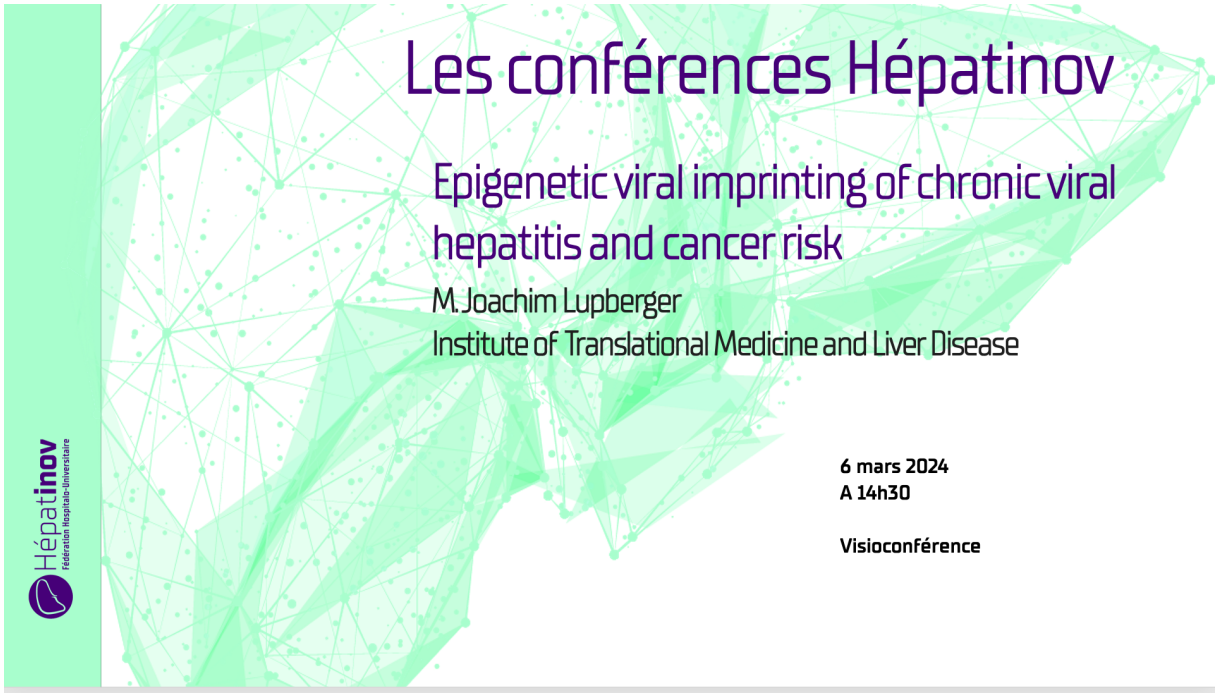
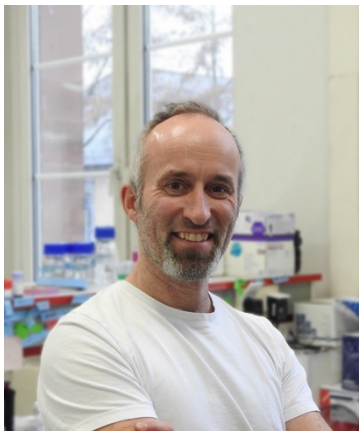
Parcours professionnel de M. Joachim Lupberger





Dr. Joachim Lupberger is research director (DR2, Inserm) and group leader at Institute of Translational Medicine and Liver Disease, Inserm U1110 in Strasbourg. He trained in Biotechnology and Molecular Virology at the Beuth University of Applied Sciences in Berlin, the MIT in Cambridge, Massachusetts, the Humboldt University of Berlin, and the University of Freiburg in Germany. In 2007, he joined the team of Prof. Thomas Baumert at Inserm U1110 in Strasbourg studying the relevance of signaling molecules for the life cycle of hepatitis C virus (HCV). Among others, he identified receptor tyrosine kinases as novel entry factors for HCV and regulators of the co-receptor complex (Lupberger and Zeisel et al., Nature Medicine 2011; Zona and Lupberger et al., Cell Host and Microbe 2013) and highlighted the importance of virus-induced signal transduction for viral pathogenesis and cancer (Mailly et al., Nature Biotechnology 2015; Van Renne et al., Gut 2018; Lupberger et al. Gastroenterology 2019; Butterworth et al. Pathogens 2021, Mukherji et al. Nature Communications, in revision RII).

His current research is focuses on the characterization of signal transduction induced by chronic viral hepatitis and its consequences for viral life cycle and the development of advanced liver disease, including fibrosis and hepatocellular carcinoma. Liver disease progresses from liver steatosis, inflammation, fibrosis, cirrhosis with an ultimate high risk to develop hepatocellular carcinoma (HCC). This disease course is highly similarly induced by viral hepatitis, obesity, and alcohol, which suggests common molecular drivers. Combining state-of-the art infection models, high-throughput screening technologies and genome-wide -omics studies. He aims to identify specific and common signaling pathways as drivers for the viral life cycle and liver disease progression as well as HCC risk biomarkers. Targeting disease-relevant signaling pathways by small molecules will be part of future chemo-preventive strategies to reduce cancer-risk in risk patients independent of the underlying liver disease etiology.