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A Cancer-Specific Study on the Differentially Expressed Protein-Protein Interactions of Fumarate Hydratase

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Fumarate hydratase (FH) is an enzyme used in the Krebs Cycle to convert fumarate to malate, and it is controlled by the FH gene. In this paper, we will investigate its role in Uterine Corpus Endometrial Carcinoma (UCEC) and how FH-deficient cells affect tumorigenesis. It is well-established that FH has been extensively studied in connection with renal cell carcinoma, skin and uterine leiomyomas, pheochromocytoma, and paraganglioma. However, we aim to construct an interaction network of significant genes related to the FH gene under conditions of FH deficiency in the Kreb Cycle. Creating an interactive network that illustrates the interconnectedness of FH's role is crucial for comprehending cellular adaptations when FH is deficient. Unfortunately, we have not yet found a reliable and accurate representation of this complex network, which has prompted us to create our own. For our dataset, we utilized RNAseq count data from the UCSC Xena database. We followed this with a differential expression gene (DEG) analysis workflow involving Limma and EdgeR. The significantly expressed genes were contextualized through an enrichment analysis called EnrichGO. Finally, we associated the significantly expressed genes with a transcription factor (TF). Our results have allowed us to construct a network that presents our findings. Most importantly, it has revealed the significant role played by the HIF3a TF in FH-deficient cells. While HIF3a is less understood compared to its other isoforms (HIF1a and HIF2a), this research contributes to bridging that knowledge gap. Our findings suggest that the HIF3a gene is a significant, differentially expressed gene in FH deficient patients.

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