LPS aggravates fibrosis only in the early onset but not in the end stage of liver fibrosis

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**Background**
- Pathogen associated molecular patterns (PAMPs), e.g. lipopolysaccharide (LPS), are possible drivers of liver fibrosis via the gut-liver axis.
- Serum LPS concentrations are elevated during cirrhosis.
- However, human studies on how PAMPs promote liver fibrosis are scant.
- Precision-cut liver slices (PCLS) are special ex vivo models to test the fibrogenetic effect of PAMPs.

**Methods**
- Human healthy liver
- Coring
- Slicing
- Human cirrhotic liver
- Incubated in William’s E Medium (\textsuperscript{+}GlutaMAX\textsuperscript{TM}, glucose, and gentamycin) for 48h
- Shaking 90 rpm at 37 °C in an 80% O\textsubscript{2}/5% CO\textsubscript{2} atmosphere
- ±1, 5, 10 µg/ml LPS
- Expression of genes – qPCR

**Results**
- Data are expressed as mean ± SEM, * p < 0.05, ** p< 0.01, *** p< 0.001, **** p< 0.0001

**Aim**
- Healthy and cirrhotic human liver precision-cut liver slices (PCLS) were exposed to LPS to elucidate the role of this PAMP in the progression of liver fibrosis.

**Summary**
- In healthy human PCLS, LPS aggravated both an inflammatory and fibrotic response.
- In cirrhotic PCLS, LPS only elicited a limited inflammatory response.

**Conclusion**
- LPS aggravates the early onset of liver fibrosis, but has no additional effect during the end stage of the disease.

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