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Metabolic regulation of tumor microenvironment heterogeneity

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Background: Tumor microenvironment is the cellular environment in which the tumor exists, including surrounding blood vessels, immune cells, fibroblasts, bone marrow-derived inflammatory cells, lymphocytes, signaling molecules and the extracellular matrix. The tumor microenvironment contributes to the tumor heterogeneity. Exact mechanism of causing tumor heterogeneity is not clearly understood yet.

Purpose: The purpose is to study the metabolic regulation of tumor microenvironment heterogeneity.

Results & Discussion: Data from our previous research and others suggest that tumor heterogeneity may be resulted from unsynchronized differentiation of cancer progenitor cells. We then hypothesized that cell phenotype may be altered by proteome phenotype that is resulted from altered genomic phenotype that may be regulated by metabolic phenotype initiated by early signals. In another words, heterogeneity of tumor microenvironment may be resulted from dysregulated metabolic phenotypes. To test our hypothesis, we developed analytical methods to measure quantitatively the signals and its initiated metabolic phenotypes in a cell. The methods developed in our group at both UCLA and Creighton University includes stable isotopomer-based flux analysis and dynamic measurement of protein turnover in a cell, which extensively used in cancer research. In this oral communication, the details on how these methods can be used will be delivered and an example based on the methods will be also illustrated.

Conclusion: Understanding the mechanism underlying tumor microenvironment heterogeneity regulated metabolically is the key for development of anticancer drugs with minimum toxicity and maximal effectiveness.

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