Chapter 6
Conclusions

- Hepatitis E virus infection in human epithelial (A549) cells elicited inflammatory response through the activation of IRF3 and NF-κB transcription factors.
- Involvement of MyD88 and TRIF dependent signaling pathways in HEV elicited inflammatory response suggesting plausible role of TLR2, TLR3 and TLR4 in recognizing HEV associated molecular patterns.
- Hepatoma cell lines (HepG2/C3A, S10-3 and Huh7.5) supported HEV replication albeit with the differential efficiency due to the cell line specific innate immune response.
- HepG2/C3A cell line eliciting robust innate immune response was comparatively less permissive for HEV replication, while Huh7 derived cell lines S10-3 and Huh7.5, with altered innate pathways, were less responsive to HEV and supported better HEV replication.
- RNA helicase RIG-I is the major PRR molecule involved in sensing HEV RNA replication intermediates and restricting HEV replication in hepatoma cell lines by inducing antiviral effector genes.
- Inhibition of IRF3 mediated innate immune signaling by pharmacological inhibitor BX795 improved HEV replication in HepG2/C3A cells, suggesting the future use of this aspect in establishing a better cell culture model system to understand HEV replication, ORF1 processing and to study host-virus interactions.