Another instance that proves the sinister nature of Autophagy:

Lenvatinib is a drug used to treat advanced hepatocellular carcinoma (HCC) that targets multiple molecules, including vascular endothelial growth factor receptors 1–3 (VEGFR1–3), fibroblast growth factor receptors 1–4 (FGFR1–4), platelet-derived growth factor receptor α (PDGFRα), KIT, and RET.

It is an effective angiogenesis inhibitor, promotes apoptosis and inhibits cell proliferation to suppress tumour growth. However, in recent years, despite the substantial advantages of Lenvatinib in the context of advanced HCC, its efficacy remains poor with the overall median OS in patients with HCC is ~1 year, and the ORR is approximately 40%. These poor prognoses are mainly associated with the development of drug resistance. There is evidence that more than 60% of patients with HCC develop resistance to Lenvatinib within 1 year; only a few patients experience long-term benefits.

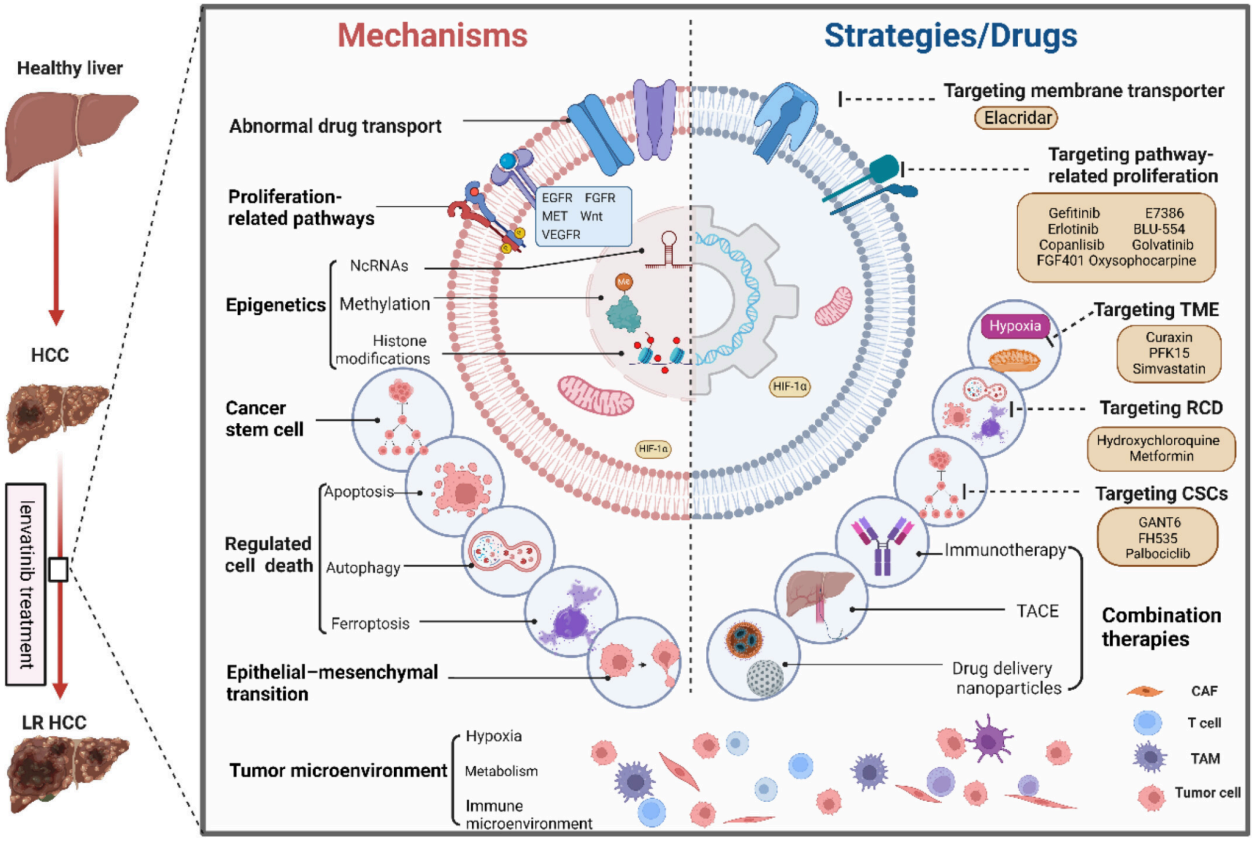

Figure is taken from: [Lenvatinib in hepatocellular carcinoma: Resistance mechanisms and strategies for improved efficacy]

A study by Shao-Qiang Li and his team (Published in Oncogene) now shows that this resistance to Lanvatinib is mediated by STX6-induced autophagy. STX6 interacts with three crucial autophagy modulators: Beclin1, VTI1A, and VAMP3.

This study highlights once again that, when properly modulated, harnessing autophagy can serve as a powerful therapeutic strategy.

Read the publication here:

Syntaxin-6 mediated autophagy confers lenvatinib resistance in hepatocellular carcinoma