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Details of the Collaborative Activity

2020-21

Name of the Collaborating Institute: PSG College of Pharmacy, Peelamedu, Coimbatore, Tamil Nadu, India.

Name of the Collaborating Department: Yenepoya Research Center

A Collaborative joint research project was undertaken between PSG College of Pharmacy, Peelamedu, Coimbatore, Tamil Nadu, India, along with Yenepoya Research Center, and as the outcome the following research article was published

1. Shaikh SB, Najar MA, Prabhu A, Rex DAB, Chanderasekaran J, Behera SK, Modi, PK, Prasad TSK, Bhandary YP. The unique molecular targets associated antioxidant and antifibrotic activity of curcumin in *in-vitro* model of acute lung injury: A proteomic approach. *BioFactors*. 2021.

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RESEARCH COMMUNICATION

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The unique molecular targets associated antioxidant and antifibrotic activity of curcumin in in vitro model of acute lung injury: A proteomic approach

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Abstract

Bleomycin (BLM) injury is associated with the severity of acute lung injury (ALI) leading to fibrosis, a high-morbidity, and high-mortality respiratory disease of unknown etiology. BLM-induced ALI is marked by the activation of a potent fibrogenic cytokine transcription growth factor beta-1 (TGFβ-1), which is considered a critical cytokine in the progression of alveolar injury. Previously, our work demonstrated that a diet-derived compound curcumin (diferuloylmethane), represents its antioxidative and antifibrotic application in TGF-β1-mediated BLM-induced alveolar basal epithelial cells. However, curcumin-specific protein targets, as well as its mechanism using mass spectrometry-based proteomic approach, remain elusive. To elucidate the underlying mechanism, a quantitative proteomics approach and bioinformatics analysis were employed to identify the protein targets of curcumin in BLM or TGF-β1-treated cells. With subsequent in vitro experiments, curcumin-related pathways and cellular processes were predicted and validated. The current study discusses two separate proteomics experiments using BLM and TGF-β1-treated cells with the proteomics approach, various unique target proteins were identified, and proteomic analysis revealed that curcumin reversed the expressions of unique proteins like DNA topoisomerase 2-alpha (TOP2A), kinesin-like protein (KIF11), centromere protein F (CENPF), and so on BLM or TGF-β1 injury. For the first time, the current study reveals that curcumin

Abbreviations: ACAT1, acetyl-CoA acetyltransferase 1; ACOX1, peroxisomal acyl-coenzyme A oxidase 1; AEC, alveolar epithelial cells; ALI, acute lung injury; BLM, bleomycin; BUB3, BUB3 mitotic checkpoint protein; CALR, calreticulin; CDK1, cyclin-dependent kinase 1; CENPF, centromere protein F; COL17A1, collagen type XVII alpha 1 chain; ER, endoplasmic reticulum; GO, Gene Ontology; IGF1R, insulin-like growth factor 2 receptor (IGF2R); IL, interleukin; KIF11, kinesin family member 11; KRT14, keratin14 (KRT14); MCM6, minichromosomes maintenance 6; MIP, 1-macrophage inflammatory protein-1; MS, mass spectrometry; NUSAP1, nucleolar and spindle associated protein 1; PDB, Protein Data Bank; PDGF, platelet-derived growth factor; PEX, peroxisomal biogenesis factor; PPI, protein–protein interaction; PTPN1, protein-tyrosine phosphatase nonreceptor type 1; ROS, reactive oxygen species; TOP2A, DNA topoisomerase II alpha.

