**Qiang *et al*.**

**Supplemental Figures: Fig. S1-S4**

**Fig. S1, related to Fig. 1**

**Fig. S2, related to Fig. 3**

**Fig. S3, related to Fig. 4**

**Fig. S4, related to Fig. 5**

**Supplemental Figures: legends**

**Figure S1.** Related to **Figure 1.** Autophagy deficiency inhibits nucleotide excision repair and downregulates XPC. (**A** to **F**) Slot blot analysis of CPD levels (**A**, **C**, and **E**) and quantification of CPD repair (**B**, **D**, and **F**) in MEF cells with wild-type (WT) genes or a deficiency for *Atg7* (**A** and **B**) (knockout), *Atg12* (**C** and **D**) (knockout), or *Atg14* (**E** and **F**) (cKO, Atg14fl/fl MEF cells treated with Cre-expressing retrovirus). Cells were collected at 0, 24, and 48 h post-UVB (10 mJ/cm2) for CPD analysis. (**G** to **H**) immunoblot analysis of LC3-I, LC3-II, SQSTM1, XPC, DDB2, ATG5, GAPDH (**G**), XPA, XPB, XPD, XPF, XPG, RAD23B and GAPDH (**H**) in *Atg5* WT and KO MEF cells. (**I**) immunoblot analysis of LC3-I, LC3-II, SQSTM1, XPC, DDB2, and GAPDH in HaCaT cells treated with vehicle or Spautin-1 for 24 h. Numbers in I indicate fold of XPC levels. \*, *P* < 0.05, Student t test, significant differences between vehicle and Spautin-1 groups (**I**). (**J**, **K**) Immunoblot analysis of SQSTM1, LC3-I, L3-II and GAPDH in WT and *atg5* KO iBMK cells (**J**), and WT and *atg7* KO, *atg12* KO and *Atg14* cKO MEF cells (**K**). (**L**) Autophagic flux analysis in WT and *atg5* KO MEF cells (From Qiang *et al.*[1](#_ENREF_1)). Cells were exposed to UVB (20 mJ/cm2) and incubated for 6 h or treated with rapamycin (500 nM) for 6 h with or without pretreatment with BfnA1 (50 nM) for 2 h. The levels of LC3-I, LC3-II, and SQSTM1 were analyzed by immunoblot assay.

**Figure S2.** Related to **Figure 3.** Autophagy deficiency inhibits *Xpc* transcription through the TWIST1-AKT pathway. (**A**) Schematic representation of the E-Box sites of the mouse *Xpc* promoter. Red nucleotides indicate mutations of E-Box sites made in the mouse *Xpc* promoter. (**B**) Schematic representation of the E2F site of the mouse *Xpc* promoter. Red nucleotides indicate mutations of the E2F site made in mouse *Xpc* promoters. (**C**, **D**) Slot blot analysis of the levels of CPD (**C**) and 6-4PP (**D**) in WT and *atg5* KO MEF cells treated with vehicle or the PI3K-AKT pathway inhibitor LY294002 (LY, 10 μΜ).

**Figure S3.** Related to **Figure 4.** Autophagy deficiency inhibits 6-4PP repair via decreasing XPC while it inhibits CPD repair via both decreasing XPC availability and damage recognition by DDB2.(**A**, **B**) Slot blot analysis of the levels of CPD (**A**) and 6-4PP (**B**) in WT and *atg5* KO MEF cells transfected with Con or *Xpc*. (**C**, **D**) Slot blot analysis of the levels of CPD (**C**) and 6-4PP (**D**) in *XPC-/-*-*CMV-XPC* cells transfected with Con and *MYC-TWIST1*. (**E**) Quantification of percentage (%) of 6-4PP repair in *XPC-/-*-CMV*-XPC* cells transfected with Con and *MYC-TWIST1*.

**Figure S4.** Related to **Figure 5.** Autophagy deficiency inhibits DDB2 recruitment through TWIST1 binding and inhibition of EP300.(**A**) Slot blot analysis of the levels of CPD in WT and *atg5* KO MEF cells transfected with Con or the combination of *Xpc* and *Ep300* constructs. (**B**) Schematic representation of the deletion (36 to 72) of *MYC-TWIST1*.