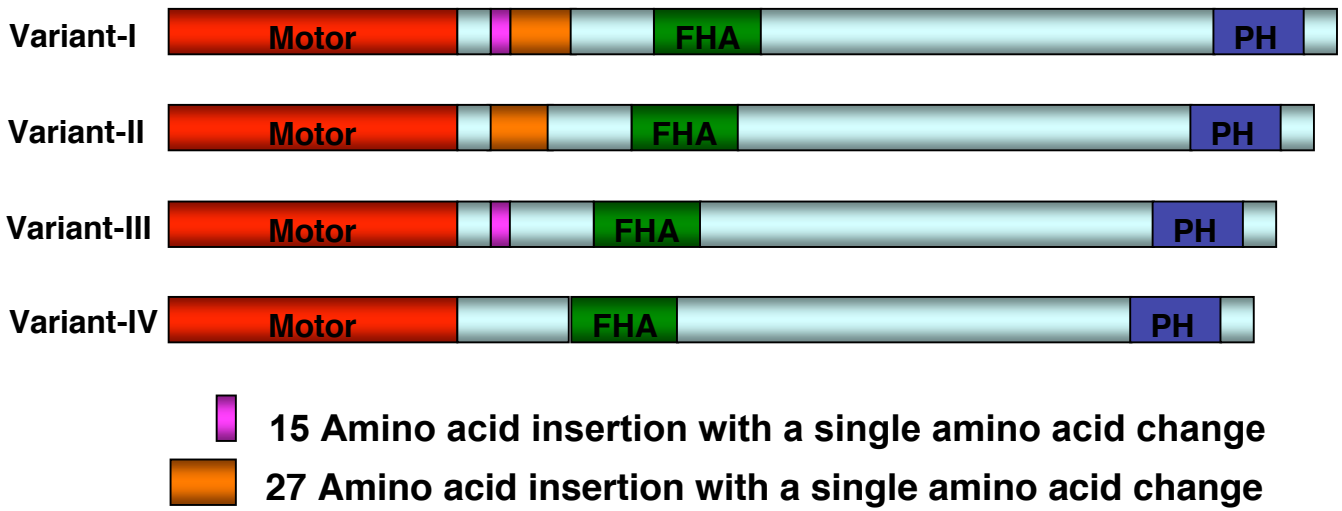
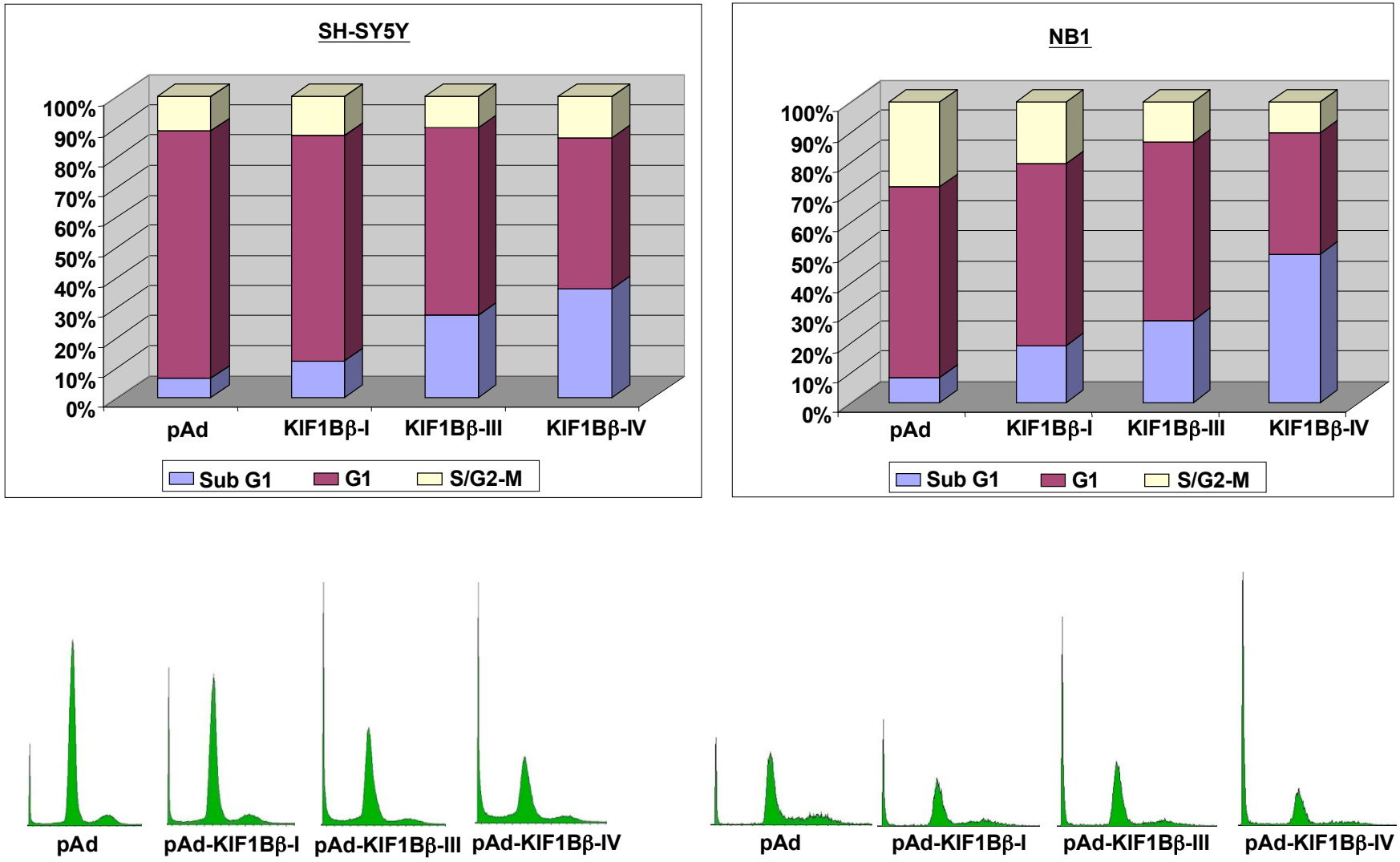
SUPPLEMENTARY FIGURE S1. Schematic drawing of the splicing variants of KIF1Bβ. NH_2-terminal Motor domain (Motor), central FHA domain (FHA) and COOH-terminal PH domain (PH) are indicated.

Munirajan *et al.*, Supplementary Figure S1



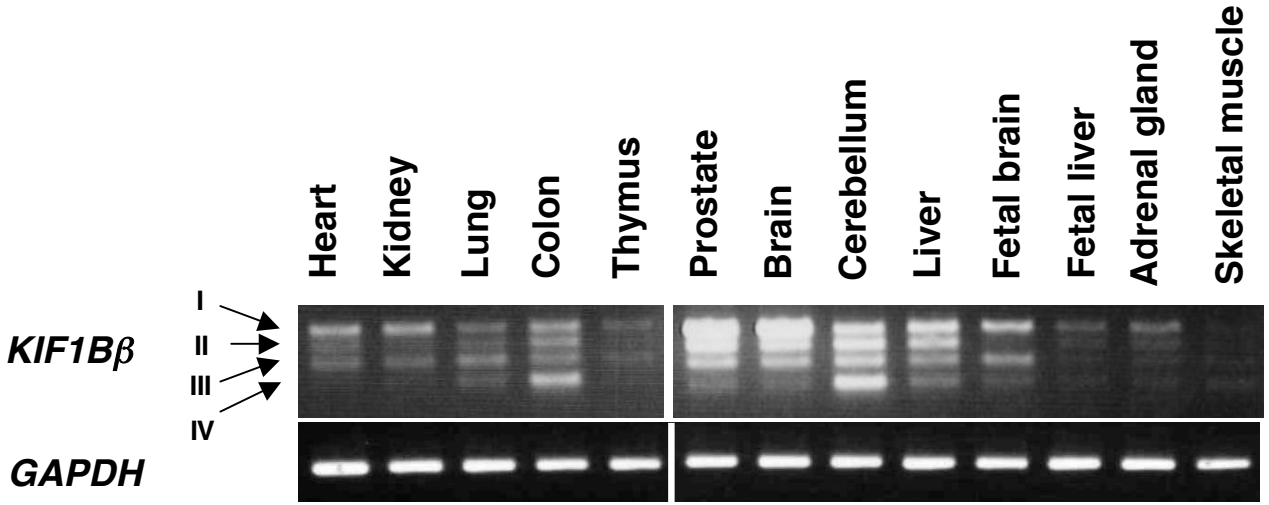
SUPPLEMENTARY FIGURE S2. KIF1Bβ has a pro-apoptotic activity *in vitro*. SH-SY5Y and NB1 cells were infected with an empty adenovirus or with the indicated adenoviruses encoding *KIF1Bβ* splicing variants. Seventy-two hours after infection, floating and attached cells were collected and their cell cycle distributions were examined by FACS analysis.

Munirajan *et al.*, Supplementary Figure S2



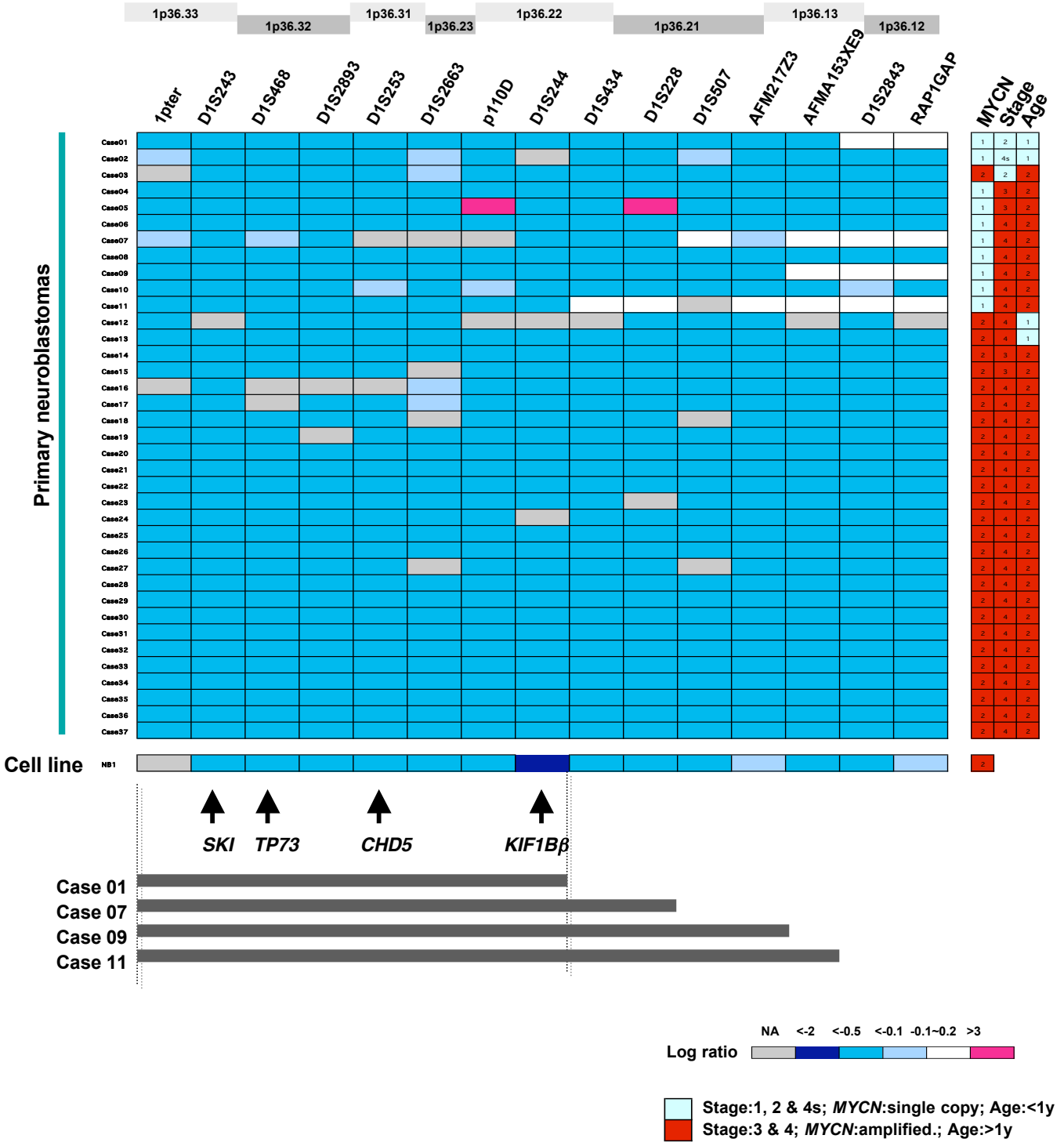
SUPPLEMENTARY FIGURE S3. Expression of *KIF1Bβ* splicing variants in normal human tissues. Total RNA was prepared from the indicated human normal tissues and subjected to RT-PCR to examine expression levels of splicing variants of *KIF1Bβ. GAPDH* was used as an internal control.

Munirajan *et al.*, Supplementary Figure S3



SUPPLEMENTARY FIGURE S4. Deletion mapping of chromosome 1p36 region based on array-CGH analysis using 112 primary neuroblastomas. Among 112 sporadic neuroblastomas examined, 37 primary tumors displayed clear genomic losses at 1p36 region (33). Normalized tumor-normal log ratio in each spot on the UCSF BAC array is schematically indicated by dark blue (log ratio <-2, loss), blue (log ratio <-0.5, loss), light blue (log ratio <-0.1, loss), white (log ratio -0.1~0.2, no loss or gain) and pink (log ratio >3, gain). Each column indicates BAC clone on the UCSF BAC array with representative STS or gene marker. The result obtained from neuroblastoma-derived cell line NB1, whose *KIF1Bß* locus is homozygously deleted, is also indicated (*left panel*). *Right panel* indicates clinical status of each sample: *MYCN* amplified (*red*); *MYCN* single copy (*light blue*); INSS stage 3 or 4 (*red*); stage 1 or 2 or 4s (*light blue*); age at diagnosis $\geq$1 year (*red*); <1 year (*light blue*). Most of tumors had large allelic losses at 1p36, while four tumors (case 01, case 07, case 09 and case 11) had relatively smaller terminal deletions which contribute to narrowing down the smallest region of deletion from *D1S434* to telomere.

Munirajan *et al.*, Supplementary Figure S4



SUPPLEMENTARY FIGURE S5. Enforced expression of KIF1Bβ causes the accumulation of cells with G2/M DNA content. COS7 cells were transiently transfected with GFP-tagged KIF1Bβ-IV, KIF1Bβ-IV-Del2 or with KIF1Bβ-IV-Del3 expression vectors. Forty-eight hours after transfection, cell cycle distributions of the attached cells (*left panels*) and the GFP-positive attached cells (*right panels*) were examined by FACS.

Munirajan *et al.*, Supplementary Figure S5