<u>In order of importance, list of ten best papers</u>

1. B Hazra, K L Kumawat, A Basu (2017) The host microRNA miR-301a blocks the IRF1-mediated neuronal innate immune response to Japanese encephalitis virus infection. ***Science Signaling*** 10(466):eaaf5185. (Cover page article)
   (*We have showed that inhibition of miR-301a in mouse brain rescued the production of IRF1 and SOCS5, increased the generation of IFN-γ, and reduced the extent of JEV replication, thus improving mouse survival. Thus, our study suggests that the JEV induced expression of miR-301a assists viral pathogenesis by suppressing IFN production, which might be targeted by antiviral therapies*)
2. M Bhaskar, S Mukherjee, and A Basu (2021) Involvement of RIG-I Pathway in Neurotropic Virus-Induced Acute Flaccid Paralysis and Subsequent Spinal Motor Neuron Death. ***mBio*** 12(6):e0271221
   (*Here we have showed that Retinoic Acid Inducible Gene-1 is involved in neurotropic virus induced acute flaccid paralysis. We have also showed that the death of motor neuron happened via interferon independent pathway*)
3. B Hazra, S Chakraborty, M Bhaskar, S Mukherjee, A Mahadevan, A Basu (2019). miR-301a regulates inflammatory response to Japanese Encephalitis Virus infection via suppression of NKRF activity. ***Journal of Immunology*** 5;203(8):2222-2238
   (*We have observed that JEV infection induces classical activation (M1) of microglia that drives the production of proinflammatory cytokines while suppressing alternative activation (M2) that could serve to dampen the inflammatory response. Thus, our study suggests that the JEV-induced expression of miR-301a positively regulates inflammatory response by suppressing NKRF production, which might be targeted to manage viral-induced neuroinflammation*).
4. M C Thounajam, K Kundu, D K Kaushik, S Swaroop, A Mahadevan, S K Shankar, and A Basu (2014) MicroRNA-155 Regulates Japanese Encephalitis Virus Induced Inflammatory Response by Targeting src Homology 2-Containing Inositol Phosphatase-1**.** ***Journal of Virology*** 88(9): 4798–4810
   (*Inhibition of SHIP1 by miR-155 resulted in higher beta interferon (IFN-b) and proinflammatory cytokine production through activation of TANKbinding kinase 1 (TBK-1). Based on these observations, we conclude that miR-155 modulates the neuroinflammatory response during JEV infection via negative regulation of SHIP1*

expression. Thus, modulation of miR-155 could be a novel strategy to regulate JEV-induced neuroinflammation).

5. S Ghosh, K Dutta and A Basu (2013) Chandipura Virus induces Neuronal Death through Fas-mediated Extrinsic Apoptotic Pathway. ***Journal of Virology*** 87(22):12398-406
   (*The present study elucidates the mechanism of cell death in neurons after CHPV infection that answers the basic cause of CHPV-mediated neurodegeneration. Knocking down the expression of caspase-3, the final executioner of apoptosis, in neuronal cell by endoribonuclease-prepared small interfering RNA (siRNA) validated its pivotal role in CHPV-mediated neurodegeneration by showing reduction in apoptosis after CHPV infection*)
6. A Ghoshal, S Das, S Ghosh, M K Mishra, V Sharma, P Koli, E Sen and **A Basu** (2007) Proinflammatory mediators released by activated microglia induces neuronal death in Japanese Encephalitis. Glia 55:483-496
   (*We have showed that microglia mediated inflammation is critical for neuronal death following Japanese Encephalitis Virus infection. Our findings suggest that the increased microglial activation following JEV infection influences the outcome of viral pathogenesis. It is likely that the increased microglial activation triggers bystander damage, as the animals eventually succumb to infection*)
7. S Chakraborty, and **A Basu** (2022) miR-451a Regulates Neuronal Apoptosis by Modulating 14-3-3ζ-JNK Axis upon Flaviviral Infection. ***mSphere*** 7(4): e0020822
   (*We have identified a novel miRNA which is responsible for neuronal death in flaviviral infection. Hence, findings from this study provide a possible therapeutic target for preventing JEV/WNV-induced neuronal apoptosis thus improving disease outcome in flaviviral infection-associated encephalitis*)
8. S Mukherjee, I Akbar, R Bhagat, B Hazra, A Bhattacharyya, P Seth, D Roy, **A Basu** (2019) Identification and classification of hubs in miRNA target gene networks in human neural stem/progenitor cells following Japanese encephalitis virus infection. ***mSphere*** 4(5). pii: e00588-19.
   (*We identified two types of hub genes in the protein protein interaction network, namely, connector hubs and provincial hubs. These two types of miRNA target hub genes critically influence the participation strength in the networks and thereby significantly impact up- and downregulation in several key biological pathways. Computational analysis of the PPI networks identifies key protein interactions and*

hubs in those modules, which opens up the possibility of precise identification and classification of host factors for viral infection in Neural Stem Progenitor Cells).

9. S Mukherjee, N Singh, N Sengupta, M Fatima, P Seth, A Mahadevan, S K Shankar, A Bhattacharyya, **A Basu** (2017) Japanese encephalitis virus induces human neural stem/progenitor cell death by elevating GRP78, PHB and hnRNPC through ER stress. ***Cell Death & Disease***; 8(1):e2556
   (*Our investigations uncovered three viral RNA interacting proteins GRP78, Prohibitin (PHB) and hnRNPC, whose suppression not only reduced viral infection, but also minimized stress response and resultant apoptosis. Hence, we propose these proteins as cardinal modulators of ER stress progressing to apoptosis in human neural stem cells, post JEV infection*).
10. R Kumar, **A Basu**, S Sinha, Das M, Tripathi P, Jain A, Kumar C, Atam V, Khan S, Singh AS (2016) Role of oral Minocycline in acute encephalitis syndrome in India - a randomized controlled trial. ***BMC Infect Dis***. 2016 Feb 4;16(1):67.
    (*We have conducted a randomized, controlled trial of nasogastric/oral minocycline in JE and AES at a single centre in Uttar Pradesh, northern India. A trend towards better outcomes was observed with minocycline, especially in those patients who survived the initial day in hospital. These findings should form the basis for planning a larger study and possibly including minocycline in the initial management of AES as seen here*).