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Advances in hepatic stem/progenitor cell biology and their therapeutic potential for liver diseases

Dollé Laurent, Stefaan Verhulst, Jan Best, Kimberly Lagaisse, Danielle Blyweert and **Leo A Van Grunsven**
Free University of Brussel, Belgium

The liver is famous for its strong regenerative capacity, employing different modes of regeneration according to type and extent of injury. Mature liver cells are able to proliferate in order to replace the damaged tissue allowing the recovery of the parenchymal function. In more severe scenarios hepatocytes are believed to arise also from a facultative hepatic stem/progenitor cell (HSPC) compartment. In human, severe acute liver failure and liver cirrhosis are also both important clinical targets in which regeneration is impaired, where the role of this stem cell compartment seems more convincing. In animal models, the current state of ambiguity regarding the identity and role of liver progenitor cells in liver physiology dampens the enthusiasm for the potential use of these cells in regenerative medicine. The aim of this talk is to give the basics of liver progenitor cell biology and discuss recent results vis-à-vis their identity and contribution to liver regeneration. In these last couple of years we have learned much about the pathways and conditions involved in HSPC activation thanks to sophisticated genetically modified mouse models. These same models are currently in conflict about the existence and function of HSPCs during liver injury and regeneration. Perhaps novel mouse liver injury models, more representative of human disease, need to be developed to fully unravel the existence, identity and function of HSPCs.

ldolle@vub.ac.be

Combination of body mass index and oxidized low density lipoprotein receptor 1 in prognosis prediction of patients with squamous non-small cell lung cancer

Long Jiang
University of California, USA

Lung cancer, especially non-small cell lung cancer (NSCLC), represents enormous challenges in continuously achieving treatment improvements. Besides cancer, obesity is becoming ever more prevalent. Obesity is increasingly acknowledged as a major risk factor for several types of common cancers. Significant mechanisms overlap in the pathobiology of obesity and tumorigenesis. One of these mechanisms involves oxidized low density lipoprotein receptor 1 (OLR1), as a link between obesity and cancer. Additionally, body mass index (BMI) has been widely used in exploiting the role of obesity on a series of diseases, including cancer. Significantly, squamous NSCLC revealed to be divergent clinical and molecular phenotypes compared with non-squamous NSCLC. Consequently, OLR1 immuno staining score and BMI were assessed by Fisher's linear discriminant analysis to discriminate if progression-free survival (PFS) would exceed 2 years. In addition, the final model was utilized to calculate the discriminant score in each study participant. Finally, 131 patients with squamous NCSLC were eligible for analysis. And a prediction model was established for PFS based on these 2 markers and validated in a second set of squamous NCSLC patients. The model offers a novel tool for survival prediction and could establish a framework for future individualized therapy for patients with squamous NCSLC.

Long.Jiang@ucsf.edu

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