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METABOLIC ALTERATIONS INDUCED BY EPIGALLOCATECHIN-3-GALLATE IN A MYELODYSPLASTIC SYNDROME MOUSE MODEL: A METABOLOMIC APPROACH

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Introduction: Myelodysplastic syndromes (MDS) are hematologic malignancies characterized by ineffective hematopoiesis and high risk of progression to acute myeloid leukemia (AML). Epigallocatechin-3-gallate (EGCG), a green tea polyphenol, exhibits anti-cancer effects through diverse mechanisms, including metabolic modulation. **Objectives:** This study assessed EGCG-induced metabolic alterations in a transgenic MDS mouse model using untargeted metabolomics. **Material and methods:** C57BL/6-Tg(Vav1-NUP98/HOXD13) mice were treated for 30 days with EGCG (50 mg/kg/day, intraperitoneally) or saline (control). Serum samples were analyzed by LC-ESI-QToF-MS in positive and negative ionization modes. Data processing (Progenesis^{TM} QI) and statistical analysis (MetaboAnalyst^{TM} 5.0) included PCA, VIP score ranking, and KEGG-based pathway enrichment. **Results:** From 3,910 detected features, 72 metabolites were identified—predominantly glycerophospholipids, fatty acids, and steroids. PCA revealed distinct metabolic profiles between groups. Seven significantly altered pathways were identified, including unsaturated fatty acid biosynthesis, amino acid metabolism (alanine, aspartate, glutamate, glycine, serine, and threonine), and ketone body synthesis/degradation. Elevated β-hydroxybutyrate suggested enhanced fatty acid β-oxidation and a metabolic shift from glycolysis toward lipid-based energy production. **Discussion and conclusion:** EGCG treatment in MDS mice induced broad metabolic reprogramming, reducing unsaturated fatty acid synthesis while enhancing β-oxidation and ketogenesis—conditions less favorable to glycolysis-dependent neoplastic cells (Warburg effect). These changes may lower membrane fluidity, disrupt signaling pathways, restrict nucleotide biosynthesis, and modulate epigenetic regulation via one-carbon metabolism. Such metabolic stress could contribute to EGCG's anti-cancer activity by impairing tumor cell proliferation and survival. Further preclinical and translational studies are warranted to confirm these mechanisms and explore EGCG's potential as a metabolic modulator and adjuvant therapy in MDS and related hematologic malignancies.

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MICALL2 IN ACUTE MYELOID LEUKEMIA: AN INTEGRATIVE APPROACH USING BIOINFORMATICS AND ARTIFICIAL INTELLIGENCE

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Introduction: Acute Myeloid Leukemia (AML) is a hematological malignancy of well-recognized clinical and epidemiological relevance, due to its high incidence and mortality rates worldwide. Its pathogenesis involves the uncontrolled proliferation of immature myeloid precursors in the bone marrow, impairing normal hematopoiesis. Recent bioinformatics studies supported by artificial intelligence tools have identified the MICALL2 protein as a promising prognostic marker, given its involvement in cytoskeletal regulation and in cellular processes such as adhesion, migration, metabolism, and intracellular signaling. Although MICALL2 has already been implicated in solid tumor development, its role remains unexplored in hematological malignancies. **Objectives:** In this context, the present study aimed to investigate the functional and prognostic role of the MICALL2 protein in Acute Myeloid Leukemia through bioinformatic analyses applied to transcriptomic data from public databases. **Material and methods:** Data from AML patients in the TCGA cohort were analyzed. Samples were dichotomized based on MICALL2 expression levels, with the cut-off defined by ROC curve analysis. Overall survival and disease-free survival were assessed using Kaplan–Meier analysis, and the same stratification was used for gene ontology and enrichment analyses. MICALL2 expression in normal hematopoiesis was investigated using NCBI data and visualized through hierarchical plots generated via