Supporting Information Available

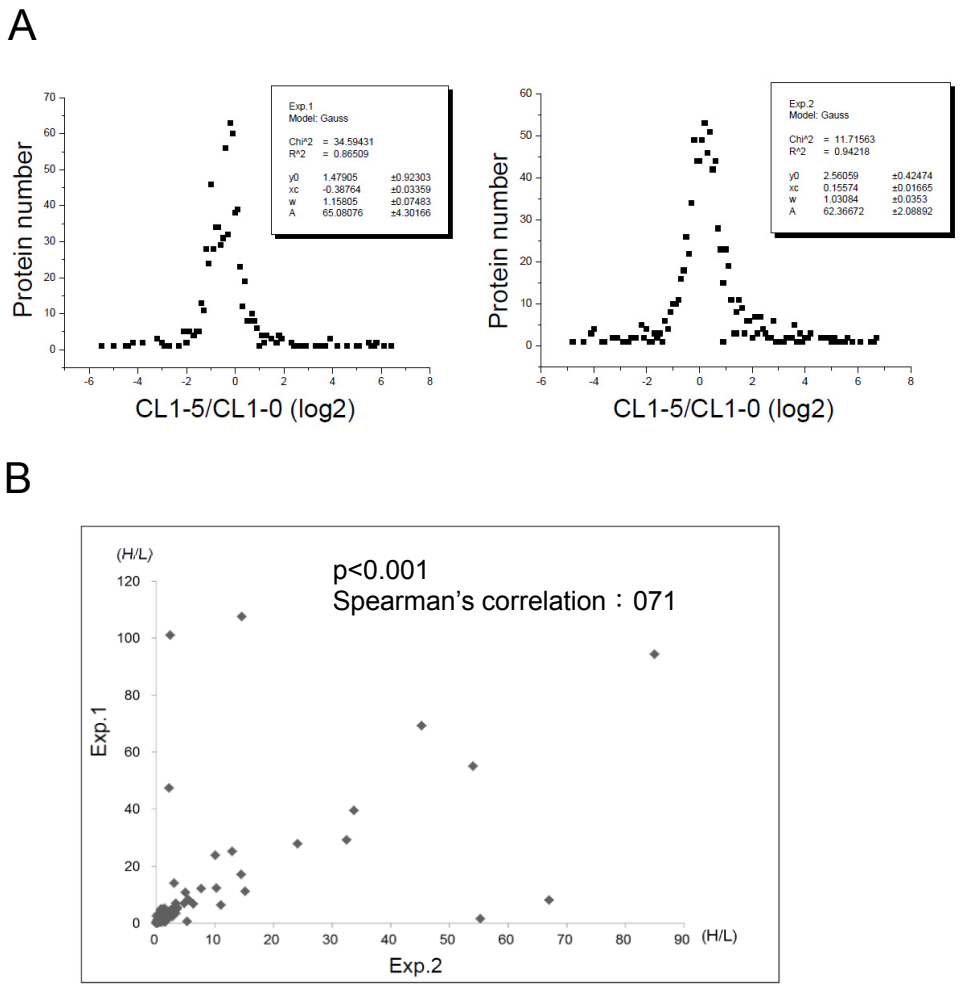
Supplemental Figure S1. The ratio distributions and correlation of the quantified protein levels between Exp. 1 and Exp. 2.

Supplemental Figure S2. Detection of vimentin by immunofluorescence staining with a methanol fixed method.

Supplemental Figure S3. KPNA2 knockdown does not change the level of pMEK.

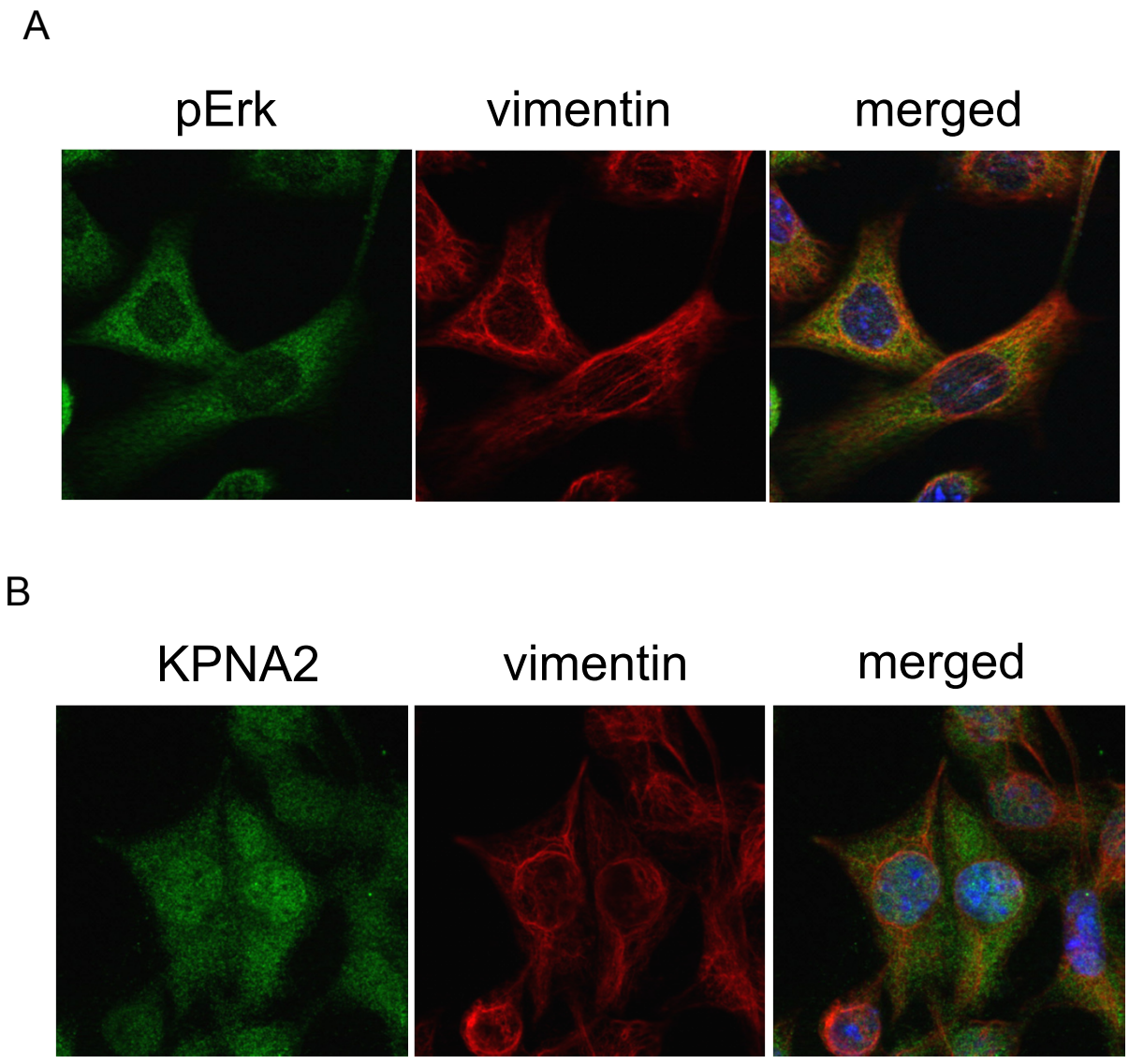
Supplemental Figure S4. Treatment of cells with a low dose of a DUSP inhibitor restores the pErk level in KPNA2-knockdown cells.

Supplemental Figure S1



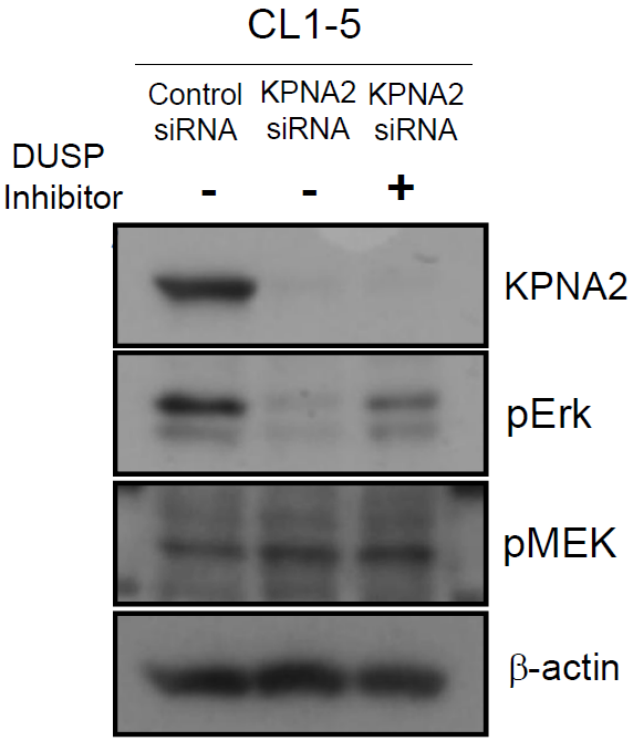
Supplemental Figure S1. The ratio distributions and correlation of the quantified protein levels between Exp. 1 and Exp. 2. (A) The log2 ratio distributions of quantified proteins were fitted with a Gaussian function using Origin 6.0 software (OriginLab Corp., Northampton, MA). The mean (xc) and two standard deviation (w) values were calculated using the following formula: $y = y_0 + (A/\sqrt{\pi W^2/2})e(-2((x-xc)/w)^2)$. (B) The reliability of protein quantification was examined between the two experiments. The linear correlation of the quantification results in Exp. 1 and Exp. 2 was demonstrated by the correlation coefficient of 0.71. A p value of less than 0.05 indicates statistical significance according to Spearman's correlation coefficient analysis.

Supplemental Figure S2



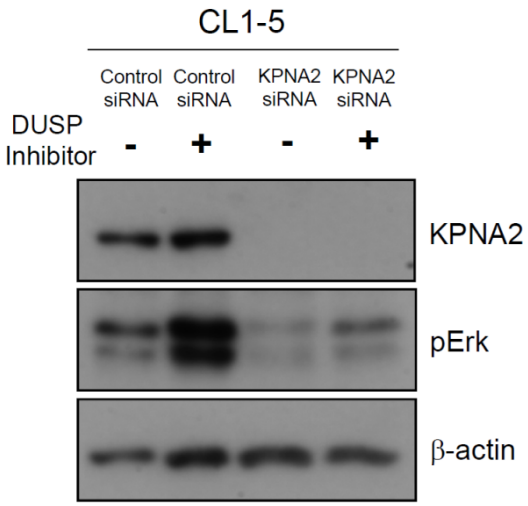
Supplemental Figure S2. Detection of vimentin by immunofluorescence staining with a methanol fixed method. CL1-5 cells were fixed by cold methanol for 3 min followed by dual labeling with anti-vimentin and anti-pErk antibodies (A), or anti-vimentin and anti-KPNA2 antibodies (B) to detect the expressions of KPNA2, pErk and vimentin as indicated.

Supplemental Figure S3



Supplemental Figure S3. KPNA2 knockdown does not change the level of pMEK. CL1-5 cells were transfected with control siRNA or KPNA2 siRNA, followed by treatment with DUSP inhibitor (10 μM) for 15 min. After treatment, the cells were lysed and analyzed via Western blot using antibodies against KPNA2, pErk and pMEK as indicated. β-actin was used as an internal control.

Supplemental Figure S4



Supplemental Figure S4. Treatment of cells with a low dose of a DUSP inhibitor restores the pErk level in KPNA2-knockdown cells. To reduce the cell toxicity caused by DUSP treatment followed by the long-term MTT assay, the CL1-5 cells transfected with control siRNA or KPNA2 siRNA were treated with a DUSP inhibitor (0.25 μM) for 15 min. After treatment, the cells were lysed and analyzed via Western blot using anti-KPNA2 and anti-pErk antibodies as indicated. β-actin was used as an internal control.