Eun-Beom Lee et al., J Cancer Sci Ther 2017, 9:9(Suppl)
DOI: 10.4172/1948-5956-C1-112

Potential transcription factor involves NFE2L1 in hepatoma cell invasiveness through ROS induction

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Mitochondrial dysfunction is an important metabolic feature in human cancer. However, underlying mechanisms how mitochondrial dysfunction affects tumorigenesis remain unclear. To address the role of transcriptomic regulation by mitochondrial defects in liver cancer cells, we performed gene expression profiling for three different cell models of mitochondrial defects: cells with chemical respiratory inhibition, cells with mitochondrial DNA depletion, and liver cancer cells harboring mitochondrial defects. By comparing gene expression in the three models, we identified 10 common mitochondrial defect (CMD)–related genes that may be responsible for retrograde signaling from cancer cell mitochondria to the intracellular transcriptome. Among the CMD genes, we found that NFE2L1 is a key regulator to regulate hepatoma invasiveness. This study aims to elucidate how mitochondrial defect regulates NFE2L1 transcription. Interestingly, SNU354 and SNU423 cells showed high intracellular reactive oxygen species (ROS) levels. Exogenous treatment of H2O2 increased intracellular ROS and NFE2L1 expression in SNU387 cell harboring active mitochondria. We further selected 11 transcription factors (TFs) that could bind to promoter region of NFE2L1 by using TRANSFAC program. By monitoring NFE2L1 mRNA levels after knocking-down of the TFs, 4 TFs (USF2, STAT3, JUN and SREBP1) were identified to regulate NFE2L1 transcription. Further detailed molecular mechanisms of how mitochondrial ROS regulates NFE2L1 transcription are currently under investigation.

Biography

Eun-Beom Lee is pursuing Master's Degree from Ajou University School of Medicine. Her major Research Interest is in mitonuclear communication in tumorigenesis and retrograde signaling in hepatoma cells.

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