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Supplemental Material to:

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A role of autophagy in PTP4A3-driven cancer progression

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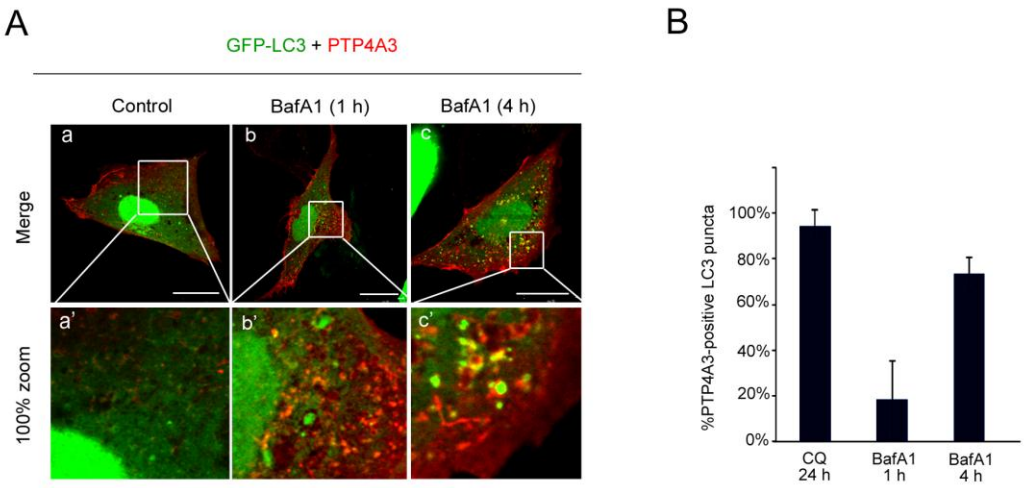

Figure S1. PTP4A3 colocalizes with LC3 puncta in BafA1-treated CHO-PTP4A3. (**A**) CHO-PTP4A3 cells transiently overexpressing EGFP-LC3B (green) were treated with BafA1 and immunolabeled with anti-PTP4A3 antibody (red). Bar: 20 µm. (**B**) Quantification of PTP4A3 and EGFP-LC3B colocalization shown as percentage of LC3 puncta positive for PTP4A3 (n = x).

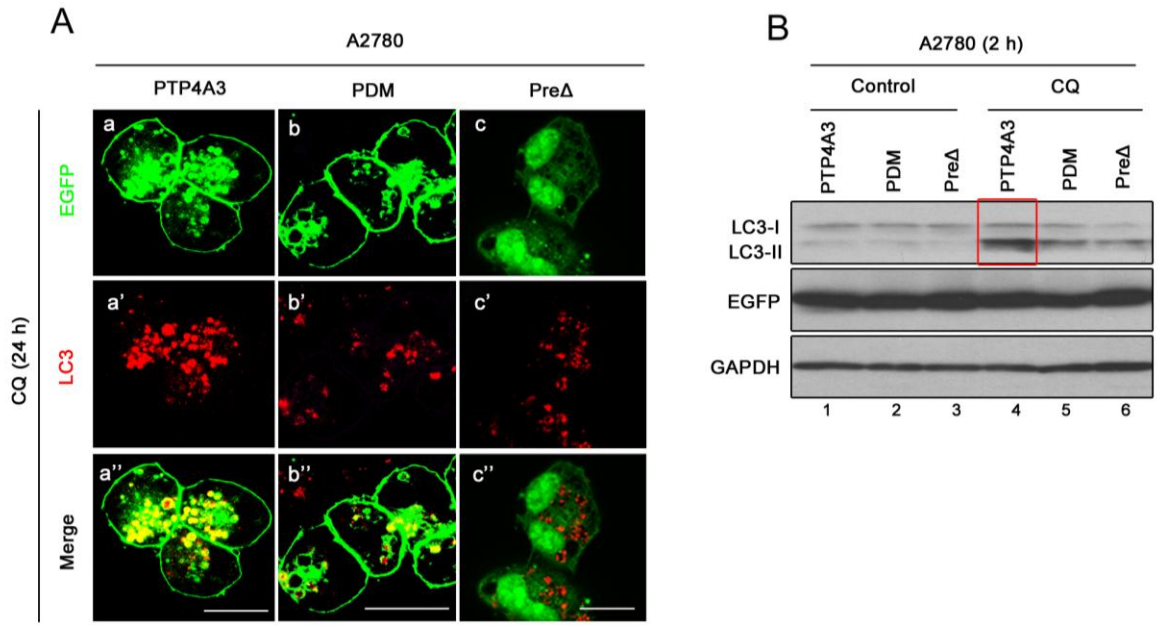

Figure S2. PDM, but not PreΔ, colocalizes with LC3 puncta upon CQ treatment, but neither PDM nor PreΔ could promote LC3 conversion compared to PTP4A3. (**A**) A2780 cells overexpressing EGFP-PTP4A3, EGFP-PTP4A3-PDM or EGFP-PTP4A3-PreΔ (prenylation mutant) were treated with CQ (50 μM) for 24 h. Endogenous LC3 was then immunolabeled with an anti-LC3 antibody. Bar: 15 μm. (**B**) Cells in (**A**) were untreated (control) or treated with CQ (50 μM) for 2 h. LC3 and EGFP levels were than analyzed by western botting. GAPDH served as loading control.

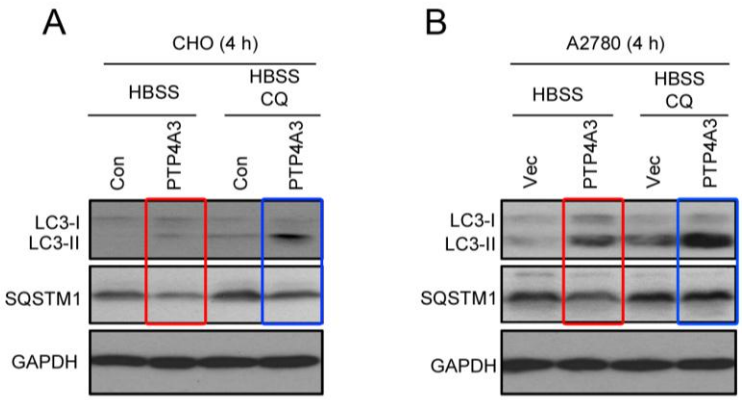

Figure S3. PTP4A3 promotes both LC3-I to LC3-II conversion and SQSTM1 degradation under HBSS starvation. (**A**) CHO-Con and CHO-PTP4A3 cells were treated as indicated before lysis for western blotting analysis. (**B**) A2780-Vec and A2780-PTP4A3 cells were treated as indicated before lysis for western blotting analysis.

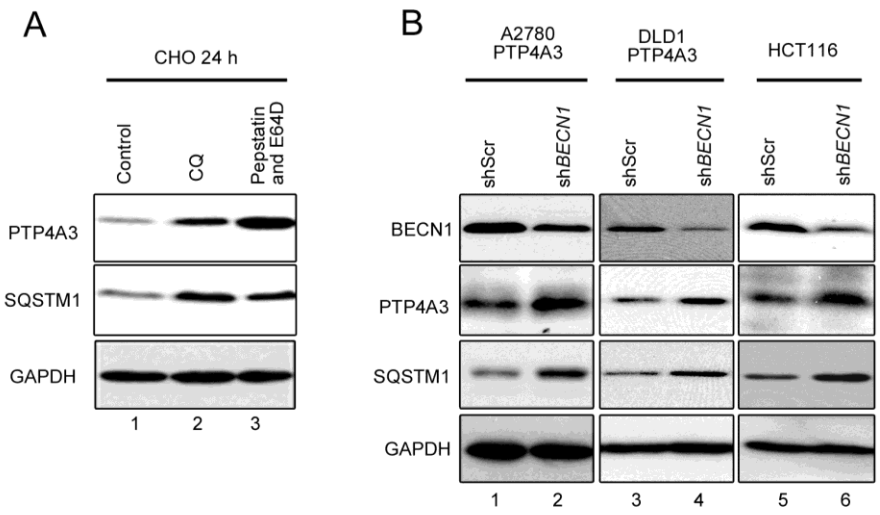

Figure S4. PTP4A3 protein accumulates in cells treated with pepstatin and E64D or upon *BECN1* knockdown. (**A**) CHO-PTP4A3 cells were untreated (Control), or treated with either CQ (50 µM) or pepstatin and E64D (final 10 µg/mL each) for 24 h. PTP4A3 and SQSTM1 expression levels were then analyzed by western blotting, and GAPDH served as a loading control. (**B**) BECN1 was knocked down using shRNA in A2780-PTP4A3, DLD1-PTP4A3 and HCT116 cells. Exponentially growing cells in full media were lysed for western blotting analysis with the indicated antibodies. GAPDH served as loading control.

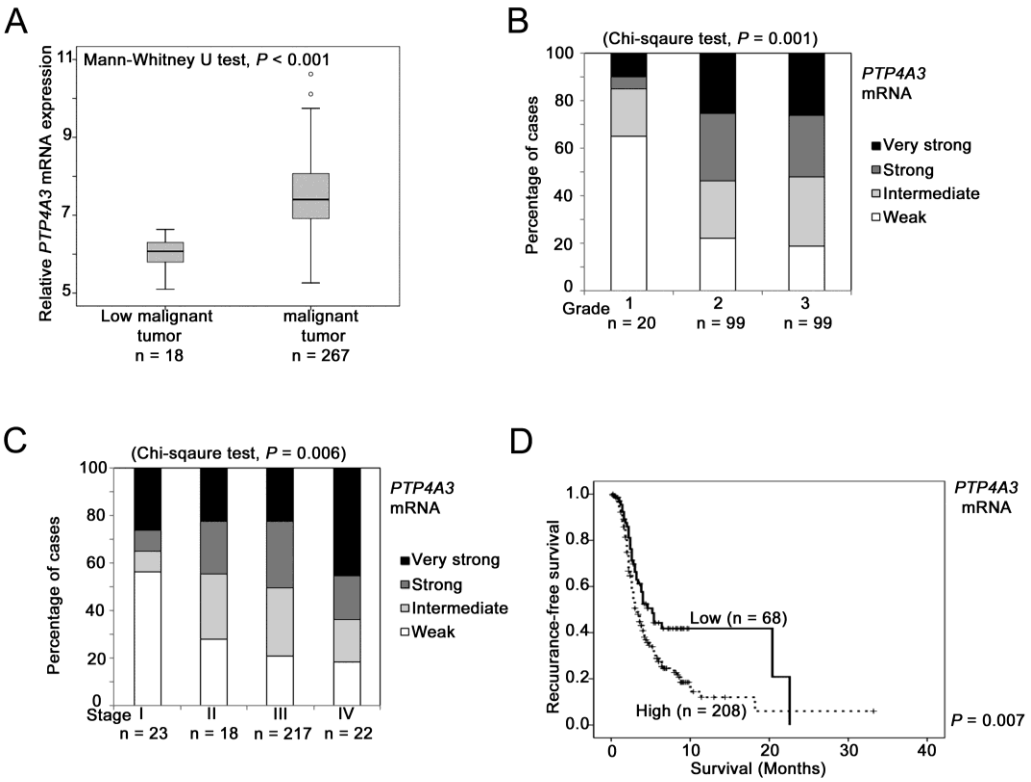

Figure S5. High *PTP4A3* mRNA expression is significantly associated with advanced tumor grade, late tumor stage and poor survival in the GSE9899 ovarian cancer cohort. (**A**) *PTP4A3* mRNA expression level was significantly higher in patients with malignant ovarian tumors compared to those with low malignant potential tumors. (**B and C**) *PTP4A3* mRNA expression was significantly higher in tumors with higher tumor grades (**B**) and stage (**C**). (**D**) A high level of *PTP4A3* mRNA expression was significantly associated with a shorter recurrence-free survival time of ovarian cancer patients.

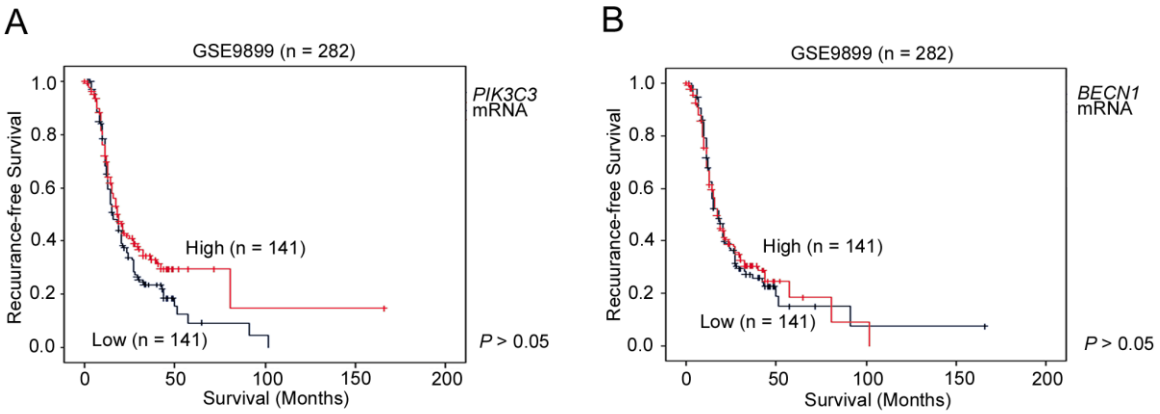

Figure S6. Autophagy genes alone have no prognostic value for recurrence-free ovarian cancer survival in the GSE9899 patient cohort. No significant prognostic value was observed for stratified (**A**) *PIK3C3* or (**B**) *BECN1* mRNA expression.

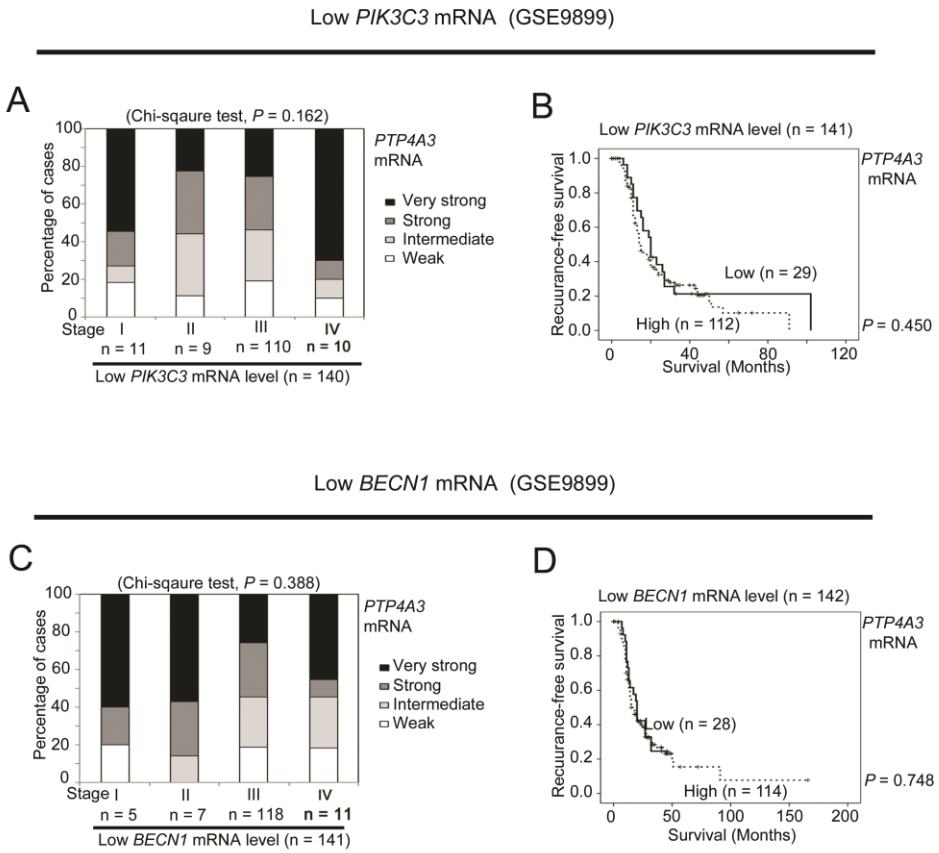

Figure S7. Low levels of autophagy genes and *PTP4A3* expression levels in ovarian cancer cohort (GSE9899). (**A**) In ovarian cancer patients expressing low levels of *PIK3C3*, *PTP4A3* expression levels were not significantly correlated with higher pathological stage. (**B**) No significant correlation between *PTP4A3* expression and recurrence-free survival in patients with low *PIK3C3* expression levels. (**C and D**) Similar results as in (**A and B**) were obtained when the tumors were stratified by *BECN1* instead of *PIK3C3*.