

Regulation of Cellular Metabolism by SOD1 in Lung Cancer

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High levels of reactive oxidative species (ROS), a byproduct of aerobic metabolism, result in oxidative stress that leads to cellular damage. Cells can respond to oxidative stress by reprogramming metabolic pathways to produce more antioxidants. Abnormality in Superoxide Dismutase 1 (SOD1), an antioxidant enzyme that breaks down ROS, can lead to the development of diseases such as cancers and motor neuron diseases. SOD1 has been identified as a potential target for small molecule inhibitors of lung cancers. SOD1 also plays a role in regulating the cellular response to oxidative stress, but its specific mechanisms in regulating metabolic pathways are unclear. My study aims to identify novel metabolic pathways that are related to SOD1 in non-small cell lung cancer. Metabolomics data from the Cancer Cell Line Encyclopedia (CCLE) datasets was used to study alterations in metabolic pathways of 122 non-small cell lung cancer cell lines. R and MetaboAnalyst were used for statistical analysis and data visualization. Preliminary results show a correlation between SOD1 expression and alterations in specific metabolic pathways. Further studies should use metabolics and isotope tracing experiments to establish how SOD1 plays a role in regulating these metabolic pathways and shed new light on novel therapy targets for cancers with SOD1 abnormality. Supported by NIH R25ES020721 and ORED.

