

## **Liver Fibrosis and Cirrhosis: Role of The Fibroblastic Cell Subpopulations**

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Liver fibrosis is defined as the excessive deposition of extracellular matrix in an organ, is the main complication of chronic liver damage. Its endpoint is hepatic cirrhosis, which is responsible for significant morbidity and mortality. The accumulation of extracellular matrix observed in fibrosis and cirrhosis is due to the activation of fibroblasts, which acquire a myofibroblastic phenotype. Myofibroblasts are absent from normal liver. They are produced by the activation of precursor cells, such as hepatic stellate cells and portal fibroblasts. These fibrogenic cells are distributed differently in the hepatic lobule: the hepatic stellate cells resemble pericytes and are located along the sinusoids, in the Disse space between the endothelium and the hepatocytes, whereas the portal fibroblasts are embedded in the portal tract connective tissue around portal structures (vessels and biliary structures). Differences have been reported between these two fibrogenic cell populations, in the mechanisms leading to myofibroblastic differentiation, activation and "deactivation", but confirmation is required. Second-layer cells surrounding centrilobular veins, fibroblasts present in the Glisson capsule surrounding the liver, and vascular smooth muscle cells may also express a myofibroblastic phenotype and may be involved in fibrogenesis. It is now widely accepted that the various types of lesion (e.g., lesions caused by alcohol abuse and viral hepatitis) leading to liver fibrosis involve specific fibrogenic cell subpopulations. The biological and biochemical characterisation of these cells is thus essential if we are to understand the mechanisms underlying the progressive development of excessive scarring in the liver. These cells also differ in proliferative and apoptotic capacity, at least in vitro. All this information is required for the development of treatments specifically and efficiently targeting the cells responsible for the development of fibrosis/cirrhosis.

**Key words:** *Myofibroblast, Fibrogenesis, Liver Damage, Chirosis*