PPAR Research

Special Issue on
PPARs in the Alcoholic Liver Disease

CALL FOR PAPERS

The peroxisome proliferator-activated receptors (PPARs) are ligand-activated transcription factors that are known to have critical roles in the regulation of lipid homeostasis. Three PPAR isoforms, designated PPARα, PPARβ/δ, and PPARγ, have been identified.

The relationships between ethanol treatment and decreases of hepatic PPARα in mouse and human liver have been demonstrated. PPARα-null mice fed a diet containing a low concentration of ethanol exhibit marked symptoms similar to those observed in patients with alcoholic liver disease (ALD), including hepatomegaly, steatosis, elevated serum aspartate aminotransferase and alanine aminotransferase levels, inflammation, spotty necrosis, and centrilobular fibrosis and apoptosis.

PPARα seems to regulate the expression of the variety of proteins directly or indirectly linked to the increased susceptibility of human and mouse liver to alcohol-induced liver disease regulating the lipid metabolism, controlling the expression of genes involved in the transport, oxidation, and export of free fatty acids. Since fatty liver represents a very common finding in ALD, the effect of ethanol metabolism on PPARα regulated processes has been intensively investigated in the past ten years.

Several studies demonstrated the importance of PPARγ in the activation of hepatic stellate cells (HSC). Active PPARγ is required for the maintenance of the resting "fat storing" phenotype by HSC, and its expression and transcriptional activity decrease during cell activation in culture.

PPARβ/δ is probably the less characterized isoform of the PPAR family. Recent reports demonstrated the role for PPARβ/δ in the regulation of glucose metabolism insulin sensitivity and the protective effects of PPARβ/δ agonist on ethanol-induced liver injury.

Dysregulation of PPARs activity plays a central role in the onset and perpetuation of the mechanisms underlying all steps of the clinical progression in ALD.

We invite authors to submit original research articles as well as review articles that will help in understanding physiological and pathological roles of PPARs in ALD.

Potential topics include, but are not limited to:

- Mechanisms of PPAR protein family dysregulation in ALD
- Role of PPARs in the regulation of lipid metabolism
- PPARs and oxidative stress
- Role of PPARs in the regulation of liver fibrosis
- Dysregulation of PPARs in steatosis and steatohepatitis
- Role of PPARs in cirrhosis and hepatocellular carcinoma

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/ppar/pald/.

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