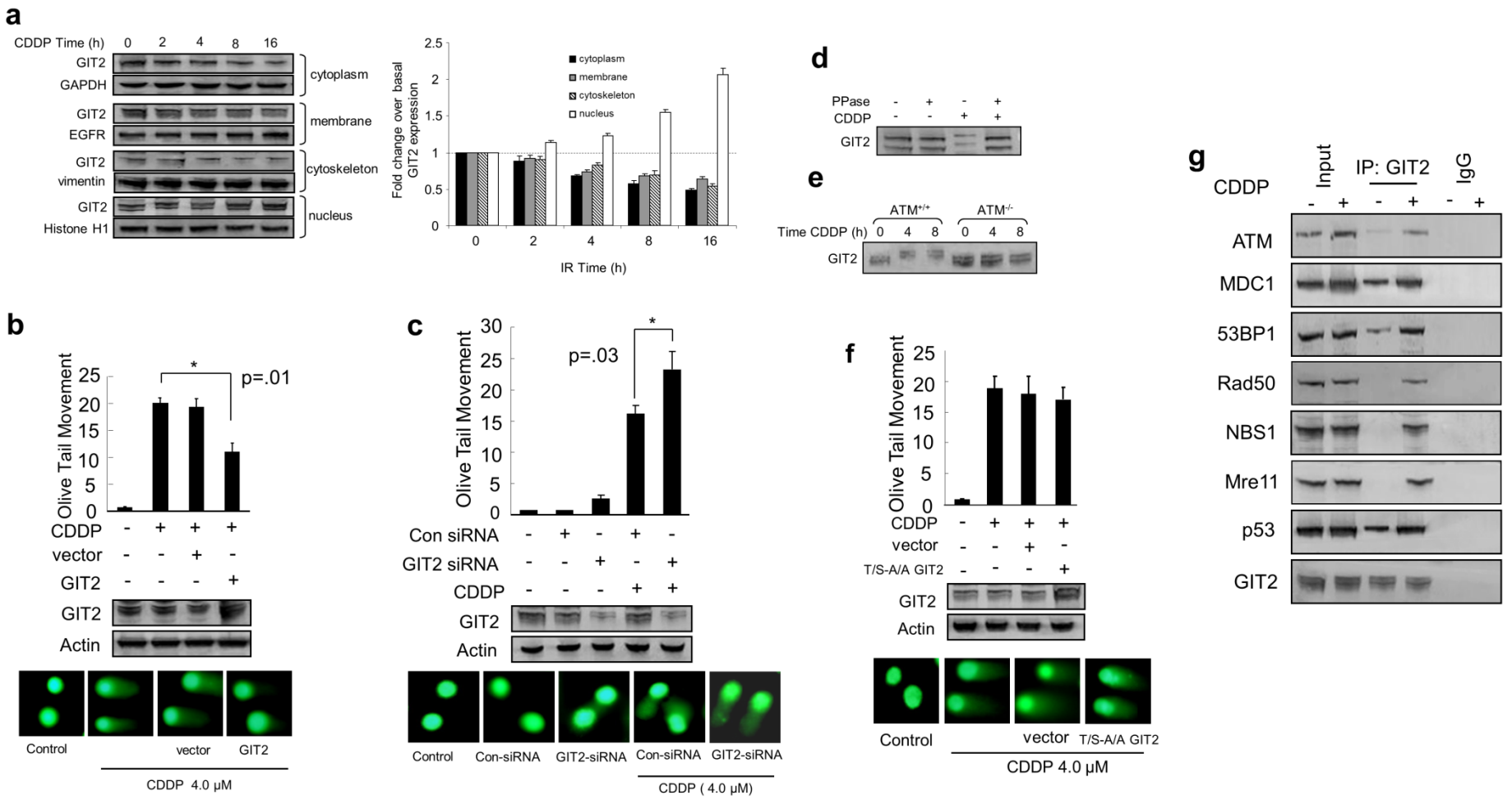

Figure S1. GIT2 is sensitive to CDDP induced DNA damage. (**a**) SH-SY5Y cells were treated with 4μM cisplatin (CDDP) for the indicated time followed by subcellular fractionation of proteins and subsequent immunoblotting (IB) analysis. The histogram depicts the relative quantitation of GIT2 expression changes in diverse cellular compartments. (**b**) Overexpression of GIT2 protects DNA from excessive CDDP induced damage indicated by neutral comet assay (100 nuclei counted for each group: data were expressed as mean ± S.E.M.). (**c**) Knockdown of GIT2 exacerbates CDDP-mediated DNA damage. (**d**) GIT2 is phosphorylated in response to DNA damage. SH-SY5Y cells were harvested immediately after CDDP treatment and cell extracts were incubated with or without λ protein phosphatase (λ-PPase) and subjected to immunoblot gel-migration analysis using GIT2 antibody. λ-PPase incubation reversed CDDP induced GIT2 gel retardation. (**e**) GIT2 gel retardation is dependent upon ATM. CDDP-treated $ATM^{+/+}$ (GM0637) and $ATM^{-/-}$ (GM5849) cells were harvested at the indicated time point and cell extracts were subjected to immunoblot gel-migration analysis using GIT2 antibody. CDDP induced GIT2 gel retardation was lost in $ATM^{-/-}$ cells. (**f**) DNA protective role of GIT2 is dependent on ATM phosphorylation sites. SH-SY5Y cells were transfected with the T/S-A/A GIT2 mutant for 24 hours before CDDP treatment, comet assay was performed immediately after treatment. Overexpression of T/S-A/A GIT2 failed to confer protection to CDDP-mediated DNA damage. (**g**) Classical DDR proteins associating with immunoprecipitated GIT2 were identified by specific IBs. Input represents 1% of total protein used in IPs.