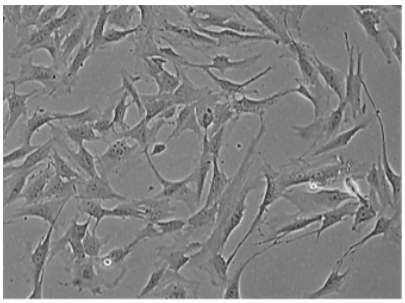
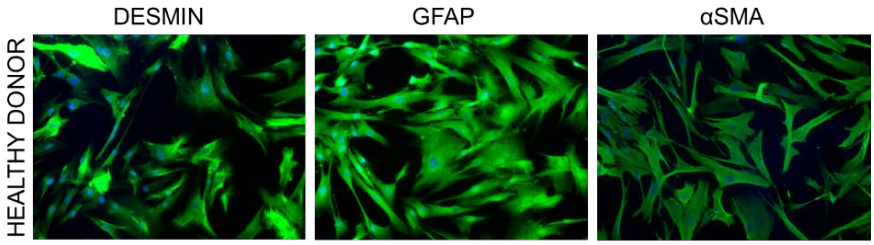
HUMAN HEPATIC STELLATE CELLS (HSC)

Samsara Sciences offers primary cryopreserved adult hHSC from both healthy and diseased liver tissues for use in research applications. hHSC can be propagated using industry-standard protocols for stellate cells, and detailed culture protocols and media recipes are provided with each lot. Hepatic stellate cells have long been recognized as key players in liver homeostasis and pathogenesis, and activation of stellate cells secondary to fatty liver disease and drug-induced liver injury is an accepted component of the progression of liver injury with fibrosis. Human stellate cells are identified by expression of glial fibrillary acidic protein (GFAP) and, to a lesser extent, desmin. Activated stellate cells gain expression of smooth muscle actin (SMA) and may also begin producing collagen and secreting TGFβ. Standard monolayer culture of HSC leads to culture activation of the cells, with a reduction in GFAP expression and increase in SMA expression over time. Samsara's human HSC are available standardly as cryopreserved stock at passage 2, and earlier passage cells may be available upon request.

What's in the box?

Customers receive cryopreserved hHSC along with detailed protocols for thawing, plating, serial passage, and cryopreservation, including recommended media and cryopreservation formulations. Protocols include exemplary images at each stage of culture, and our standard product characterization includes lot-specific data on post-thaw viability, cell size, population doubling rate, and expression of standard markers, such as GFAP and SMA. Furthermore, each lot is tested for mycoplasma using a sensitive molecular test (Roche MycoTool) prior to commercial release. Typical stellate cell morphology in culture is shown at passage 2.



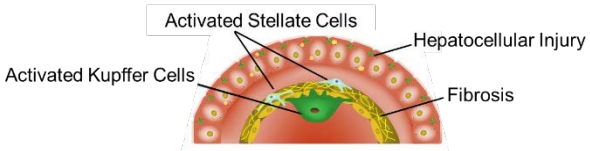


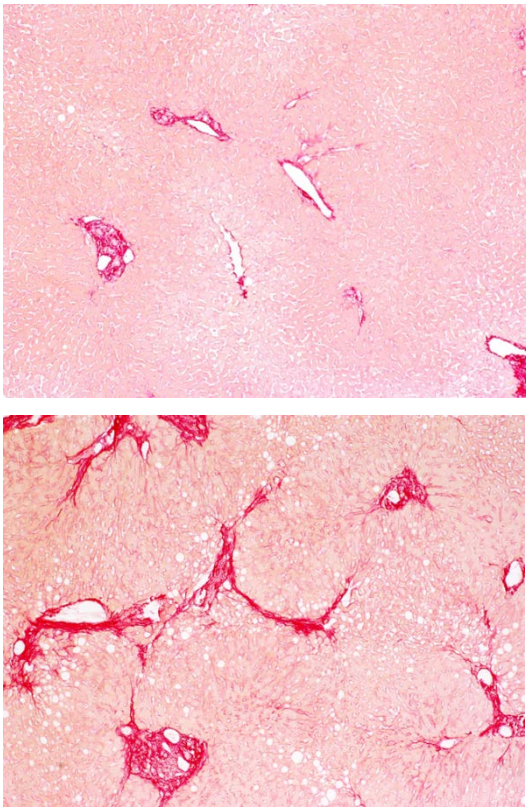
Inflammation Grade 0; NAFLD score 0/8; Fibrosis score 0/4

Inflammation Grade 2; NAFLD score 3/8; Fibrosis score 2/4; Type 2 Diabetes

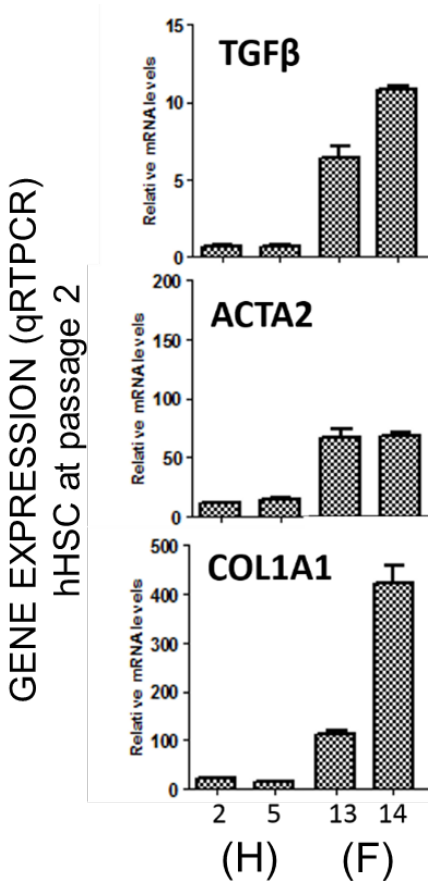
Comparison of HSC derived from healthy- vs. disease-origin tissues

HSC are available from both healthy and diseased donor tissues, and stellate cells isolated from disease-origin tissues retain some features associated with a disease-activated phenotype. Both morphologic and phenotypic differences can be detected. Generally, expression of GFAP is diminished and SMA is increased in disease-origin cells. This suggests that at least some aspects of the disease phenotype are retained in the *in vitro* culture system.





Picrosirius Red staining of tissue from a healthy (top) vs. diseased (bottom) donor.



Characteristics of healthy-origin vs. disease-origin stellate cells

Disease origin tissues are assessed and scored by a board-certified pathologist with respect to steatosis, inflammation, fibrosis, and degree of Non-Alcoholic Fatty Liver Disease (NAFLD). Disease-origin tissues often display a greater degree of steatosis or fibrosis (picrosirius red histological stain, above). In a simple experiment comparing smooth muscle actin (ACTA2), collagen (COL1A1) and TGFβ expression in (2) healthy-origin (H) and (2) disease-origin (F) lots of stellate cells at passage 2, expression of all three activation-associated genes was higher in the disease-origin cells. To learn more about hepatic stellate cells, please visit our website and review the following references from Kisseleva et al.

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Curr Pathobiol Rep. 2013 Sep;1(3):209-214.

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Gastroenterology. 2012 Sep;143(3):765-76.e1-3.

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