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Autophagy regulated by IncRNA HOTAIR contributes to the cisplatin-induced resistance in endometrial cancer cells

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Figure Legends

Fig. 1 Ishikawa CP presents stronger autophagy than normal Ishikawa. (a) *ptf*LC3 transfection data for examination of autophagy. (b) Immunofluorescence detection of autophagosomes between Ishikawa and its resistant cell line.

Fig. 2 LncRNA HOTAIR is responsible for effective proliferation of Ishikawa CP in CP treatment. (a) Effect of HOTAIR siRNA on HOTAIR expression in Ishikawa cells. (b) Representation of cell proliferation of normal Ishikawa and Ishikawa CP in absence or presence of scrambled siRNA or HOTAIR siRNA at indicated time by using CCK-8 assay. Error bars \pm SD *p < 0.05.

Fig. 3 LncRNA HOTAIR impacts the autophagy activity of Ishikawa and Ishikawa CP in absence or presence of CP treatment. Plasmid tfLC3 was transfected into cells through a typical procedure that specifically and simultaneously labeled autophagosome green.

Fig. 4 LncRNA HOTAIR mediates the expression of Beclin-1, MDR and P-gp in Ishikawa and Ishikawa CP cells upon CP treatment.

Figures





b



Fig. 1

lshikawa CP

а













Beclin-1

MDR

P-gp

GAPDH