

## Supplemental Materials

### **The intrinsically disordered C-terminal domain triggers nucleolar localization and function**

#### **switch of PARN in response to DNA damage**

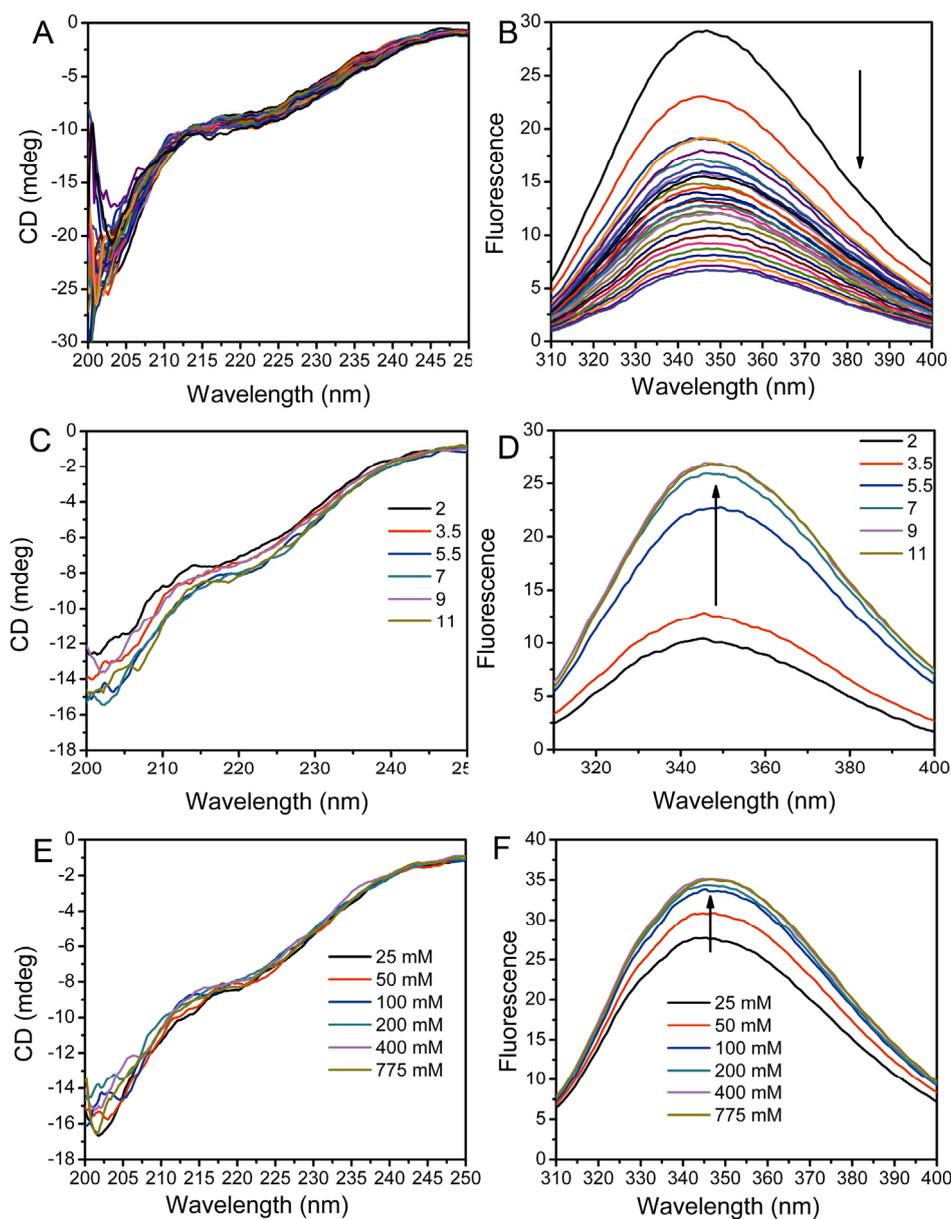
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**Running Title:** Role of C-terminal domain in PARN function



**Figure S1. Effect of temperature (A and B), pH (C and D) and K<sup>+</sup> (E and F) on PARN-CTD structural features monitored by far-UV CD (A, C and E) and Trp fluorescence excited at 295 nm (B, D and F).** Similar results were also obtained for Trp and Tyr fluorescence excited at 280 nm (data not shown). The factors did not influence PARN-CTD secondary and tertiary structures. Similar results were obtained for factors including divalent metals (Mg<sup>2+</sup>, Mn<sup>2+</sup> and Ca<sup>2+</sup>), low concentration of ureal and macromolecular crowding reagent including dextran-70 and PEG-20000 (data not shown).

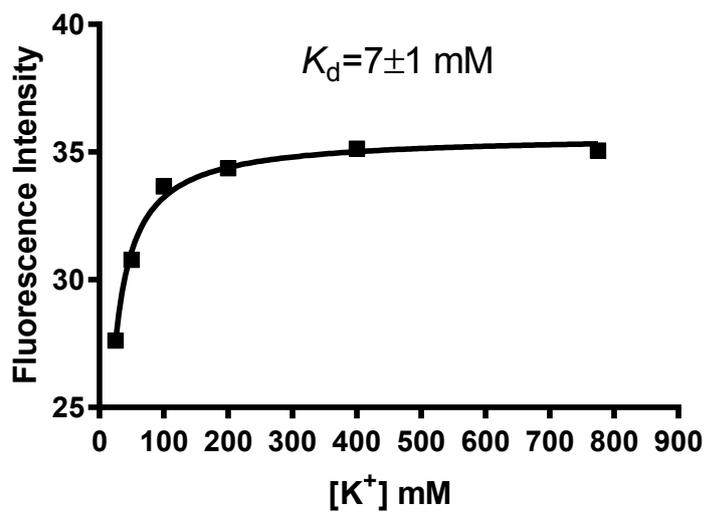
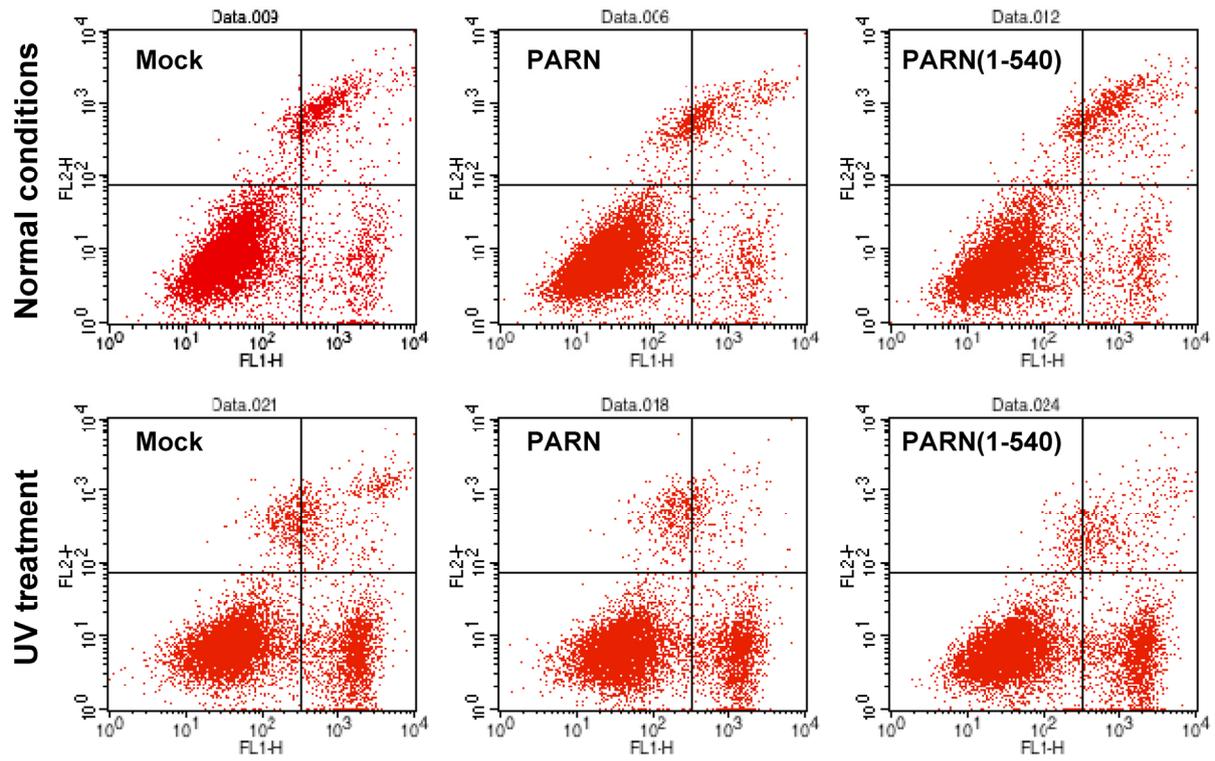
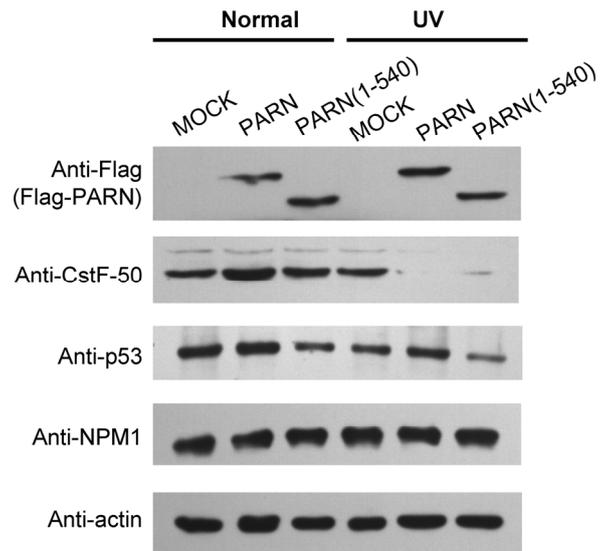


Figure S2. K<sup>+</sup> enhances PARN-CTD Trp fluorescence in a concentration-dependent manner, implying that there is specific binding of K<sup>+</sup> with PARN-CTD.





**Figure S4. Representative profiles of flow cytometry analysis of cell apoptosis determined by Annexin V-FITC binding (horizontal) and PI exclusion (vertical). The HEK-293T cells were transfected by the empty vector (MOCK), Flag-PARN or Flag-PARN(1-540).**



**Figure S5.** Western blot analysis of the protein levels of CstF-50 and p53 in untreated or UV-treated HEK-293T cells transfected with plasmids containing the full length or truncated PARN. NPM1 and actin were used as the controls.