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Summary

ACTIVATION OF NECROPTOSIS AS AN APPROACH TO OVERCOME DRUG RESISTANCE IN CANCER CELLS

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Newly synthesized ruthenium (II)-triazene hybrids induce both apoptotic and necroptotic cell death in cancer cells, offering a dual mechanism to target apoptosis-resistant tumors.

KEY POINTS

- Ru-TRZ naturally induce apoptosis but remain highly cytotoxic under apoptosis-blocking conditions, particularly in cancer cell lines HT29 and HepG2, through necroptosis as demonstrated by reversal with necrostatin-1 and Mlkl silencing
- Treatment with Ru-TRZ increases expression and activation of necroptosis mediators RIPK3 and MLKL, induces mitochondrial ROS production and membrane depolarization, while Ripk3 ablation protects hepatocytes from these effects
- Ru-TRZ synergistically potentiate the cytotoxic effects of commonly used chemotherapeutic drugs such as 5-fluorouracil and oxaliplatin
- The authors conclude this dual cell death mechanism is relevant for situations where apoptosis is compromised and may enhance cancer treatment effectiveness in combination therapy
- This material is relevant for researchers investigating novel metal-based anticancer agents and mechanisms to overcome drug resistance

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