Supplementary Figures

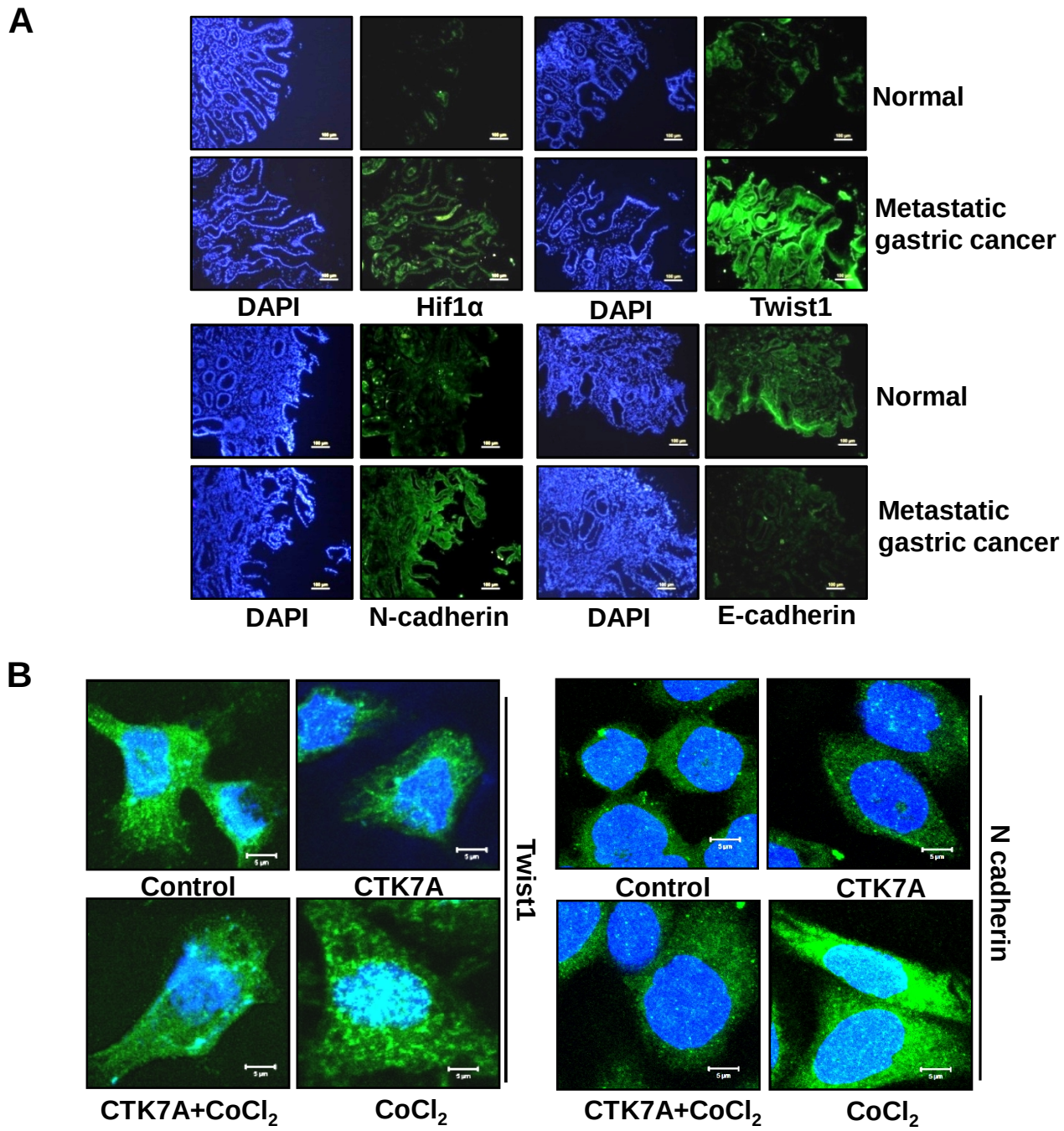
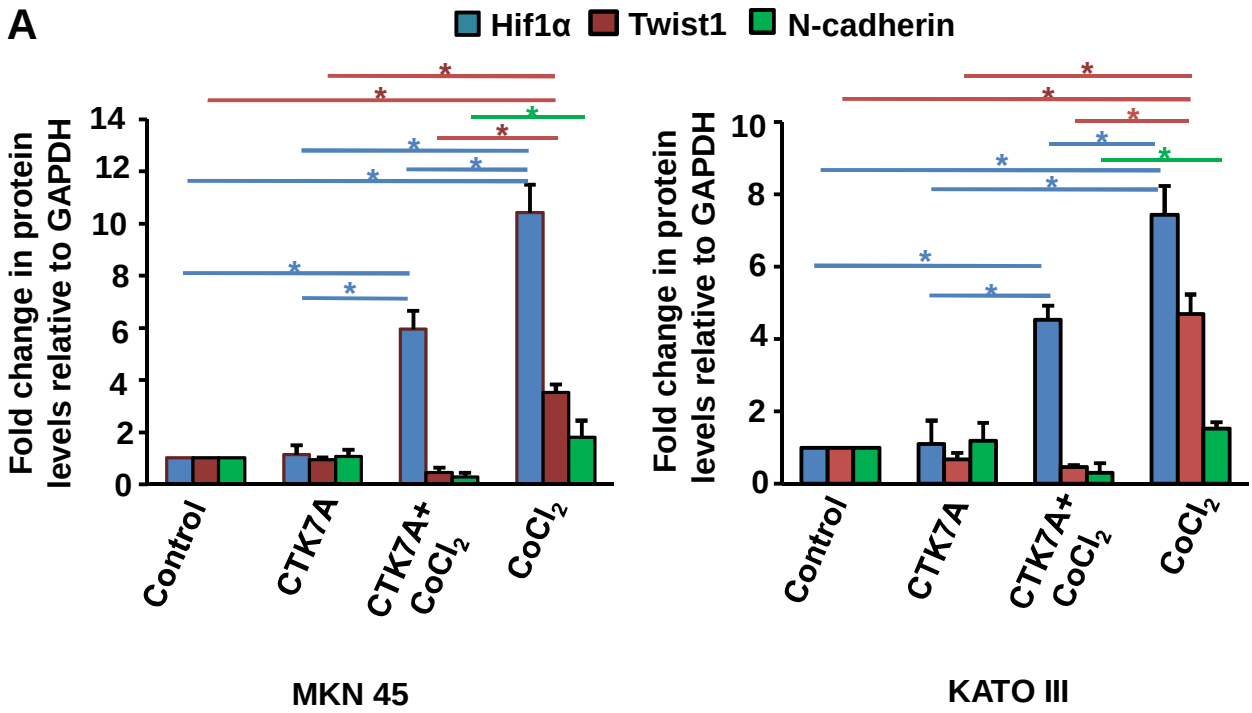


Fig. S1. *Metastatic factors expressed in metastatic gastric cancer biopsy samples and in CTK7A and/or $CoCl_2$-treated GCCs.* (A) Immunofluorescence microscopy of metastatic gastric biopsy samples and adjacent non cancerous gastric tissue samples (n=4 from each group) revealed upregulation of Hif1α, Twist1 and N-cadherin in metastatic samples as compared to paired non-cancerous antral biopsies. DAPI was used for nuclear staining. Scale bar 100 μm. (B) A representative confocal microscopy image of AGS cells treated with $CoCl_2$ (200 μM) alone or in combination with CTK7A (100 μM) showed downregulation of EMT markers Twist1 and N-cadherin in CTK7A+$CoCl_2$-treated cells. Scale bar 5 μm.



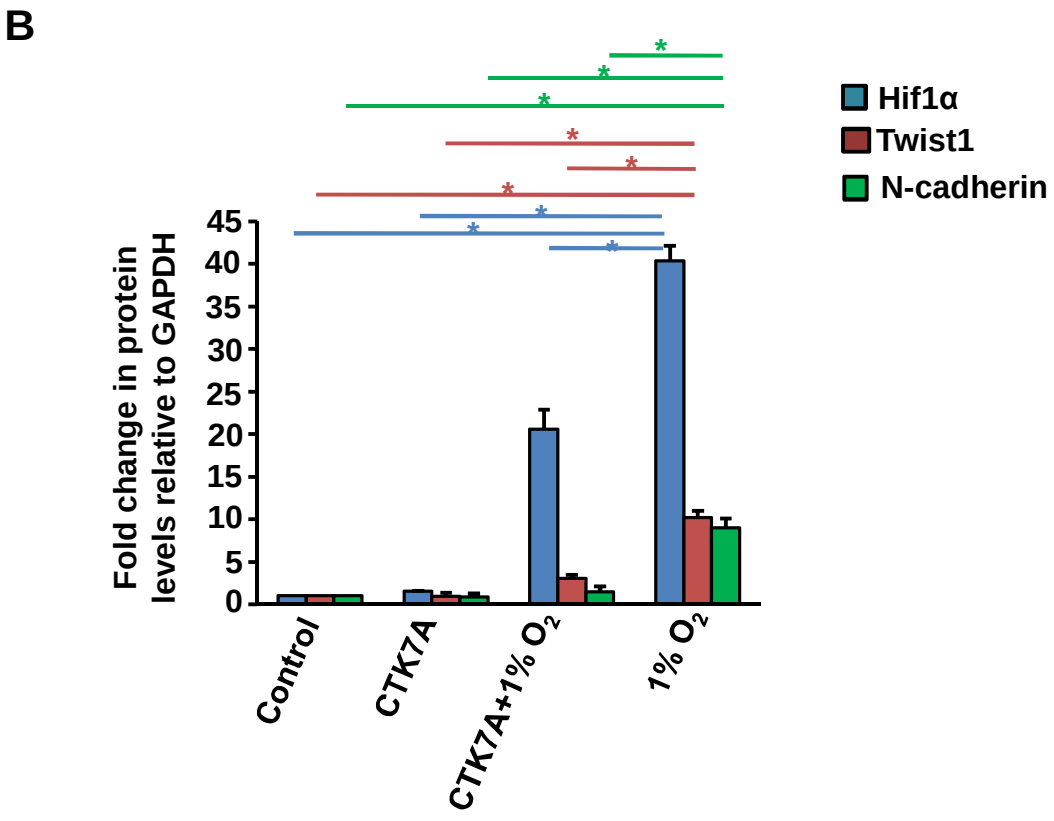


Fig. S2. *CTK7A downregulated expression of metastatic markers in hypoxic GCCs.* (A) Graphical presentation of data shown in Figure 1C for MKN 45 and KATO III cells by Student's t-test demonstrated expression pattern of metastatic markers in CTK7A and $CoCl_2$ treated cells as compared to other 3 experimental groups (n=3 independent experiments, **P*<0.05). (B) Bar diagram of Figure 1D depicted expression pattern of metastatic markers in the above experimental conditions by Student's t-test (n=3 independent experiments, **P*<0.05).

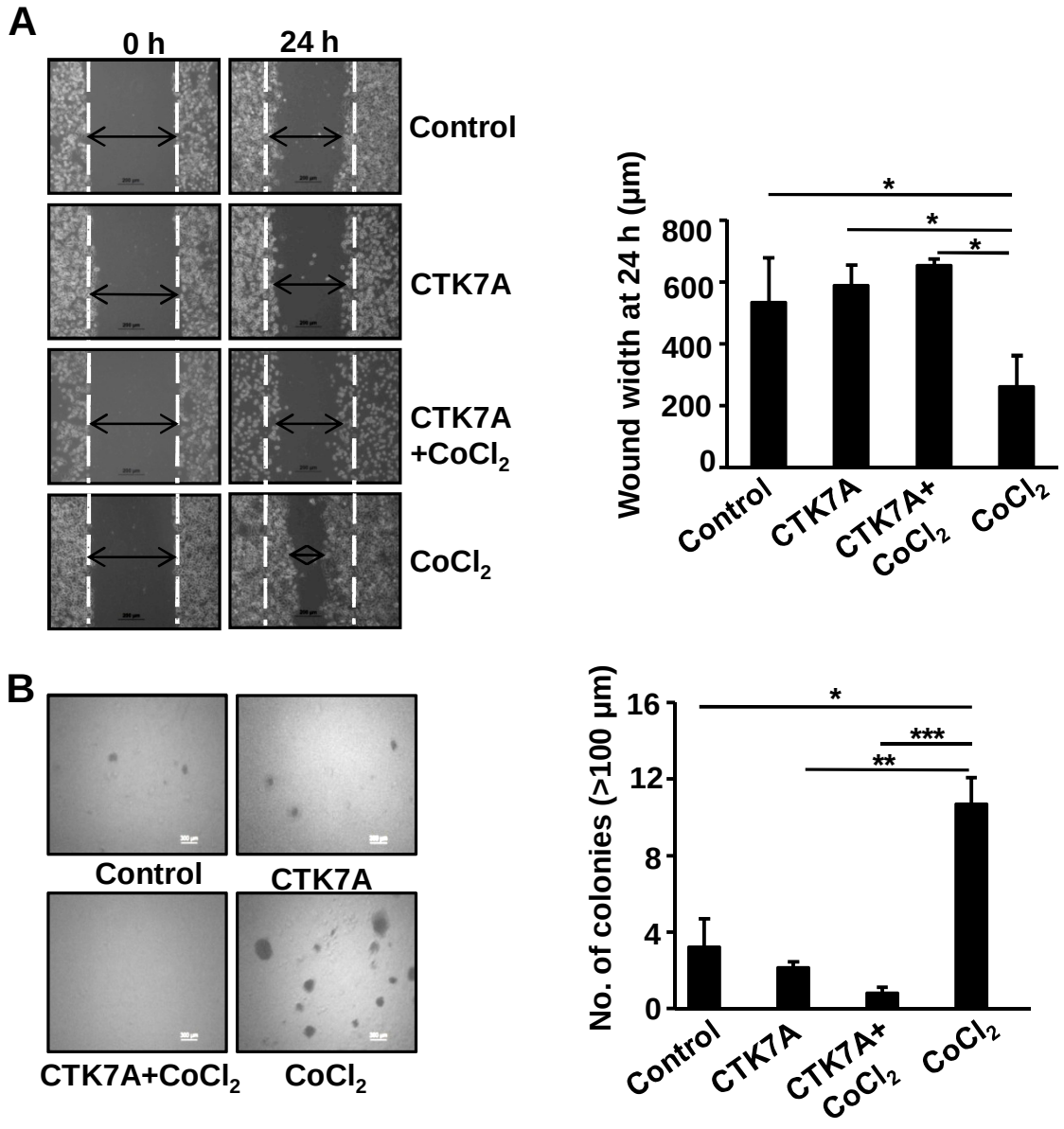


Fig. S3. *CTK7A downregulated metastatic properties in $CoCl_2$-treated GCCs.* (A) AGS cells were treated with $CoCl_2$ (200 µM) alone or in combination with CTK7A (100 µM) or left untreated for 24 h and wound healing property of cells was assessed by measuring the wound width (n=4 independent experiments, *P<0.05). Photographs were taken using an inverted microscope equipped with camera (Primo Vert Carl Zeiss, Germany). Scale bar 200 µm. (B) Anchorage-independent growth of CTK7A and $CoCl_2$-treated cells was evaluated by soft agar assay. AGS cells were treated with 200 µM $CoCl_2$ and/or CTK7A (100 µM) for 24 h or left untreated. Cells were incubated for 21 days. Colonies were counted by an inverted microscope. Scale bar 300 µm. Bar graphs depict quantification of anchorage-independent growth (*n*=3) by Student's *t*-test; *P<0.05, **P<0.01, ***P<0.001.

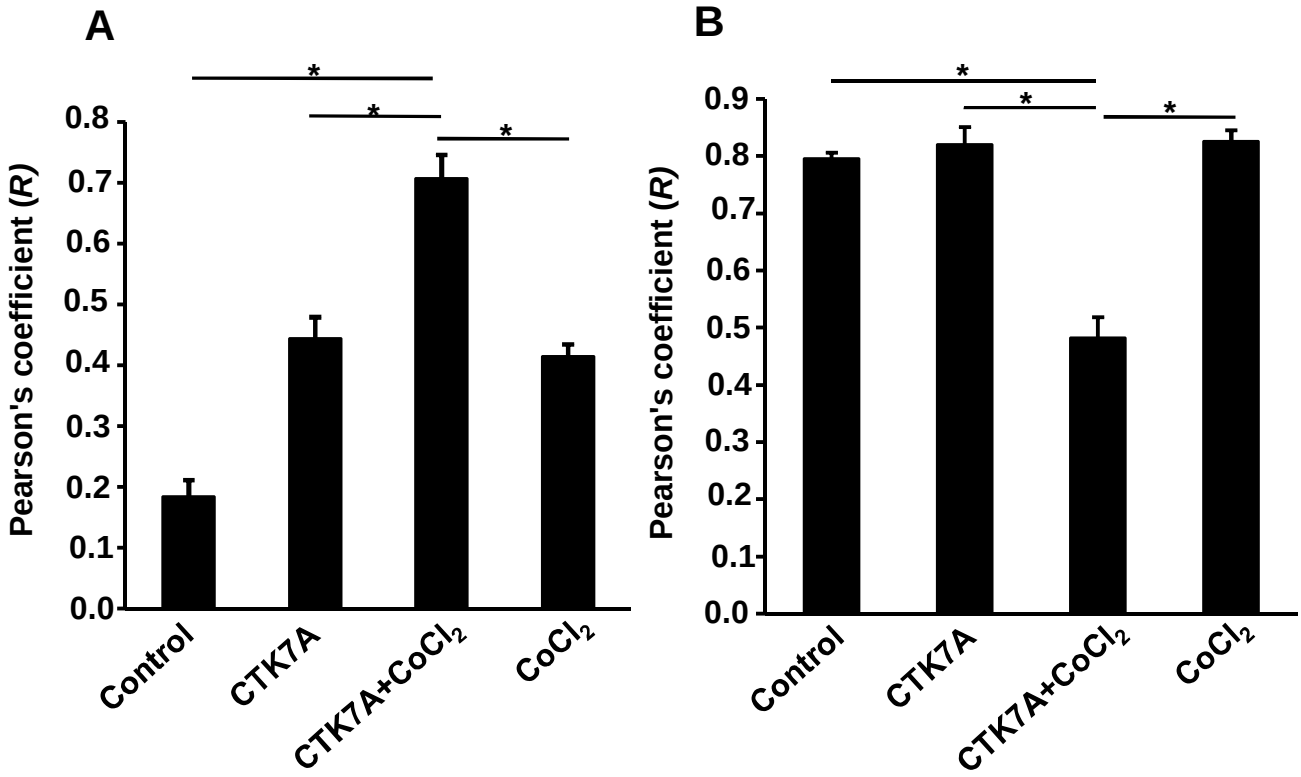


Fig. S4. *CTK7A increased Noxa translocation to mitochondria and cytochrome c release in $CoCl_2$-treated GCCs.* (A) Colocalization analysis of figure 4B was performed by ImageJ plugin Coloc 2 considering 4 separate mitochondrial regions in four different cells excluding the nucleus. A representative bar diagram (n=4) indicated significantly high Noxa in mitochondria in the CTK7A+$CoCl_2$-treated GCCs as compared to the other three treatment groups. (mean±SEM, n=4), **P*<0.05 (B) Graphical analysis of figure 4C by using Coloc 2 software demonstrated significantly less Cytochrome *c* in the CTK7A+$CoCl_2$-treated mitochondria as compared to the other three experimental groups (mean±SEM, n=4), **P*<0.05.

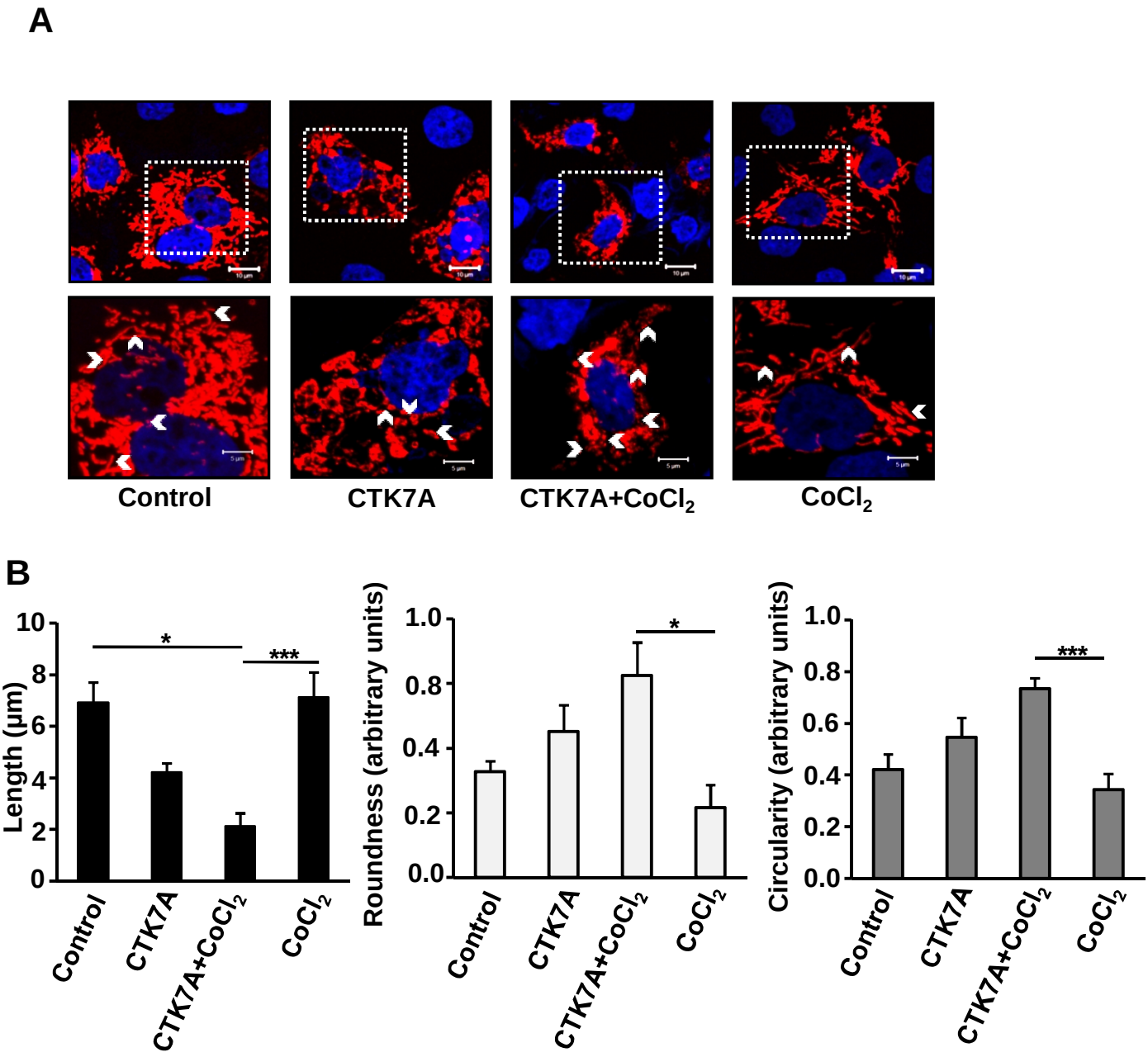


Fig. S5. *CTK7A induced mitochondrial fragmentation in $CoCl_2$-treated GCCs.* (A) Upper panel: mitochondrial morphology was examined by confocal microscopy after treatment with $CoCl_2$ (200 µM) alone or in combination with CTK7A (100 µM) for 24 h in p-DsRed2 stably-expressing AGS cells. Scale bar 10 µm. Lower panel: zoomed-in images of the selected areas shown in the upper panel. Scale bar 5 µm. (B) Mitochondrial morphology was analyzed in terms of length, roundness and circularity based on information collected from four cells (from four independent experiments). The mean length of five mitochondria was taken from each cell for statistical analysis. Circular mitochondrial appearance, indicative of mitochondrial stress, was most apparent in $CoCl_2$+CTK7A treated cells (mean±SEM, n=4), **P*<0.05, ***P*<0.001.