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# Evaluation of Lobaplatin Chemosensitivity and Mechanistic Insights Using a 3D Bioprinted Primary Colorectal Cancer Model

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## INTRODUCTION & AIM

To evaluate the chemosensitivity of lobaplatin in primary colorectal cancer using a bioprinted tumor model and to explore its effects on the tumor.

## METHOD

Primary CRC cells derived from 20 patients were used to establish 3D bioprinted tumor models. After stabilization, constructs were treated with increasing concentrations of lobaplatin. Cell viability was assessed using the CellTiter-Glo 3D assay to determine $IC_{50}$ values and inter-patient variability. Chemosensitivity of lobaplatin was compared with 5-fluorouracil, oxaliplatin, raltitrexed, and TNF-α. In parallel, SW480 and SW620 cell lines were evaluated under both 2D and 3D culture conditions. Low-dose lobaplatin ($IC_{20}$ and $IC_{50}$) was applied to assess migration and invasion using wound-healing and 3D invasion assays. ELISA was used to detect invasion-related secreted factors, and RNA sequencing was performed to identify differentially expressed genes and enriched signaling pathways.

[Insert your conclusions here]

## RESULTS & DISCUSSION

[Insert your introduction & aim here]

Lobaplatin demonstrated a clear dose-dependent inhibitory effect in all 20 primary CRC models, with a mean $IC_{50}$ of 21.40 μM (range: 0.67–74 μM), indicating marked inter-patient heterogeneity. At clinically relevant concentrations, lobaplatin significantly reduced tumor cell viability and exhibited stronger cytotoxicity than comparator drugs at high doses. In cell line experiments, $IC_{50}$ values were consistently higher in 3D cultures than in 2D cultures. Functional assays revealed that low-dose lobaplatin significantly suppressed migration and invasion in metastatic SW620 cells but showed minimal effects in SW480 cells. Transcriptomic analysis showed upregulation of genes involved in drug metabolism, oxidative stress, and differentiation, and downregulation of pathways related to cell migration, inflammation, immune regulation, and extracellular matrix remodeling.

[Insert your results and discussions here]

[Insert your methodology here]

## CONCLUSIONS

Lobaplatin may exert antitumor activity by modulating stress-response pathways and suppressing pro-migratory and pro-inflammatory signaling. This model represents a promising platform for individualized chemotherapy evaluation and mechanistic studies in colorectal cancer.

[Insert your references/acknowledgment/future work ideas here]