A novel method of isolating high quality RNA from Kupffer cells

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Kupffer cells, resident tissue macrophages that line the liver sinusoids, play a key role in modulating inflammation in a number of experimental models of liver injury. Since Kupffer cells represent only a small portion of the entire liver cell population, greatly outnumbered by the parenchymal cells, Kupffer cell isolation faces major technical obstacles. Laser capture microdissection (LCM) offers a method of isolating a single cell type from specific regions of tissue sections.

A research team led by Dr Stephen H Gregory from United States outlines isolation of Kupffer using LCM. This will be published on April 14, 2009 in the World Journal of Gastroenterology.

LCM is an essential approach used in conjunction with molecular analysis to study the functional interaction of cells in their native tissue environment. The process of labeling and acquiring cells by LCM prior to mRNA isolation can be elaborate, thereby subjecting the RNA to considerable degradation. Kupffer cell labeling was achieved by injecting India ink intravenously, thus circumventing the need for in vitro staining. The significance of this novel approach was validated using a cholestatic liver injury model.

They found that mRNA extracted from the microdissected cell population displayed marked increases in colony stimulating factor-1 receptor and Kupffer cell receptor message expression, which demonstrated Kupffer cell enrichment. Gene expression by Kupffer cells derived from bile-duct-ligated, versus sham-operated, mice was

compared. Microarray analysis revealed a significant (2.5-fold, q value liver injury

The methodology outlined herein provides an approach to isolating high quality RNA from Kupffer cells, without altering the tissue integrity.

More information: Gehring S, Sabo E, San Martin ME, Dickson EM, Cheng CW, Gregory SH. Laser capture microdissection and genetic analysis of carbon-labeled Kupffer cells. *World J Gastroenterol* 2009; 15(14): 1708-1718 www.wjgnet.com/1007-9327/15/1708.asp

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