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Role of NKT cells in liver repair after hepatic ischemia-reperfusion injury in mice

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Invariant natural killer T (iNKT) cells produce large amounts of cytokines to induce an inflammatory or reparative function. In addition, NKT cells are highly enriched in mice liver. The objective of the present study was to examine whether iNKT cells contribute to liver repair following hepatic ischemia and reperfusion (I/R) in mice. Wild-type (WT) mice and CD1d-deficient (CD1d^{-/-}) mice were subjected to hepatic I/R. Compared with WT mice, CD1d^{-/-} mice showed delayed liver repair as indicated by increases in ALT levels and hepatic necrotic area and by decreases in PCNA expression. These were associated with decreases in mRNA levels of IL-4 and IL-13 and in accumulation of Ly6C^{low} reparative macrophages in the liver. By contrast, treatment of WT mice with α -galactosylceramide (α -GalCer) at 0 h post-reperfusion stimulated liver repair after hepatic I/R. At 72 h post-reperfusion, iNKT cells and Ly6C^{low} macrophages were accumulated in the liver treated with α -GalCer, which were associated with enhanced expression of IL-4 and IL-13. These results suggest that iNKT cells and reparative macrophages are involved in promotion of liver repair after hepatic I/R injury.