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Opinion Article

Significance of Nrf2 Signaling in Development of Hepatocyte-Like Cells

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DESCRIPTION

Every year, more than 1 million people worldwide pass away from liver disease. The sole treatment option for acute or chronic liver failure linked to end-stage liver dysfunction is liver transplantation. Hepatocyte transplantation, a prospective alternative to liver transplantation, however, shows promise in light of the scarcity of healthy donors. The supply of donor organs is greatly outpacing the need for liver transplants. In order to increase the number of available donor organs, various strategies have been used, such as opt-out organ donation programs, the use of inferior donor organs, such as those from deceased cardiac donors or steatotic (fatty) livers, split-donor transplantation, and living-donor liver transplantation.

Fortunately, unlike most other organs, it is relatively easy to introduce exogenous hepatocytes into the liver parenchyma, indicating that the liver is well-suited for tissue treatment employing hepatocytes produced from induced pluripotent stem cells. In specialized in vitro growth conditions, human Mesenchymal Stem Cells (MSCs) obtained from adipose tissue, bone marrow, or umbilical cord blood can differentiate into Hepatocyte-Like Cells (HLCs). Recent research has also shown that Human Adipose Stem Cell-Derived Hepatocytes (hADSCs) are a potentially scalable and practical substitute for human hepatocytes. Uncertainty still exists regarding the specific method that produces mature HLCs from hADSCs.

Cells are shielded from oxidative stress *via* the nuclear factor erythroid-2 related factor 2 (Nrf2)/Kelch-like ECH-associated protein 1 (Keap1 pathway). When there is oxidative stress, Nrf2 separates from Keap1 and moves into the nucleus, where it controls the transcription of the hemeoxygenase-1 gene and other target genes that code for proteins involved in energy metabolism, detoxification, or other cytoprotective processes. For instance, overexpression of hepatic Nrf2 can functionally heal liver injury in mouse models, and enhanced Nrf2 signaling is purportedly protective in liver injury associated with non-alcoholic steatohepatitis. Furthermore, it was shown that the murine hepatocytes' lipogenesis and CYP3A expression are controlled by the Nrf2/farnesoid X receptor/liver X receptor pathway. Here, they looked into Nrf2's role in the separation of HLCs from hADSCs.

According to a three-step process, they established human HLCs produced from hADSCs in the current study. During the technique, Nrf2 was activated, and knockdown of Nrf2 delayed HLC maturation and impaired some activities. Therefore, Nrf2 may be a significant target for creating highly functional human HLCs. A promising alternative source of human hepatocytes could be the production of hepatocytes from hADSCs. The best cell source for hepatocytes may be MSCs, which can overcome issues with genetic damage, rejection, and ethics, among the donor sources of hepatocytes that have been suggested, including induced pluripotent stem cells, embryonic stem cells, and MSCs.

Patients can be harvested from with little to no invasiveness, especially for the collection of ADSCs, a kind of MSC produced from fat tissues. They discussed how well three-dimensional hADSC-derived HLCs cultured in a system containing a recombinant peptide -piece. But the mechanics underpinning how hepatocytes diverge from hADSCs are not fully understood. To produce hepatocytes from hADSCs, definitive endoderm differentiation is a crucial and early stage. Therefore, producing definitive endoderm in differentiation cultures effectively and consistently is essential for producing hepatocytes of high quality and homogeneity.

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