Liver disease is caused by several insults. Chronic injury results in cirrhosis and loss of organ function. The prevalence of liver disease in the E.U. is regulated and to investigate mechanisms involved in the inactivation of the cells.

Hepatic organoids of human cells and mouse freshly isolated cells, were developed and optimized in the lab as an attempt to predictability of potential side effects of drugs in development and reduces the number of animals needed for reliable drug testing. These inactivated HSCs (iaHSC) display an intermediate phenotype between quiescent and activated HSCs. Once reprogrammed to single and repeated exposure to reference compounds, they can developed fibrotic features that resemble the fibrosis in the liver. Ongoing work is using the same model to try to reproduce features of other drug-induced liver diseases (DILI) such as cholestasis, phospholipidosis and steatosis.

Liver disease models

Isolation of liver cells

- FACS
- 2D culture
- 3D culture

RNA analysis: QPCR

Protein analysis:

- WB
- IF
- IHC

Functional assays:

- Cell viability
- Cell proliferation
- Collagen cross-linking

Frequently used Techniques @ LIVR

Frequently used Techniques @ LIVR

CURRENT PROJECTS @ LIVR

Mechanisms of fibrosis development and resolution

Healthy Liver

Early HSC activation

Late HSC activation

Fibrotic Liver

Recovering Liver

HSCs, IHC, and IHC

Optimization of liver cell type isolations

Initial steps in HAC isolation

Mechanisms of fibrosis development and resolution

Healthy Liver

Early HSC activation

Late HSC activation

Fibrotic Liver

Recovering Liver

HSCs, IHC, and IHC

Identification of HSC-specific biomarkers for fibrosis staging

Distinction between KC, LSEC and HSC-populations derived from the UPACS3 procedure. A schematic representation of the cell type-specific markers identifies these compartments as HSCs, LSECs and KCs.

Liver death causes in the E.U.

Healthy liver

Cirrhotic liver

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