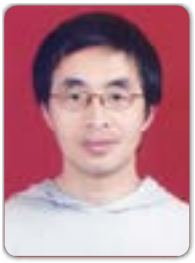
34th Euro-Global Summit on **Cancer Therapy & Radiation Oncology**
&
6th International Conference on **Big Data Analysis and Data Mining**
&
13th International Conference on **Orthopedics, Arthroplasty and Rheumatology**

July 25-27, 2019 London, UK



Wei-Dong Chen

Henan University, China

The nuclear receptor FXR in different diseases

The farnesoid X receptor (FXR) is a key metabolic and homeostatic regulator in digestion system. Our publication has shown that bile acid nuclear receptor FXR is required for the promotion of liver regeneration/repair after physical resection or liver injury, and FXR is a key negative regulator in chronic inflammation and liver carcinogenesis. We found that defective activation of FXR could be an intrinsic defect in aging regenerating livers, and other transcription factors are present as constituents of the multi-protein-DNA complex at the IR-0 element in intron 3 of Foxm1b In liver carcinogenesis, we identified a novel role of FXR in antagonizing c-Jun N-terminal kinase (JNK) signaling pathway by activating SOD3 transcription. FXR may regulate SOD3 expression to suppress ROS production, resulting in decreasing JNK activity. The results highlight FXR as a potential target for drug design for prevention and treatment of liver cancer and insufficient liver regeneration after segmental liver transplantation or resection, which may also have potential implications for treatment of other age-related diseases. In recent days, we found that FXR activation suppresses cervical cancer and ovarian cancer cell proliferation and induces cancer cell apoptosis (unpublished data). These findings identify FXR as a negative mediator not only for digestive system cancers but also reproductive system cancers that may serve as an attractive therapeutic tool for human different cancers.

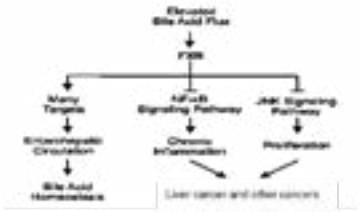


Figure 1. FXR activation suppresses different cancers through antagonizing NF-κB, JNK and other signaling pathways

Recent Publications

1. Lv S Y, Cui B, Yang Y, Du H, Zhang X, Zhou Y, Ye W, Nie X, Li Y, Wang Q, Chen W D, and Wang Y D (2019) Spexin/NPQ induces FBJ osteosarcoma oncogene (Fos) and produces antinociceptive effect against inflammatory pain in the mouse model. Am J Pathol. 189(4):886-899.

2. Feng Q, Chen W D and Wang Y D (2018) Gut microbiota: an integral moderator in health and disease. Front Microbiol. 9:151.

3. Wang Y D, Chen W D, Li C, Guo C, Li Y, Qi H, Shen H, Kong J, Long X, Yuan F, Wang X and Huang W (2015) Farnesoid X receptor antagonizes JNK signaling pathway in liver carcinogenesis by activating SOD3. Mol Endocrinol. 29(2):322-31.

4. Chen W D, Fu X, Dong B, Wang Y D, Shiah S, Moore D D and Huang W (2012) Neonatal activation of the nuclear receptor CAR results in epigenetic memory and permanent change of drug metabolism in mouse liver. Hepatology. 56(4):1499-509.

5. Chen W D, Wang Y D, Zhang L, Shiah S, Wang M, Yang F, Yu D, Forman B M and Huang W (2010) Farnesoid X receptor alleviates age-related proliferation defects in regenerating mouse livers by activating forkhead box m1b transcription. Hepatology. 51(3):953-62.

Biography

Wei-Dong Chen is currently working as a Professor in the School of Medicine, Head of Key Laboratory of Receptor-Mediated Gene Regulation and Drug Discovery, Henan University, P R China. He has received his PhD from Tianjin University, P R China in 2002. He then worked at the GSF in Germany and Beckman Research Institute, City of Hope National Medical Center in USA. Then he served as a Professor at Henan University. He has authored several publications in various journals and books. His publications reflect his research interests in nuclear receptor functions in different diseases.

wdchen666@163.com

Notes: