

List of Figures

Figure 1	Toll like receptor signaling in viral infection	8
Figure 2	RIG-I like receptor (RLR) mediated signaling in viral infection	12
Figure 3	Signaling pathway activated by type I IFNs (IFN- α/β)	14
Figure 4	Schematic representation of genome organization of HEV.	32
Figure 5	Infection of HepG2/C3A and A549 cells with HEV.	62-63
Figure 6	IFN- β secretion in response to poly (I:C) by A549 cells	65
Figure 7	Relative gene expression in HEV infected A549 cells.	70
Figure 8	Cytokines/chemokines in the cell supernatants.	73
Figure 9	Influenza A infection induces production of inflammatory cytokines/chemokines in A549 cells	75
Figure 10	HEV infection induces IRF3 and NF- κ B activation in A549 cells.	77
Figure 11	HEV induced secretion of inflammatory cytokines/chemokines requires MyD88 adaptor.	81-82
Figure 12	TRIF adaptor knockdown reduces IL-8 induction in HEV infected cells.	83-84
Figure 13	MAVS is dispensable for HEV elicited inflammatory response.	85-86
Figure 14	Influenza A virus infection elicits inflammatory response by recruiting TLR and RLR adaptors.	87-88
Figure 15	Differential replication efficiency of HEV replicon in different hepatoma cell lines	89-90
Figure 16	Interferon α treatment inhibits HEV RNA replication in Huh7.5 cells	94
Figure 17	RIG-I is an essential pattern recognition receptor in sensing HEV RNA replication	95-96
Figure 18	Inhibition of pattern recognition receptor signaling enhances HEV RNA replication efficiency.	97-98
Figure 19	BX795 blocks the phosphorylation and activation of IRF3 leading to suppression of interferon stimulated genes (ISGs).	99
Figure 20	Inhibition of IFN signaling enhances HEV RNA replication efficiency.	100-101