Supporting Online Material

Figure Legends

Table S1. Cytogenetic analysis of AIMP3$^{+/-}$ stable cells transformed with c-Myc or H-Ras. Ras-induced AIMP3$^{+/-}$ stable cells demonstrated a tetraploid clone with loss of X chromosome, gain of chromosome 18 and deletion in chromosome 3. Myc-transformed AIMP3$^{+/-}$ cells showed a hypotetraploid clone with up to four chromosomes 4 with a deletion with breakpoint probably in 4A5, although it is also possible that this is not a terminal deletion, but an interstitial deletion with breakpoints A2D3. Furthermore, different dicentric chromosomes, all of them derived from chromosome 4, were detectable. The breakpoints of these dicentrics also seem to involve breakpoint 4A5 with different partner chromosomes, i.e. 10, 11, 15, or 17. Representative karyograms of Ras- or Myc-transformed AIMP3$^{+/-}$ stable cell line are shown below. Abbreviations: Del (deletion), Dic (dicentric), T (translocation), Der (derivative), mar (maker), cp (composite karyotype)

Figure S1 Growth factor-dependent induction and nuclear localization of AIMP3 and genotyping of AIMP3 MEFs. *A,* HCT116 cells were treated with IGF-1 or VEGF-1 as described in Methods, and the cellular distribution of AIMP3 was monitored by

immunofluorescence staining with anti-AIMP3 antibody. *B,* The genotypes of MEF cells were determined by PCR of the genomic DNAs with the AIMP3-specific primers as previously described (9). While the wild type cells generated only one PCR product, the AIMP3 heterozygotic cells produced additional product that was from the allele in which AIMP3 gene was disrupted by the insertion of gene trap.

Figure S2 The effect of MAPK inhibitors on the H-Ras-dependent induction of AIMP3 and p53. 293 cells were transfected with H-Ras and treated with U0126, I-JNK (JNK inhibitor), and SB203580 to block Erk, JNK and p38 MAPK, respectively and the expression levels of AIMP3 and p53 were determined by Western blotting.

Figure S3 Characteristics in cell division, nuclear structures and chromosomal segregation of the AIMP3$^{+/-}$ stable cells. *A,* Colony formation of the AIMP3$^{+/+}$ and AIMP3$^{+/-}$ MEFs transfected with the indicated oncogenes was determined by Giemza staining. *B,* Each of the selected stable cell lines was cultivated in the complete media and the proliferation of each cell line was determined by cell counting. *C,* The nuclear morphology and size were monitored by PI staining. The nuclei of the AIMP3$^{+/-}$ stable cell lines are enlarged and generate satellite structures (arrows). *D,* The satellite nuclei

generated by nuclear fragmentation are clearly observed in AIMP3$^{+/-}$ cells transformed by Ras. *E*, The abnormality in mitotic chromosomal alignment and segregation was monitored by PI staining. Mitotic spindles were stained with anti-tubulin antibody (green).

Figure S4 Chemical-dependent induction of Chk1 and Chk2 in the AIMP3 wild type and heterozygous stable cell lines. While Chk1 and Chk2 were induced by nocodazole and aphidicolin in the wild type stable cell line as expected, their activation was not observed in the AIMP3 heterozygous stable cells. Notice that p53 is constitutively activated in the Ras+Myc-transformed AIMP3 wild type cells.