Table 4 Relationship between nutrition risk screening tools among different MELD score.

MELD	RFH-NPT	NRS-2002		Total	P
		Low risk	High risk		
≤10	Low risk	62	5	67	0.004
	High risk	20	67	87	
	Total	82	72	154	
10 ~ 20	Low risk	7	1	8	0.021
	High risk	9	25	34	
	Total	16	26	42	
20 ~ 30	Low risk	2	2	4	0.50
	High risk	0	8	8	
	Total	2	10	12	
> 30	Low risk	0	0	0	-
	High risk	0	5	5	
	Total	0	5	5	

Figure 1

Kaplan–Meier curves of survival analysis for the patients stratified as low risk and high risk using the NRS-2002 scale.

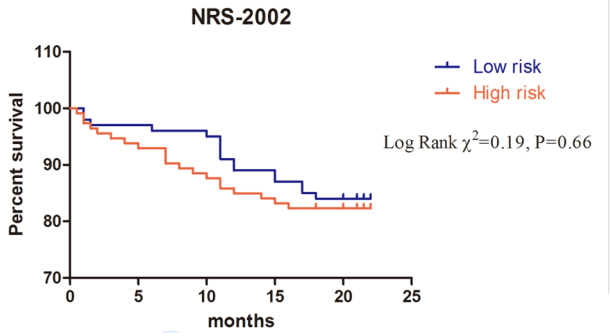
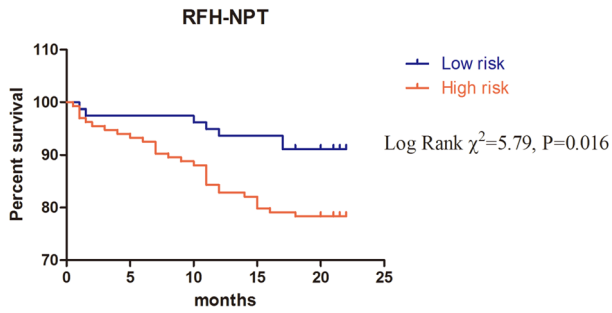


Figure 2

Kaplan–Meier curves of survival analysis for the patients stratified as low risk and high risk using the RFH-NPT scale.



Abstract #345

Fgl2 regulates liver fibrosis progression and reversal by promoting profibrotic infiltrating macrophages maintenance

Ran Tao[1], Wei Yuan[1], Fang Xiao[1], Meiqi Liu[1], Hongwu Wang[1], Di Wu[1], Jiaquan Huang [1], Xiaoping Luo[1], Xiaoyang Wan[1], Qin Ning[1]

[1]Tongji Hospital of Tongji Medical College, Huazhong University of Science and Technology

Introduction: Fibrinogen-like protein 2 (Fgl2) is known as a crucial inflammatory regulator. However, its contribution in the pathogenesis of liver fibrosis remains unclear.
Objectives: We aimed to investigate the molecular mechanism underlying the involvement of Fgl2 in macrophages function in the pathogenesis of liver fibrosis.
Methods: Twenty patients with HBV-induced fibrosis were recruited. FGL2 levels were determined via enzyme-linked immunosorbent assay and reverse transcription-polymerase chain reaction. Genetic ablation of the Fgl2 gene in rodent models were used to evaluate the phenotype of monocyte/macrophages in the liver by using flow cytometry. Administration of muramyl dipeptide (MDP) was used to alleviated fibrosis in fgl2-/- mice. Blood circulating monocytes were further analyzed. We sought investigation on macrophage-dependent regulation of hepatic stellate cells (HSCs) by using primary HSCs and HMs coculture system in vitro. The expression of fibrogenic factors in HSCs were evaluated.
Results: We identified increased Fgl2 expression was associated with high grade of liver fibrosis in chronic Hepatitis B patients and experimental models. Genetic ablation of the Fgl2 alleviated fibrosis progression and promoted reversal during the resolution, which was linked to a restorative phenotype of macrophage in Fgl2-/- mice. Moreover, administration of MDP alleviated fibrosis in fgl2 deficient mice, which was associated with coordinated an increased Ly6Clow phenotype. Fgl2 depletion in macrophages significantly dampened the activation of primary HSCs in vitro in response to macrophage-dependent stimulation.
Conclusion: Fgl2 regulates liver fibrosis by maintaining profibrotic phenotype in resident and infiltrating macrophages, thereby providing novel insights into therapeutic strategy for fibrosis treatment.

Abstract #446

Combination treatment with epigallocatechin gallate and silibinin restored antioxidant defense mechanisms and prevented N-nitrosodimethylamine induced hepatic fibrosis in rats

Joseph George[1], Takahiro Minato[1], Yasuhiro Matsue[1], Yuki Hirakawa[1], Mikihiro Tsutsumi[1], Mutsumi Tsuchishima[1]

[1]Department of Hepatology, Kanazawa Medical University, Uchinada, Ishikawa 920-0293, Japan

Introduction: Hepatic fibrosis is the result of exuberant wound healing response to a persistent stimulus. Epigallocatechin-3-gallate (EGCG) and silibinin are powerful antioxidants present in green tea and milk thistle, respectively. Oxidative stress and reactive oxygen species (ROS) play significant role in the pathogenesis of hepatic fibrosis.
Objectives: Evaluate the combination effect of EGCG and silibinin to prevent experimentally induced liver injury and fibrosis in rats.
Methodology: N-nitrosodimethylamine (NDMA) was used to induce liver injury and fibrosis. A group of animals received 0.2 mg EGCG/100 g body weight orally 2 h prior to NDMA administration. Another

group received silibinin 2 mg/100 g body weight and the next group received both EGCG and silibinin in combination. Alanine transaminase (ALT), aspartate transaminase (AST), osteopontin, collagen type IV, TGF-β1, and hyaluronic acid (HA) were measured in serum. Glutathione, glutathione peroxidase, and malondialdehyde were determined in the liver. Collagen type I, α-smooth muscle actin (α-SMA), 4-hydroxy-2-nonenal (4-HNE), and osteopontin were stained on liver sections.
Results: Serum ALT, AST, osteopontin, collagen type IV, TGF-β1, and HA were significantly decreased after EGCG and silibinin treatment. While glutathione and glutathione peroxidase significantly increased in the liver tissue, malondialdehyde levels markedly decreased indicating improved antioxidant status. Furthermore, staining depicted remarkable decrease in collagen type I, α-SMA, 4-HNE, and osteopontin after EGCG and silibinin treatment with a synergistic effect after the combination therapy.
Conclusion: The data indicates both EGCG and silibinin are effective to protect liver from oxidative stress and ROS induced liver injury and subsequent hepatic fibrogenesis.

Abstract #587

The Role of CX43 in Human Menstrual Blood-Derived Stem Cell's suppression in activating hepatic stellate cell

Sainan Zhang[1], Ya Yang[2], Fen Zhang[3], Lanjuan Li[4]

[1]State Key Laboratory for Diagnosis and Treatment of Infectious Diseases & The First Affiliated Hospital & Zhejiang University School of Medicine, [2]State Key Laboratory for Diagnosis and Treatment of Infectious Diseases & The First Affiliated Hospital, School of Medicine, & Zhejiang University, [3]State Key Laboratory for Diagnosis and Treatment of Infectious Diseases & The First Affiliated Hospital, School of Medicine, & Zhejiang University, [4]State Key Laboratory for Diagnosis and Treatment of Infectious Diseases & The First Affiliated Hospital, School of Medicine, & Zhejiang University

Introduction: Liver fibrosis is a reversible wound healing response to acute or chronic hepatocellular injury from various etiologies. Activation of hepatic stellate cell plays a pivotal role in the development of liver fibrosis. Human menstrual blood-derived stem cells (MenSCs), also known as menstrual blood-derived mesenchymal stem cells, are reported to protect liver from injury. Connexin43(CX43) is a ubiquitous gap junction protein expressed in a wide variety of tissues and organs that regulates celluar functions such as cell growth, differentiation, migration, metabolism and so on.
Objectives: The aims of the study were to verify the hypothesis that CX43 participates in activation of hepatic stellate cell. The protective effect of MenSCs against liver fibrosis was regulated by CX43 expression.
Methodology: In this study, we investigated differential expression in LX2 (an immortalized hepatic stellate cell line) that were cocultured with MenSCs. Gap26, an inhibitor of CX43, was added to clarify the communication between LX2 and MenSCs. CX43 Cell proliferation was tested by CCK8. Protein secretion in culture supernates were tested by ELISA and protein expression of cell were tested by western blotting.
Results: MenSCs suppressed proliferation of LX-2 cells and the secretion of α-SMA and TGF-β1. Expression of CX43 in LX2 increased when cocultured with MenSCs. Inhibition of CX43 suppressed the protective effect of MenSCs against liver fibrosis. MAPK signal pathway maybe the possible function method.
Conclusion: MenSCs suppressed the activation of hepatic stellate cell. Gap junction communication based on CX43 maybe the possible approach that MenSCs protected liver against fibrosis.

Abstract #792

Non-heavy drinking and worsening of non-invasive fibrosis markers in nonalcoholic fatty liver disease: A cohort study

Yong Kyun Cho[1], Yoosoo Chang[2], Yejin Kim[3], Eunju Sung[4], Jiin Ahn[5], Hyun-Suk Jung[6], Kyung Eun Yun[7], Seungho Ryu[8]

[1]Division of Gastroenterology and Hepatology, Department of Internal Medicine, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea, [2]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea & Department of Occupational and Environmental Medicine, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea & Department of Clinical Research Design & Evaluation, Saihst, Sungkyunkwan University, Seoul, South Korea, [3]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea, [4]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea, [5]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea, [6]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea, [7]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea, [8]Center for Cohort Studies, Total Healthcare Center, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea & Department of Occupational and Environmental Medicine, Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Seoul, South Korea & Department of Clinical Research Design & Evaluation, Saihst, Sungkyunkwan University, Seoul, South Korea

The effect of modest alcohol consumption on fibrosis progression in the general population with NAFLD remains unclear. We examined the association of non-heavy alcohol consumption with worsening of non-invasive fibrosis indices in a large-scale, low-risk population with non-alcoholic fatty liver disease (NAFLD). A cohort study was performed in 58,927 Korean adults with NAFLD and low fibrosis scores who were followed for a median of 8.3 years. Non-, light, and moderate drinkers were defined as 0 g/day, 1–9.9 g/day, and 10–29.9 g/day (10–19.9 g/day for women), respectively. Progression from low to intermediate or high probability of advanced fibrosis was assessed using non-invasive indices including NAFLD fibrosis score (NFS) and FIB-4. Parametric proportional hazards model was used to estimate the multivariate-adjusted hazard ratios and 95% confidence intervals. During 347,925.4 person-years of follow-up, 5630 subjects with low FIB-4 progressed to intermediate or high FIB-4. The multivariable-adjusted HRs (95% CI) for worsening of FIB-4 comparing light-drinkers and moderate-drinkers with non-drinkers were 1.06 (0.98–1.16) and 1.29 (1.18–1.40), respectively. Similarly, using NFS, corresponding HRs (95% CI) comparing light-drinkers and moderate-drinkers with non-drinkers were 1.09 (1.02–1.16) and 1.31 (1.23–1.40), respectively. Furthermore, the association of moderate drinkers with worsening of either FIB-4 or NFS remained significant after introducing alcohol use and confounders treated as time-varying covariates.