

Alex B. Lentsch, PhD

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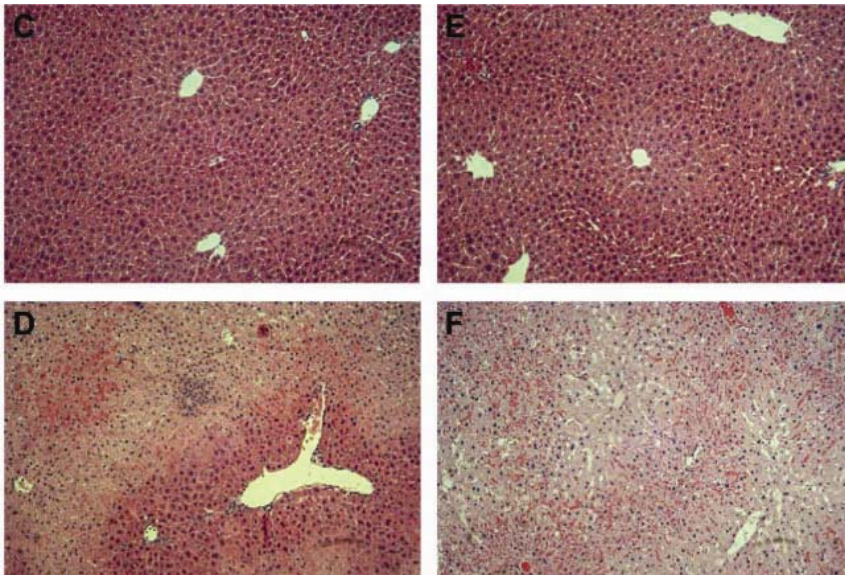
Description of Research:

Dr. Lentsch's work investigates the intricate roles of various soluble mediators in the initiation and regulation of the acute inflammatory response to hepatic ischemia/reperfusion. His work also examines age-related mechanisms of liver injury in this setting. He has recently shown that there are significant alterations in the inflammatory response and the injury response in young versus adult mice. The different responses among age groups appear to relate to altered regulation of a number of cellular and molecular aspects of this injury. For example, his laboratory has observed that adult mice have a much higher resident population of CD4+ lymphocytes in the liver compared to young mice. Parallel studies have shown that these cells play an intricate function in the dynamics of both the inflammatory response and direct hepatocellular injury. Furthermore, Dr. Lentsch's work has shown that some key cytoprotective proteins, such as the heat shock protein-70 (HSP70) is induced to a much lesser extent after ischemia/reperfusion in the livers of adult mice compared to young mice. Such divergence in the injury response due to age may relate to selective, age-dependent activation of specific genes which may lead to therapeutic targets for a number of liver diseases.

Collaborations:

Dr. Lentsch has collaborated with Dr. Matlin on the ultrastructural function of human neutrophils following trauma injury. He is collaborating with Dr. Waltz on the role of the Ron tyrosine kinase receptor in prostate cancer.

Representative Figure:



Liver injury and neutrophil accumulation in wild-type and CD4^{-/-} mice after ischemia-reperfusion injury. C-F: liver histopathology was examined by hematoxylin and eosin staining of liver sections. Sham-operated wild-type mice (C) and CD4^{-/-} (E) had normal histology. After ischemia and 8 h of reperfusion, wild-type mice had a typical pattern of focal necrosis with areas of intense neutrophil recruitment (D), whereas CD4^{-/-} mice had much more extensive hepatocellular necrosis but limited neutrophil accumulation (F). Fig. 3 from *Am J Physiol Gastrointest Liver Physiol*, 2005; 289:G969-976.