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# MODULATION OF AUTOPHAGY IN A STABLE SUB GENOMIC DENGUE VIRUS REPORTER REPLICON CELLS

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**Autophagy is a dynamic phenomenon operated during cellular starvation and some viral infection. Dengue virus (DENV) utilizes autophagy machinery for its effective replication; however, involvement of its genes in the autophagy induction is obscure. To study, the importance of Dengue viral genes in the autophagy activation, DENV2 NGC sub genomic Renilla luciferase reporter stable Baby Hamster Kidney cells were developed and characterized. The enhancement of initial autophagy markers proteins (LC3 and p62) in stable cells revealed that, dengue virus utilized the autophagy machinery for active replication. The formation of LC3 II and p62 puncta was quite evident, indicating the autophagosome in the stable cells harboring replication of DENV2 sub-genome. High level of GFP expression in the stable cells transfected with GFP-LC3 plasmid indicated the modulation of autophagy. Also, the array of host autophagy related gene expression was studied in DENV2 stable cells by RT2 profiler to decipher cross talk between autophagy and dengue viral replication. In DENV2 stable cells, there was up regulation of the autophagy initiation genes, such as ULK2, *Cathepsin* D, TMEM 74 and ESR1 which are used as raw materials for the induction of autophagy. Surprisingly, there was no elevation in the expression levels of LC3 and p62 genes at the transcription level. This indicates that, DENV2 induces post-translational modification to the LC3 and p62 proteins for the generation of autophagic vesicles along with the aforesaid autophagy initiator proteins. Further, Mycophenolic acid (MPA) treatment inhibited the dengue viral replication by the up-regulation of ATG 4 (codes for the cystein proteases) in stable cells. Subsequent studies concerning the role of individual non-structural protein in the autophagy induction, may provide better understanding of cross talk between autophagy and viral replication, which also may help in the development of novel host targeted antiviral drugs.**

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