Information for Supplemental Figures:

Supplemental Figure legends:

Supplemental Figure 1. UV-induced Rad17 proteolysis is mediated by ATR. (**A**) Mutation of ATR in human fibroblast GM18366 results in loss of checkpoint response as indicated from measurement of Chk1 phosphorylation in response to UV radiation. (B) Malfunction of ATR in GM18366 leads to significant attenuation of UV-induced Rad17 proteolysis, while Rad17 protein levels increase at early time points followed by a drop over four to six hours after exposure to UV. (C) ATR-mediated Rad17 phosphorylation is associated with its degradation after exposure to UV. Flag-Rad17 and Flag Rad17 (S635A/S635A) were transfected into HNF cells, respectively. Cells were treated with UV (10 J/M^2) and harvested at indicated time points. UV-induced alteration of ectopically expressed wilt-type or mutant Rad17 was measured by using antibody against Flag.

Supplemental Figure 2. Alteration of Rad17 protein half-life in response to UV radiation. HNF cells were pretreated for 15 min with 20 mM cycloheximide followed by UV treatment or no treatment. Rad17 protein levels drop to basal levels in two to four hours following exposure to UV, while natural Rad17 levels decade in the presence of cycloheximide occurs eight hours after treatment.

Supplemental Figure 3. Interaction between ectopically expressed Rad17 and endogenous Rad17. HNF cells were transfected with or without HA-Rad17. Cells were treated with UV. Endogenous Rad17 was pulled down using antibody against Rad17. The association of ectopically expressed Rad17 with endogenous Rad17 was tested by immunoblotting of IP complex using antibody against HA tag.