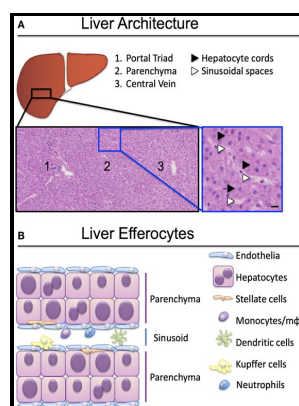


# Hepatocyte and Kupffer Cell interactions

CRC Press - Kupffer cell



Description: -

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Kupffer cells.

Liver cells.

Liver -- Pathophysiology.Hepatocyte and Kupffer Cell interactions

-Hepatocyte and Kupffer Cell interactions

Notes: Includes bibliographical references and index.

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Tags: #Interactions #between #macrophage/Kupffer #cells #and #hepatocytes #in #surgical #sepsis #(Thesis/Dissertation)

## Isolation of Kupffer Cells and Hepatocytes from a Single Mouse Liver

Corrao and colleagues 1998 found that 98 percent of cirrhosis cases in men but only 67 percent of cases in women could be attributed to alcohol metabolism and alcohol consumption, HCV, and hepatitis B.

## Endotoxin and Kupffer Cell Activation in Alcoholic Liver Disease

Both agents are known to cause robust increase in hepatocellular proliferation in rodent liver during the first few days of treatment; however, only WY-14,643 sustains rates of proliferation with long-term treatment as reported in. The role of Kupffer cells in hepatitis B and hepatitis C virus infections. These results illustrate that tumor promoter exposure results in a sustained increase in growth but just in preneoplastic cells.

## Interactions between hepatocytes and liver sinusoidal endothelial cells — Mayo Clinic

Journal of Immunology 166:4737—4742, 2001 a.

## Hepatocyte and Kupffer Cell Interactions (eBook, 2017) [ne-x.uni.rf.gd]

Roles of CD14 in LPS signaling and scavenging: Analysis of CD14 transgenic and non—transgenic mice and rats in response to LPS. Recognizing the potential limitations of these approaches, we have adopted a protocol that utilizes clodronate-encapsulated liposomes to deplete Kupffer cells in vivo vanRoojen et al. Similarly, liver damage seen in chronic ethanol consumption appears to be modulated by Kupffer cell activation.

## Kupffer cell

Molecular Mechanisms of Steatosis The hallmark of early alcohol-induced liver injury is steatosis or fat deposition as a result of increased intracytoplasmic triglyceride formation in hepatocytes. This hypothesis fits with the suggestion of a two-part response of the liver to peroxisome proliferators: one mediated by PPAR $\alpha$  and a second mediated by Kupffer cells. I kappa B interacts with the nuclear localization sequences of the subunits of NF-kappa B: a mechanism for cytoplasmic retention.

## Role of the Kupffer Cell in Mediating Hepatic Toxicity and Carcinogenesis

In nonfocal liver, LPS produced an early increase in DNA synthesis after 7 days which was also prevented by Kupffer cell depletion. The data on mechanisms of the Kupffer cell-hepatocyte interplay described in this review fall into two broad categories; the Kupffer cell as the primary target of toxic signals and the Kupffer cell as an accessory in the overall response of the liver to a toxic signal received by the hepatocyte. Thirty-five years ago Charles Lieber and colleagues 1975 published a seminal article in liver research, showing that alcohol itself is the primary cause for the higher prevalence of liver disease seen in alcoholic patients and not dietary deficiencies and malnutrition that often accompany alcoholism.

### **Difference Between Kupffer Cells and Hepatocytes**

Experiments on isolated cells grown in culture i.

### **NIAAA Publications**

Iron release is temperature-dependent, the rate at 37 degrees C being nearly 5-fold greater than at 4 degrees C. Thus, this chapter focuses primarily on the latter model as a means to explore the biology of hepatocyte—endothelial cell EC interactions. The concept of the gut—liver axis, which emerged from ALD, revolutionized our thinking about inflammation in other liver diseases, including nonalcoholic steatohepatitis and HCV infection.

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