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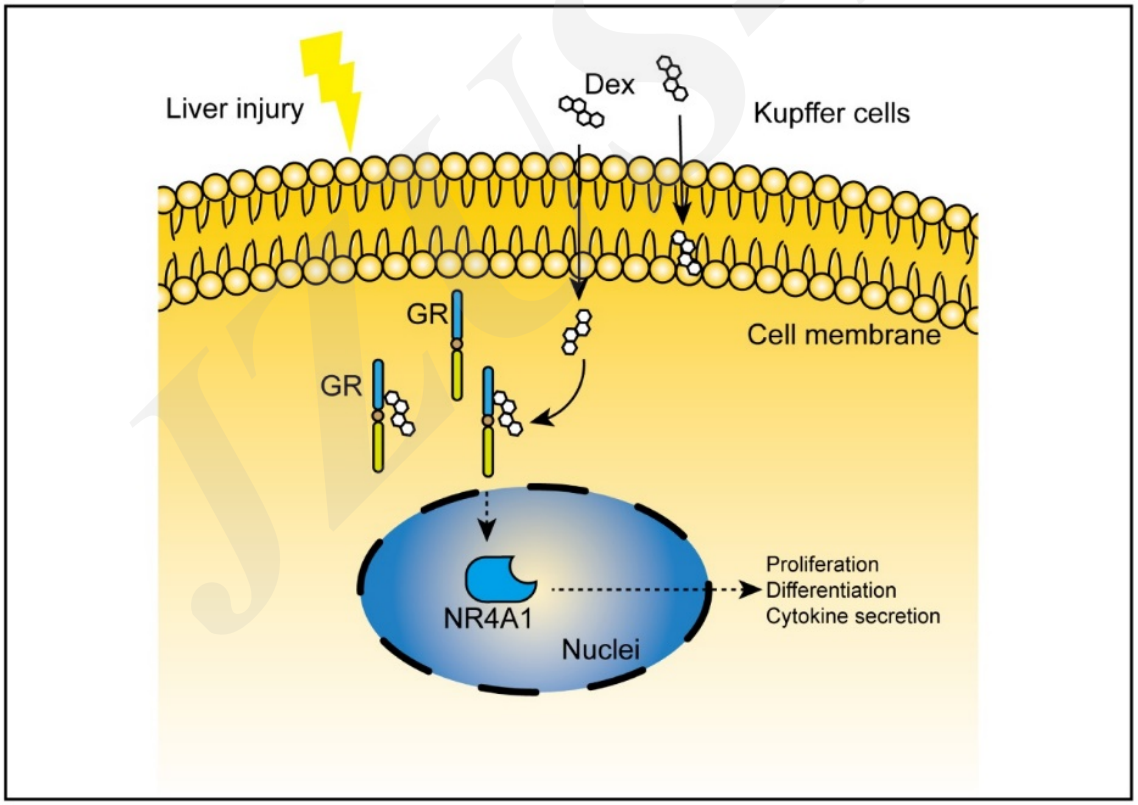
Early use of dexamethasone increases NR4A1 in Kupffer cells ameliorating acute liver failure in mice in a glucocorticoid receptor dependent manner

Key words: Glucocorticoid, Dexamethasone, Kupffer cells, Acute liver failure, Nuclear receptor subfamily 4 group A member 1

Research Summary

The purpose of this study was to investigate the specific immunological mechanism of dexamethasone (Dex) on treatment of acute liver failure (ALF) induced by lipopolysaccharide (LPS)/D-galactosamine (D-GalN) in mice.



Innovation points

- **In LPS/D-GalN-induced ALF mice, early administration of Dex improved ALF by increasing the numbers of innate immune cells, especially Kupffer cells and neutrophils.**

- **GR-dependent NA4R1 upregulation in Kupffer cells maybe an important ALF effect regulated by Dex in this process.**

Figure 4