Liver regeneration

The liver has the ability to regenerate after acute damage (e.g. toxins or drugs) or after resection of part of the liver (liver donation or hepatocellular carcinoma). In addition, regeneration is advantageous in liver part transplantation, because the recipient of the transplanted part of the liver grows back to its original size. All the cells present in the liver play a role in this process, with Ito cells being of great importance, both initiating and completing the process.

Regeneration

This is the process by which missing or damaged tissue parts are replenished and function is restored. The ability of cells to **proliferate and grow** is essential for its progress. Liver tissue regenerates only if the connective skeleton, vascular supply and bile capillaries of the lobe are preserved. Removal of part of the liver induces proliferation of the remaining hepatocytes, bile duct epithelial cells, sinusoidal endothelial cells, Kupffer cells and It cells within a few hours.

Control

This whole complex process must be properly controlled. The control involves growth factors and cytokines.

- Growth factors: HGF (Hepatocyte Growth Factor), EGF, TGF-α, FGF, VEGF...
- Cytokines: TNF-α, IL-6...

Regeneration phase

Liver regeneration can be divided into **3 phases'**: initiation, proliferation and termination[1]. The phases differ at the molecular and cellular level, i.e., in each phase a different signaling molecule leads to a different cellular response. === Initiation phase===. Activation of Kupffer cells, which secrete **TNF-α**. TNF-α autocrine activation of Kupffer cells containing the TNFR1 receptor is followed by a signaling pathway that leads to stimulation of the expression of the gene for **IL-6** and secretion of this interleukin via NF-kB. IL-6 binds to hepatocyte receptors and signals for proliferation (via the Jak/STAT3 signalling pathway).

Proliferative phase

Depends mainly on growth factor signaling. **HGF** is secreted mainly by It bb. It binds to the hepatocyte tyrosine kinase receptor c-met and triggers cell proliferation and growth.

Termination phase

Not much is known about it. However, it is known that regeneration is arrested by the cytokine **TGF-β** (produced by It cells).

References

Related articles

- Liver
- Ito cells
- Liver fibrosis
- Cell Signaling

Source

- Fujiyoshi M, Ozaki M. Molecular mechanisms of liver regeneration and protection for treatment of liver dysfunction and diseases. J Hepatobiliary Pancreat Sci (2011) 18(1):13–22.

Link

1. Fujiyoshi M, Ozaki M. *Molecular mechanisms of liver regeneration and protection for treatment of liver dysfunction and diseases*. J Hepatobiliary Pancreat Sci (2011) 18(1):13-22.

Použitá literatura

- NEČAS, Emanuel. *Obecná patologická fyziologie.* 3. edition. Praha : Karolinum, 2009. 0 pp. ISBN 978-80-246-1688-9.