining in representative xenograft tumors from the four treatment groups as described in Fig. 6I. Scale bars, 50 μm.