Genistein Reduces PFOA-Induced Lipid Accumulation and Cell Proliferation in Human Hepatocellular Carcinoma Cells

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Nonalcoholic fatty liver disease is characterized by excessive lipid accumulation in the liver and can progress to nonalcoholic steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma, affecting approximately 1 in 4 individuals worldwide. Environmental toxicants such as perfluorooctanoic acid (PFOA), to which most individuals have been exposed, are linked to disruptions in lipid metabolism and increased risk of liver disease and other health complications. This study evaluated whether genistein, a soy derived isoflavone with antioxidant and metabolic regulatory properties, can mitigate PFOA induced lipid accumulation and abnormal cell proliferation in human liver cells. HepG2 cells were treated with PFOA with or without genistein. Lipid accumulation was quantified using Oil Red O staining, and cell proliferation was assessed with MTT and growth assays. Quantitative PCR, Western blot, and luciferase reporter assays were used to examine changes in gene and protein expression and transcriptional activity in pathways regulating lipid metabolism and cell growth. PFOA exposure significantly increased intracellular lipid accumulation and cell proliferation compared to untreated controls. Genistein cotreatment reduced lipid deposition and suppressed cell growth. These effects were supported by consistent molecular changes, suggesting coordinated modulation of metabolic and proliferative pathways. These findings demonstrate that genistein partially counteracts toxicant induced metabolic and proliferative alterations, highlighting its potential as a dietary intervention to reduce environmentally associated liver disease risk and supporting further research on bioactive compounds in liver health.

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