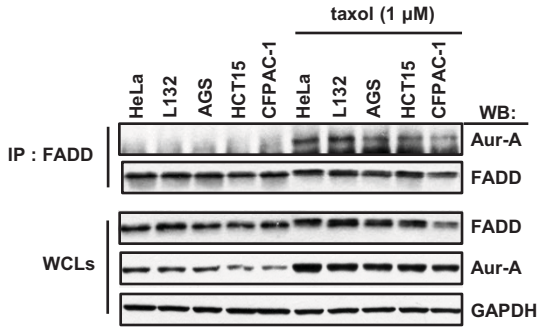
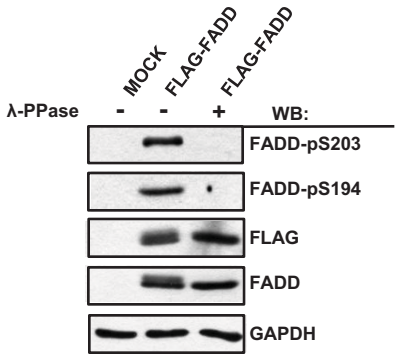
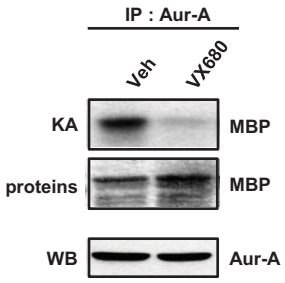


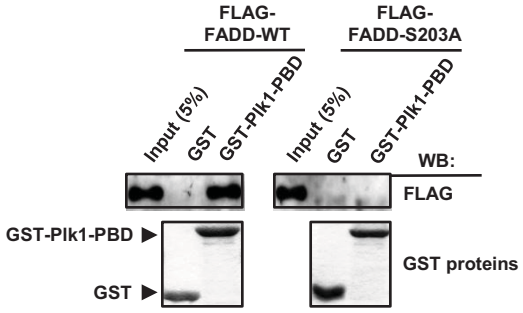
Supplementary Figure S1. Taxol stimulates the interaction between FADD and Aur-A in various cell lines. Indicated cells were left untreated or treated with taxol for 12 h. Immunopricipitated FADD and whole-cell lysates (WCLs) were immunoblotted with the indicated antibodies. L132 (normal lung cell line); AGS (gastric cancer cell line); HCT15 (colonal cancer cell line); CFPAC-1 (pancreatic cancer cell line).



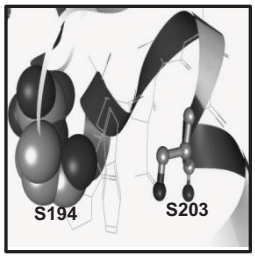
Supplementary Figure S2. S194 and S203 phosphorylation levels were decreased in lysates treated with λ-PPase. Lysates from HeLa cells treated with taxol (1 μM, 12 h) were incubated for 1 h without or with 1,000 units of λ-PPase at 30°C and analyzed by Western blotting (WB) with the indicated antibodies.



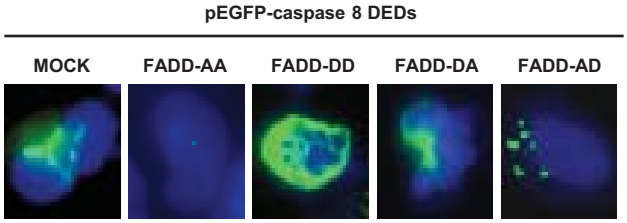
Supplementary Figure S3. VX680 inhibits the kinase activity of Aur-A. HeLa cells were pretreated with DMSO (Veh) or VX680 (3 μM) and then incubated with taxol (1 μM) for 12 h. Purified Aur-A from these lysates was incubated with MBP, a substrate of Aur-A, in the presence of [γ-^{32}P]ATP. Proteins were resolved by SDS-PAGE and visualized by autoradiography (upper) or Ponceau S staining (middle). The equivalence of proteins in immunoprecipitates (IP) was confirmed by Western blotting with an anti-Aur-A antibody.



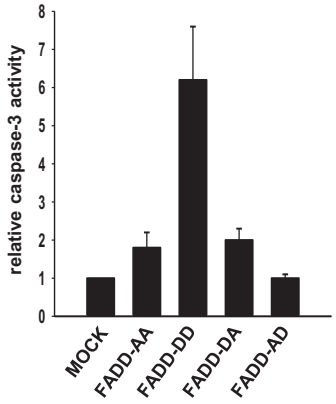
Supplementary Figure S4. Phosphorylation of FADD at S203 is required for the interaction with Plk1-PBD. HeLa cells were transfected with FLAG-tagged FADD-WT or FADD-S203A. Thirty-six hours after transfection, cell lysates were pulled down using GST or GST-Plk1-PBD and analyzed with an anti-FLAG antibody (top). The amount of total protein was determined by Ponceau S staining (bottom).



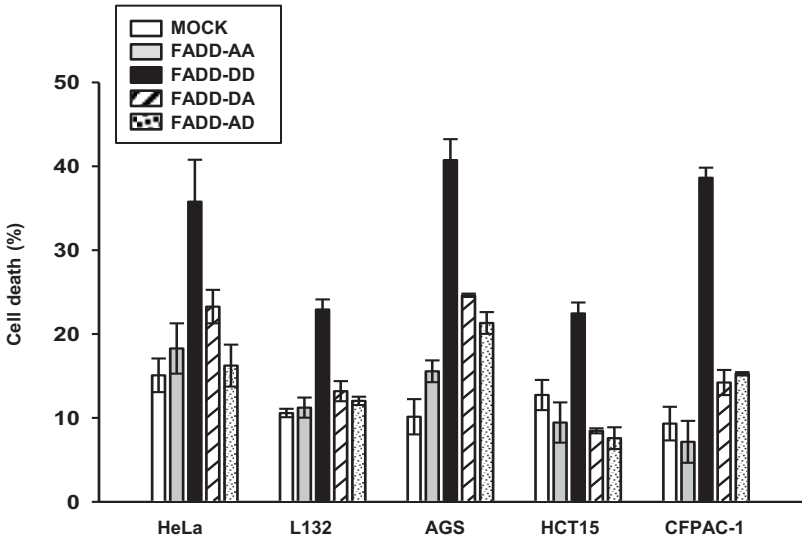
Supplementary Figure S5. Structure of FADD-pS194. A 3D structure obtained by homology modeling showing the phosphorylation sites of FADD in the phosphorylated state. Structural modifications were not predicted to occur at the S203 residue of FADD.



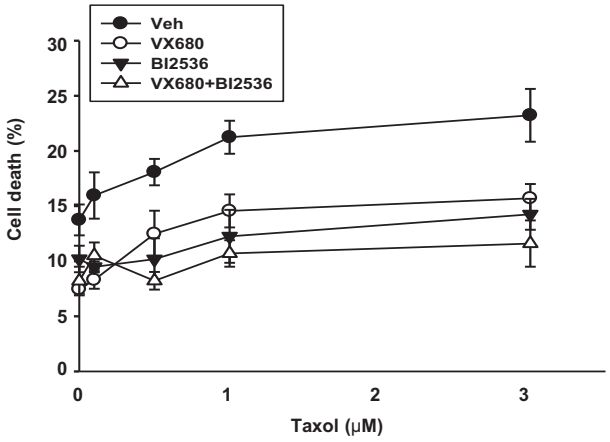
Supplementary Figure S6. dP-FADD induces caspase-8-mediated DEF formation. HeLa cells were transfected with the indicated plasmids together with pEGFP-caspase-8-DEDs, and subsequently stained with DAPI after fixation. Stained cells were visualized and photographed under a fluorescence microscope.



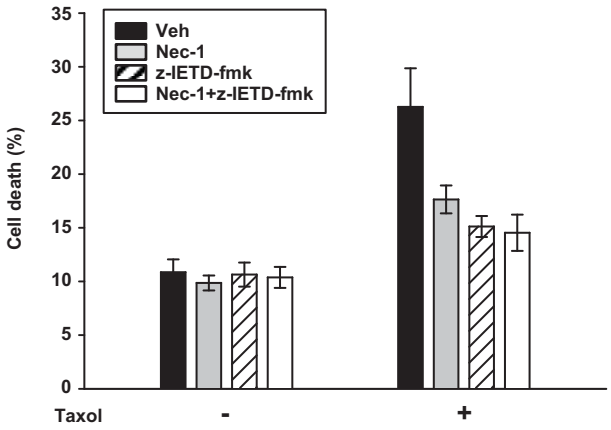
Supplementary Figure S7. dP-FADD activates caspase-3. HeLa cells transfected with FADD mutants were harvested, and equal amounts of cell lysates were used for caspase-3 assays; 2.5 mM DEVE-AMC (Peptron) was used as substrate. Caspase-3-like activity was determined by measuring relative fluorescence intensity using a spectrofluorometer.



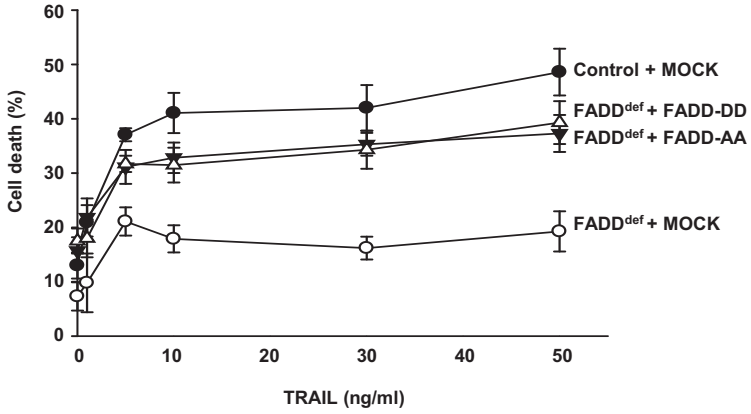
Supplementary Figure S8. dP-FADD induces cell death. Indicated cells transfected with FADD mutants and pEGFP were fixed and subsequently stained with propidium iodide (PI). The DNA content of 10,000 cells (GFP-positive cells) per sample was analyzed by flow cytometry (FACSCalibur, BD Biosciences).



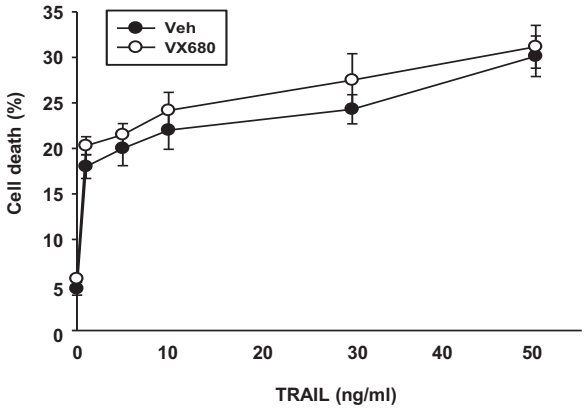
Supplementary Figure S9. Inhibition of mitotic kinases reduces sensitivity to taxol. HeLa cells were pretreated with DMSO (Veh), 3 μM VX680 or 1 μM BI2536 for 2 h and then cells were treated with taxol and mitotic kinases inhibitors for 12 h. Stimulated cells were stained with PI and analyzed by flow cytometry. VX680: Aur-A inhibitor; BI2536: Plk1 inhibitor.



Supplementary Figure S10. Activation of caspase-8 and RIP1 is required for taxol-induced cell death. HeLa cells were pretreated with DMSO (Veh), 10 μM Nec-1 (synthesized at the Korea Research Institute of Chemical Technology) or 20 μM z-IETD-fmk (Calbiochem) for 2 h before addition of 1 μM taxol for 12 h. Indicated cells were fixed and stained with PI. Stained cells were analyzed by flow cytometry. Nec-1 (necrostatin-1, RIP1 inhibitor); z-IETD-fmk (caspase-8 inhibitor).



Supplementary Figure S11. Phosphorylation of FADD is not required for the TRAIL-mediated cell death. Wild-type (control) and FADD-deficient ($FADD^{def}$) Jurkat cells were transfected with MOCK, FADD-AA or FADD-DD together with pEGFP using the Nucleofector (Lonza). After 36 h, cells were treated with indicated concentrations of TRAIL for 12 h. Stimulated cells were fixed and stained with PI. The 10,000 cells (GFP-positive cells) per sample were analyzed by flow cytometry.



Supplementary Figure S12. Aur-A inhibitor does not block TRAIL-induced cell death. HeLa cells were pretreated with DMSO (Veh) or 3 μM VX680 for 2 h and subsequently treated with indicated concentrations of TRAIL for 12 h. Stimulated cells were fixed and stained with PI. DNA content per sample was analyzed by flow cytometry.