Abstract:

Pulmonary fibrosis is a medical condition in which lung tissue becomes irreversibly scarred. The disease causes permanent lung damage in the afflicted individual, for which there is no cure. Research in the Janssen-Heininger laboratory has shown that c-Jun-N-terminal Kinase (JNK) plays a role in the development of fibrosis by increasing the pro-fibrotic action of TGF-β1 (1) and activation of epithelial-mesenchymal transition (EMT) (2). TGF-β1 increases the oxidative stress of the cell, which is mediated by S-glutathionylation. This reversible oxidation reaction affects the activation of JNK1 and development of EMT. It is hypothesized that the TGF-β1/JNK1 axis enhances S-glutathionylation, which in turn induces activation of the EMT transcriptome.