

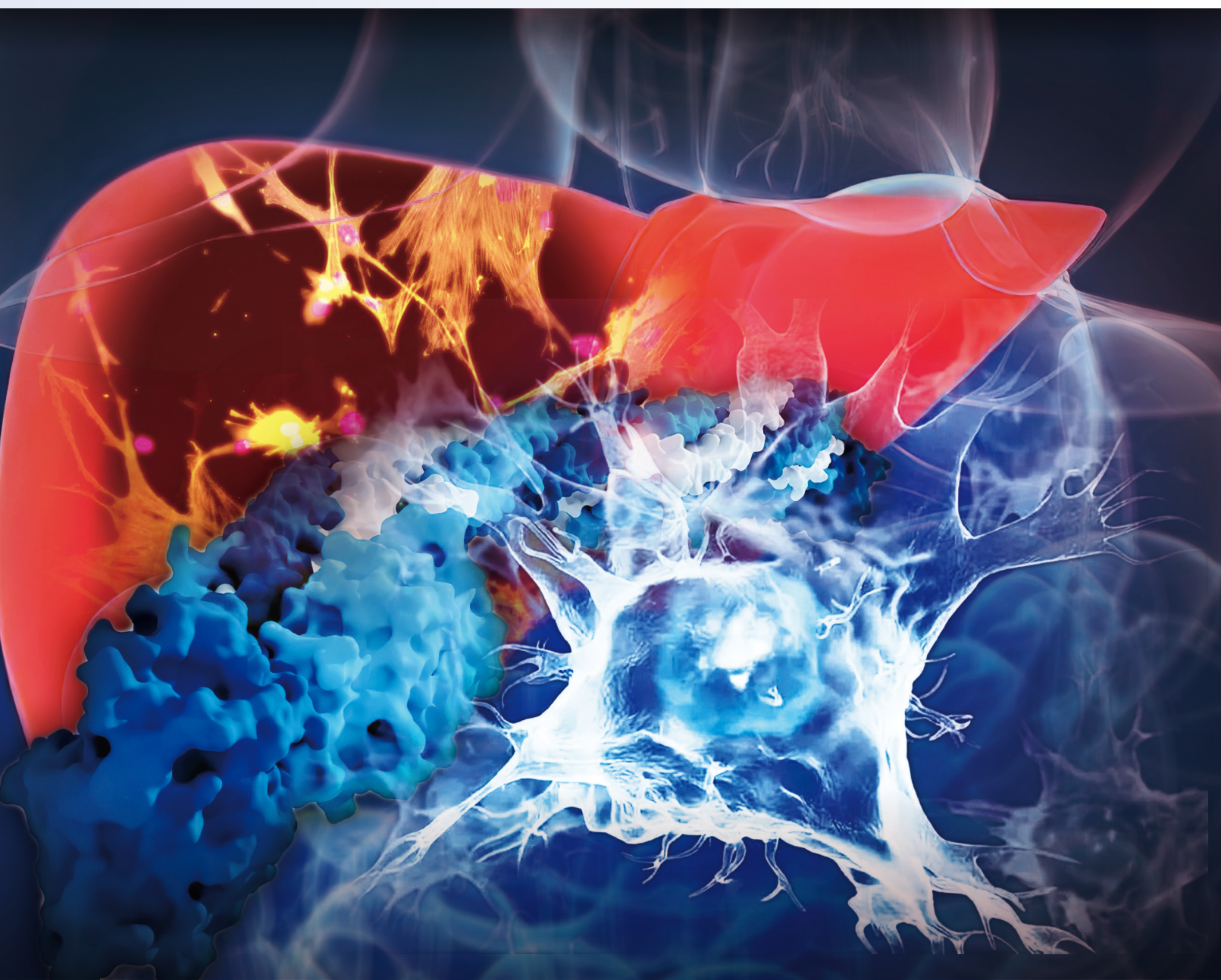
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封面图片:采用Cre/loxP转基因技术构建NDUFA13基因肝特异性杂合敲除小鼠,探究肝细胞线粒体NDUFA13蛋白表达缺失在诱导小鼠肝纤维化形成中的作用及可能机制,为肝纤维化治疗提供新思路,也为相关药物或机制研究提供一种有利的动物模型(徐小惠,重庆医科大学附属儿童医院,正文见本期529~535页)。

主 编:陈敏生

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Cover image: Hepatocyte-specific NDUFA13 knockout mice were generated based on the Cre/loxP transgenic technology to investigate the effect of NDUFA13 deficiency on liver fibrogenesis in mice and explore the possible mechanisms, which may provide a new strategy for treatment of liver fibrosis and a useful animal model for studying related drugs or mechanisms. Provided by XU Xiaohui, Children's Hospital of Chongqing Medical University. See full-text article on page 529-535 in this issue

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