

Correction

Correction: MicroRNA-25 regulates chemoresistance-associated autophagy in breast cancer cells, a process modulated by the natural autophagy inducer isoliquiritigenin

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This article has been corrected: In Figure 2C, the ‘Ctrl’ image is incorrect; it has been replaced. In Figure 3C, the 6th image in the 2nd row contains a partial overlap of the 3rd image in the 1st row of Figure 3A. Also in Figure 3C, the 1st image in the 2nd row is incorrect; it has been replaced. In Figure 4C, the 1st image contains a partial overlap of the 2nd row, 2nd panel image in Figure 5C. The 2nd image in Figure 4C contains a partial overlap of the 1st row, 2nd panel image in Figure 2E. The corrected Figures 2C, 3C, and 4C, produced using the original data, are shown below. The authors declare that these corrections do not change the results or conclusions of this paper.

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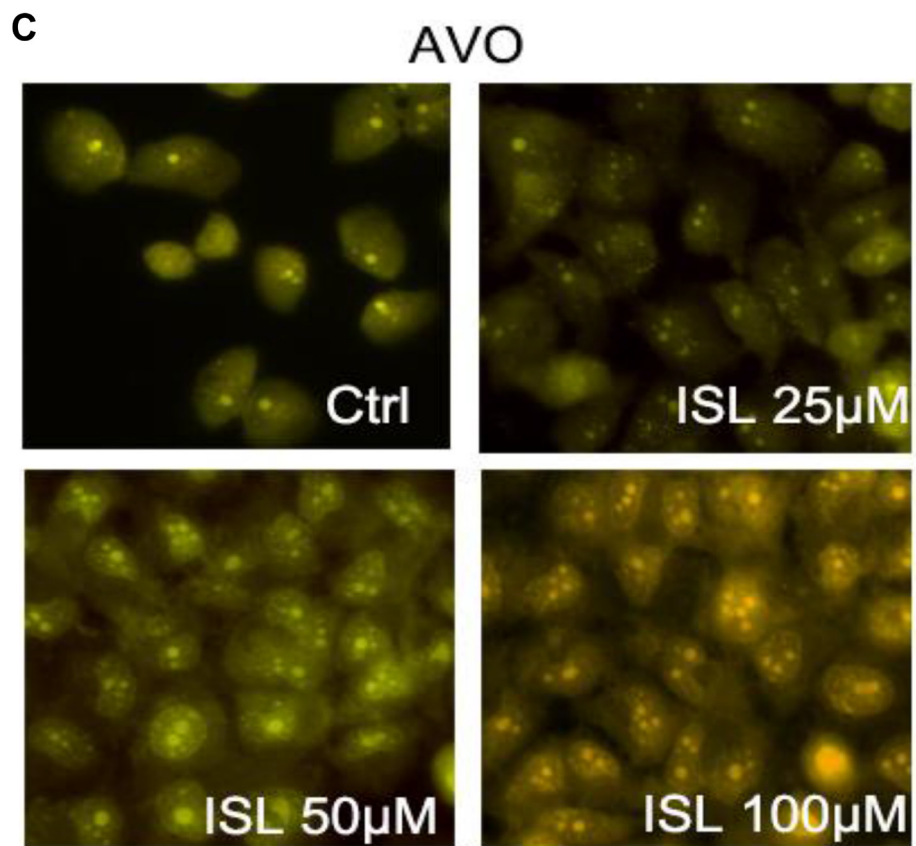


Figure 2: ISL induces autophagy in MCF-7/ADR cells. (C) ISL triggered autophagy in MCF-7/ADR cells after 24 h treatment, indicated by increased LC3-II puncta and AVO formation.

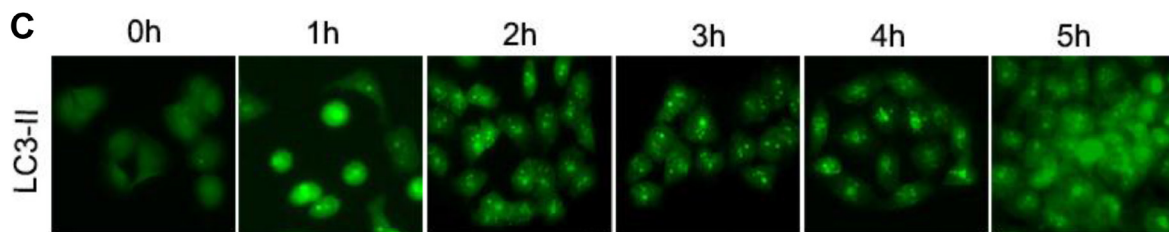


Figure 3: ISL inhibits ABCG2 expression via the autophagy-lysosome pathway. (C) time point-based immunofluorescence detection was performed to examine the correlation between autophagy induction and ABCG2 inhibition.

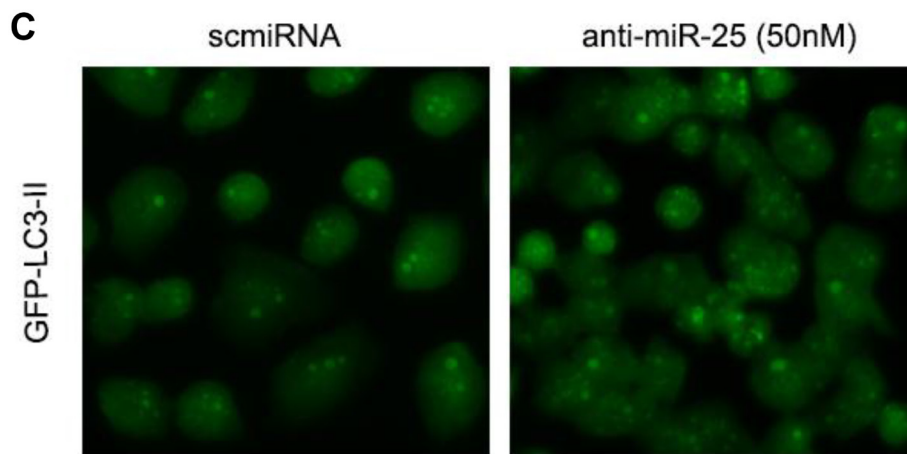


Figure 4: Identification of miR-25 as a novel regulator of ISL-induced autophagy, which functions by targeting ULK1. (C) miR-25 inhibitors were transfected into MCF-7/ADR cells. After 24 h, increases in GFP-LC3-II accumulation was observed, confirming that miR-25 independently induced autophagy flux.