



The Role of Embryonic Stem Cell in Expressed RAS (ERAS)

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DESCRIPTION

Hepatic stellate cells (also called lipocytes, Ito cells, fat storing cells, or perisinusoidal cells) contribute 5-8% of total liver resident cells and are situated between sinusoidal endothelial cells and hepatocytes in the space of Disse. HSCs play essential roles in liver growth, immunoregulation, and pathology. They display a noticeable plasticity in their phenotype, gene expression profile, and cellular function. In healthy liver, HSCs remain in an inactive state and store vitamin A mostly as retinyl palmitate in cytoplasmic membrane coated vesicles. Moreover, HSCs typically express neural and mesodermal markers (Glial Fibrillary Acidic Protein (GFAP) and desmin). They possess characteristics of stem cells, like the expression of Wnt and NOTCH, which are required for developmental fate decisions. Activated HSCs display an expression profile highly reminiscent of mesenchymal stem cells. Due to characteristic functions of mesenchymal stem cells, such as differentiation into osteocytes and adipocytes as well as support of hematopoietic stem cells, HSCs were recognized as liver-resident mesenchymal stem cells.

Liver injury, HSCs become triggered and exhibit properties of myofibroblast-like cells. During activation, HSCs produce vitamin A, up-regulate several genes, including α -smooth muscle actin and collagen type I, and down-regulate GFAP. Activated HSCs are multipotent cells, and recent studies exposed a new phase of HSCs plasticity. Physiologically, HSCs signify well known extracellular matrix-producing cells. In some pathophysiological disorders, constant activation of HSCs causes the accumulation of extracellular matrix in the liver and initiates liver diseases, such as fibrosis, cirrhosis, and hepatocellular carcinoma. Therefore, it is valuable to reconsider the impact of different signaling pathways on HSC fate decisions in order to be able to moderate them so that activated HSCs contribute to liver revival but not fibrosis. To date, several growth factors (PDGF, TGF β , and insulin) and signaling pathways have been defined to control HSC activation through effector pathways, including Wnt, NOTCH, Hedgehog, RAS-MAPK, JAK-STAT3, and PI3K-AKT, HIPPO-YAP. However, there is a need to further classify important players that arrange HSC activity and to find

out how they regulate as positive and negative regulators HSC activation in reaction to liver injury. Among these pathways, RAS signaling is one of the earliest that was known to play a role in HSC stimulation and to perform as a node of intracellular signal transduction networking. Therefore, RAS-dependent signaling pathways were the focus of the present study.

Small GTPases of the RAS family are participating in a variety of cellular processes ranging from intracellular metabolisms to propagation, migration, and differentiation as well as embryogenesis and normal progress. RAS proteins respond to extracellular signals and transform them into intracellular responses through interaction with effector proteins. The activity of RAS proteins is extremely controlled through two sets of particular regulators with opposite functions, the guanine nucleotide exchange factors and the GTPase-Activating Proteins (GAPs), as initiators and inactivators of RAS signaling, respectively.

In the current study, the expression profile of different Ras isoforms in HSCs are analyzed and found embryonic stem cell Expressed RAS (ERAS) precisely expressed in quiescent HSCs. To date, ERAS expression has been stated in identical embryonic stem cells and in colorectal, pancreatic, breast, gastric, and neuroblastoma cancer cell lines. From recent studies it's defined that ERAS signifies a unique member of the RAS family with remarkable characteristics. The most reflective features of ERAS include its GAP insensitivity, its unique N terminus among all RAS isoforms, its separate effector choice properties, and the posttranslational change site at its C terminus.

CONCLUSION

During *ex vivo* culture induced initiation of HSCs, the expression of ERAS was sensitively down-regulated at the mRNA and protein level, probably due to an upsurge in promoter DNA methylation. Through observation it is concluded that possible dealings and signaling of ERAS *via* many RAS effectors in HSCs. In contrast, RRAS, MRAS, and RAP2A and also the RAS-RAF-MEK-ERK cascade may control proliferation and differentiation in triggered HSCs.

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