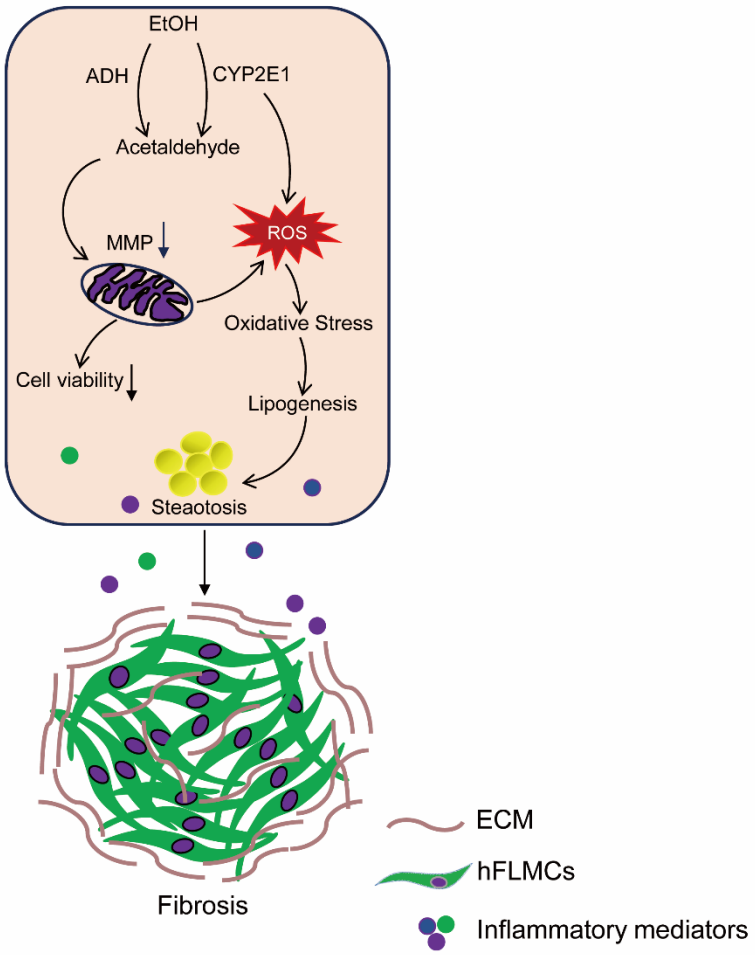
Fig. S10


EtOH
ADH
CYP2E1
Acetaldehyde
MMP
ROS
Oxidative Stress
Cell viability
Lipogenesis
Steaotosis
Fibrosis
ECM
hFLMCs
Inflammatory mediators

Supplementary information, Fig. S10 Summary of ALD-associated liver injury and pathogenesis recapitulated by hFLMC/hEHO model. In hFLMC/hEHO, EtOH is metabolized to acetaldehyde by alcohol dehydrogenase (ADH), and cytochrome P450 2E1 (CYP2E1). Acetaldehyde is both toxic and carcinogenic. In addition, reactive oxygen species (ROS) are generated as a by-product of CYP2E1 activity. ROS and acetaldehyde can lead to mitochondrial dysfunction and oxidative stress, which are presumed to induce hepatic cell death. Moreover, oxidative stress may initiate EtOH-induced lipogenesis and fatty liver. All these pathways may further contribute to liver injury. In addition, damaged hepatocytes release some inflammatory factors. Inflammatory factors and ROS both presumably stimulate hepatic stellate cells, which results in fibrogenesis.