Expression Analysis of Hepatocyte Transcription Factors in Chronic Liver Disease

Sandip Todkar, Shimpa Sharma, Meghnad Joshi
Department of Stem Cell and Regenerative Medicine, CIR, D Y Patil Hospital & Research Center, Kadamwadi,
D Y Patil Education Society (Deemed to be University) Kolhapur, Maharashtra, India

Abstract No. 33

1) Aims & Objectives :

1) To investigate gene expression profiles of hepatocyte-specific transcription factors (HETFs) in chronic liver disease (CLD).
2) To evaluate their correlation with liver function test (LFT) parameters.
3) To explore the diagnostic and prognostic potential of HETFs as molecular biomarkers.

2) Background & Methods:

Chronic liver disease presents diagnostic challenges due to nonspecific clinical signs and lack of reliable biomarkers. Hepatocyte transcription factors (TFs) are vital regulators of liver metabolism, inflammation, and regeneration.

1) Participants: 100 CLD patients subdivided into NAFLD (n=35), ALD (n=30), Cirrhosis (n=35)10 Healthy controls

2) Sample & Analysis : RNA primers for target genes were designed using NCBI Primer-BLAST. Peripheral blood- RNA extracted (TRIzol method) and converted to cDNA.

3) qPCR conducted for 5 TFs: HNF1A, HNF4A, CEBPA, APO, EGR (GAPDH as standard) Fold change calculated using 2^-ΔΔCt

4) Correlation of gene expression with LFT parameters (ALT, AST, ALP, GGT, Bilirubin)

3) Results:

1) Differential expression of hepatocyte transcription factors (HNF1A, HNF4A, CEBPA, APO, EGR) showed disease-specific trends across NAFLD, ALD, and cirrhosis.
2) HNF1A & HNF4A were significantly downregulated in cirrhosis, suggesting hepatocellular dysfunction.
3) CEBPA & APO were upregulated in NAFLD, indicating active lipid metabolism.
4) EGR displayed variable expression, potentially linked to regeneration or fibrosis.
5) Findings suggest that hepatocyte transcription factors hold promise as biomarkers for chronic liver disease.

4) Charts and Figures:

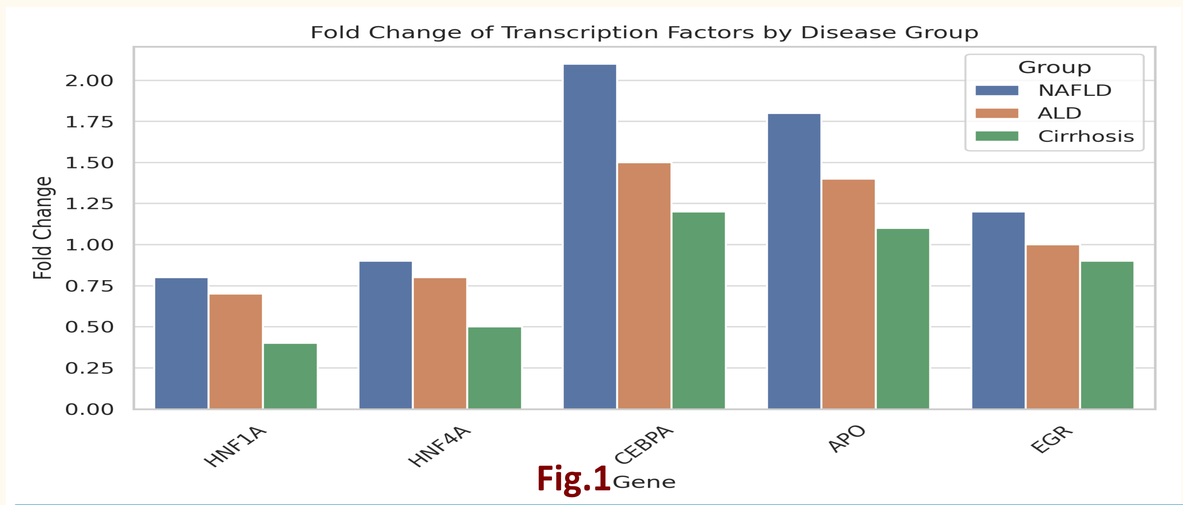

Fig.1

a) Bar Chart (Fold Change by Group)

"Transcription factor expression patterns across NAFLD, ALD, and cirrhosis. HNF1A and HNF4A are downregulated in cirrhosis; CEBPA and APO show highest upregulation in NAFLD.“
Interpretation: Highlights disease-specific expression profiles.

Future plans:

Validate findings in larger cohort (n>200) including liver tissue biopsy-based data Perform RNA-seq for broader transcriptomic analysis

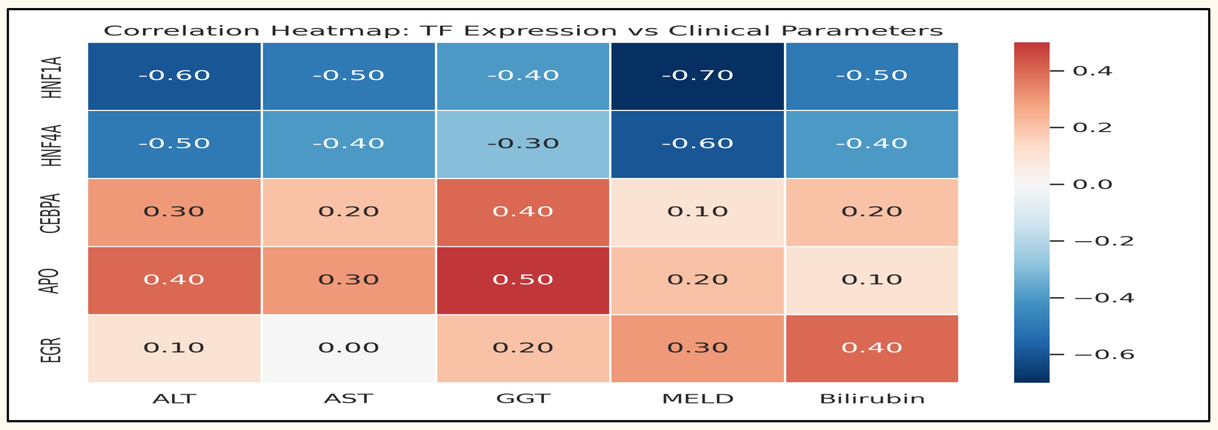

Fig. 2

b) Heatmap (TF vs Clinical Markers)

"Correlation matrix showing relationship between TF expression and liver function scores. Blue = negative correlation (e.g., HNF1A ↔ MELD), Red = positive (e.g., APO ↔ ALT)."
Interpretation: Links molecular expression with clinical status.

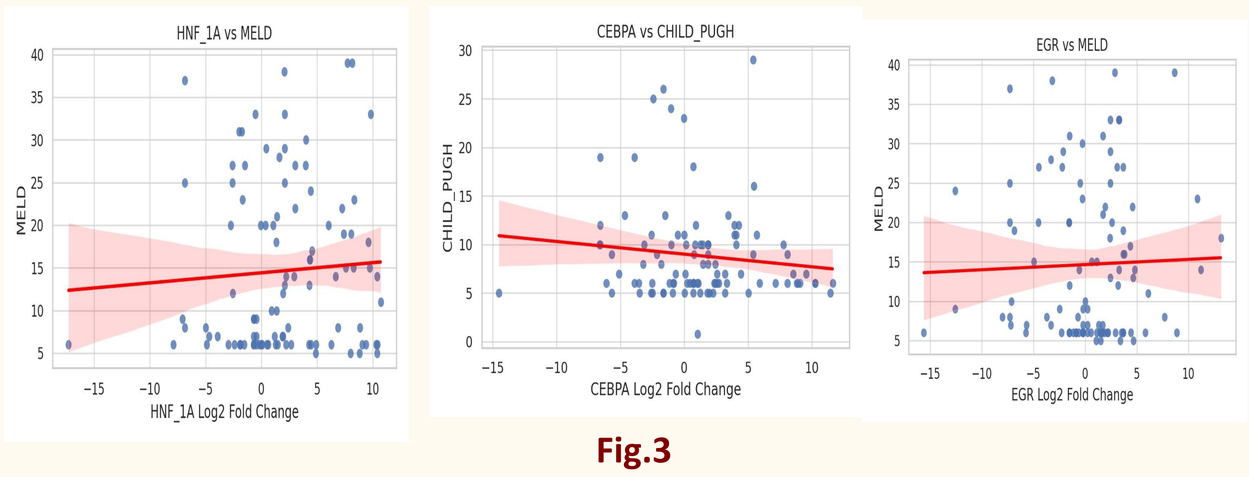

Fig.3

c) Scatter Plots:

"Linear regression plots showing individual TF expression vs clinical severity indices. Significant trends reinforce biomarker potential.“
Interpretation: Shows continuous trends in gene expression and clinical worsening.

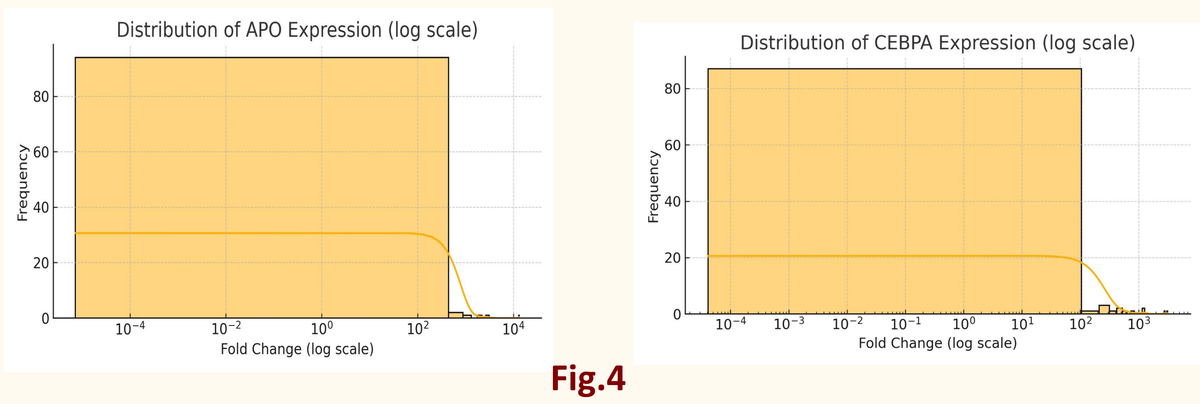

Fig.4

d) Histograms (Expression Distribution in Log Scale)

"Log-scaled distribution of fold-change values reveals expression heterogeneity. Right-skew in CEBPA/APO (NAFLD); suppressed expression in HNF1A/4A (cirrhosis)."
Interpretation: Visualizes variability and clustering in gene expression levels.

5) Conclusion:

Distinct transcriptional patterns in hepatocyte-specific TFs reflect CLD progression and etiology. Their significant correlation with biochemical markers supports their potential use in early detection and monitoring of liver disease severity.

6) Acknowledgements

We thank, D.Y. Patil University, Kolhapur, for their support. We acknowledge the contributions of Akshay Kawale. We are grateful to all patients and healthy volunteers who participated in the study.
Email: sandiptodkar7733@gmail.com

Asian Hepatology Summit – Singapore Hepatology Conference 22-24th August 2025