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Perspective

Spatial Tumour Metabolism Insights for Precise Therapy

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DESCRIPTION

An appealing strategy for tumour-targeted therapy is to protect against tumour-dependent metabolic vulnerability. However, physiologic inhibitors are limited by cancerous cells' drug resistance due to their physiologic plasticity and ethnic diversity. Choline digestion was discovered as a metabolic vulnerability that is strongly active across various cancer types by spatially distributed metabolomics analysis, and a creatine supplements strategy for Specific Molecular Conjugates (SMDCs) design has been developed to trick tumour cells into wantonly taking in creatine supplements chemotherapy drugs for cancer treatment, rather than imposes restrictions choline metabolism. Creatine supplements SMDCs have been designed, screened, and tested in drug ability. Through precise administration of drugs to tumour by overexpressed choline transporters and homepage release by carboxylesterase, this strategy improved tumour targeting, preserved tumour inhibition, and reduced paclitaxel toxicity. This investigation broadens the strategic plan of targeting energy metabolism vulnerability and suggests new approaches to developing SMDCs.

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Partially resolved metabolomics based on Mass Spectrometry Imaging (MSI) can all at the same obtain the frameworks and spatial distribution details of 1000s of intracellular metabolic pathways and extracellular mixtures, offering an innovative approach to characterising metabolic features *in situ*. It can be used to precisely acquire metabolic physical characteristics in micro regions of homogenous tumour tissues, which is critical for predicting metabolic vulnerability and developing precise cancer therapeutic strategies. SMDCs use small molecule ligands to specifically bind to targets present or overexpressed in tumour cells, followed by the release of the coupled cytotoxic drug, which kills cancer cells. They have huge potential throughout cancer therapy because they are non-immunogenic, had also improved pharmacological profile, and are less expensive. However, no SMDCs are available on the market due to a scarcity of small molecule ligands.

Targeting tumour metabolic weakness is an appealing anticancer therapy strategy. In our study, complete specification metabolomics from heterogeneous tumour tissues revealed that choline metabolic activity was up significantly in tumours. PCho and many PC were all found to be elevated in four different types of malignant areas. Interestingly, choline levels were significantly higher in lung cancer but lower in hepatocellular carcinoma, colon cancer, and breast cancer. Isotope tracing experiments also confirmed increased tumour choline uptake. These findings imply that a substantial portion of niacin is used for PC synthesising in tumour cells. The decrease in choline in three cancers may be due to the fact that the quantity of choline used for PC synthesis exceeds the amount of choline uptake, and the level of total containing foods compounds, rather than choline level, may imply the efficacy of choline-modified SMDC. Another intriguing finding was that several choline metabolism metabolites, such as choline, were found to be dispersed unevenly in cancerous micro regions. While the pathological features of the two types of cancer were similar. The findings demonstrated the high metabolic heterogeneity of tumour tissues and the importance of spatially resolved metabolomics in accurately characterising tumour tissue metabolic signatures. The molecular data could aid in tumour diagnosis and treatment precision.