List of 10 publications highlighting important contributions in the area of Virology

S.No:	Author, Title and Journal	Findings (In Brief)
1.	Mishra R, Chhatbar C, **Singh SK** (2012), HIV-1 Tat C-mediated regulation of tumor necrosis factor receptor-associated factor-3 by microRNA 32 in human microglia, ***Journal of Neuroinflammation***, Jun 18;9:131. doi: 10.1186/1742-2094-9-131	1. **This study by Mishra *et al* reported the role of a microRNA, miR-32, in regulating the neuroinflammation observed in HIV-associated neurological disorder (HAND).** 2. The findings of this study show that HIV-1 Tat C upregulates miR-32 in human microglial cells which in turn downregulates the expression of TRAF3 3. We have further shown that TRAF3 levels are inversely related to IRF3/7 expression, affecting the regulation of inflammatory genes, leading to neuroinflammation 4. The study thus reveals a potential mechanism by which Tat C induces miRNA-mediated dysregulation of an immune adaptor molecule TRAF3 in human microglial cells.
2.	Mishra R, **Singh SK (2013)**, HIV-1 Tat C modulates expression of miRNA-101 to suppress VE-Cadherin in Human Brain Microvascular Endothelial Cells, ***The Journal of Neuroscience*** 33(14):5992-6000; doi:10.1523/JNEUROSCI.4796-12.2013.	1. **The findings, of this study provide crucial insights on how the HIV-1 Tat C protein leads to neurovascular damage by disrupting the integrity of blood-brain barrier.** 2. This study identifies the molecular mechanism by which HIV-1 disrupts the blood-brain barrier through miR-101 mediated suppression of an adherent junction protein VE-Cadherin. 3. The findings of the study report that HIV-1 Tat C protein increased the expression of miR-101 in human brain microvascular endothelial cells, which lead to downregulation of VE-Cadherin and subsequently a tight junction protein Claudin-5. Such perturbation may compromise the integrity of Blood Brain Barrier and helpful in entry of HIV into brain.
3.	Jadhav V; Krause KH; **Singh SK**, 2014, HIV-1 Tat C modulates NOX2 and NOX4 expressions through miR-17 in Human Microglial Cells, ***Journal of Neurochemistry***. Dec; 131(6):803-15	1. **The study by Jadhav *et al* reported the role of miR-17 in regulating the oxidative stress and neuroinflammation in HIV-1 Tat C exposed human microglial cells.** 2. The study reported that Tat C downregulates miR-17, which leads to increased expression of reactive oxygen species (ROS) regulating genes NOX2 and NOX4, ultimately resulting in increased cytokine production. 3. The findings of this study highlight potential therapeutic targets to mitigate neurocognitive damage in HIV patients.
4.	Manocha GD, Mishra R, Sharma N, Kumawat KL, Basu A, **Singh SK** (2014), Regulatory role of TRIM21 in type-I interferon pathway in Japanese encephalitis	1. **The study by Manocha *et al* highlights the role of a E3 ubiquitin-protein ligase, TRIM21, as a crucial modulator of immune responses during Japanese Encephalitis virus (JEV) induced encephalitis.**

	virus infected human microglial cells, ***Journal of Neuroinflammation***, Feb 1;11:24. doi: 10.1186/1742-2094-11-24.	2. The study reported that JEV infection in human microglial cells increased the expression of TRIM21, which through a feedback mechanism negatively regulates IRF-3 phosphorylation, leading to reduced IFN-β production. 3. The findings of this study suggest potential therapeutic target to manage neuroinflammation and enhance the antiviral state in JEV infected patients.
5.	Sharma N, Verma R, Kumawat KL, Basu A, **Singh SK** (2015), miR-146a suppresses cellular immune response during Japanese encephalitis virus JaOArS982 strain infection in human microglial cells, ***Journal of Neuroinflammation***, Feb 18;12:30. doi: 10.1186/s12974-015-0249-0	1. **This study by Sharma *et al* identifies the strain-specific modulation of immune responses, revealing how the JaOArS982 strain of JEV upregulates miR-146a to suppress the antiviral signaling pathways in human microglial cells.** 2. The study reported that JEV infection elevates miR-146a leading to downregulation of key immune response genes TRAF6, IRAK1, IRAK2, STAT1, IFIT-1 and IFIT-2 together with suppressed activation of NF-κB.
6.	Rastogi M, **Singh SK**, 2020, Zika Virus NS1 affects the Junctional Integrity of Human Brain Microvascular Endothelial Cells, ***Biochimie***, 176 (2020), 52-61, 10.1016/j.biochi.2020.06.011.	1. **The study by Rastogi *et al* reports the mechanisms by which Zika virus' Non- Structural Protein 1(NS1) leads to neurovascular damage, specifically through the ROS mediated disruption of the endothelial barrier.** 2. The findings of the study show that exposure of human brain microvascular endothelial cells to ZIKV NS1 disrupts the tight and adherent junction proteins, increases the endothelial barrier permeability, elevates the ROS levels which negatively impact the tyrosine kinase, PYK2 and phosphatases, SHP2. 3. The findings suggest potential therapeutic strategies to address the neurological complications and compromised blood-brain barrier that are associated with Zika virus infections.
7.	Shukla A, Rastogi M, **Singh SK** (2021), Zika virus NS1 suppresses the innate immune responses via miR-146a in human microglial cells, ***International Journal of Biological Macromolecules*** Dec 15;193(Pt B):2290-2296. doi: 10.1016/j.ijbiomac.2021.11.061	1. **The study by Shukla *et al* demonstrates the bystander role of ZIKV-NS1 in suppressing the pro-inflammatory and cellular antiviral responses through miR-146a in human microglial cells.** 2. The study shows that ZIKV-NS1 induces miR-146a, which in turn suppresses the ROS activity, expression of TRAF6 and STAT1 in human microglial cells. 3. The study also reported the reduction in NF-κB activity thus suggesting how ZIKV-NS1 suppresses immune responses through human microglial cells.
8.	Bharadwaj U, **Singh SK** (2023), Zika Virus NS1 suppresses VE-Cadherin via hsa-miR-29b-3p/DNMT3b/MMP-9 pathway in Human Brain Microvascular	1. **This study elucidates the miRNA-mediated mechanisms of neurovascular damage in CNS through dysregulation of a metalloproteinase and a DNA methylation enzyme during ZIKV NS1 exposure.**

	Endothelial Cells; ***Cellular Signalling*** DOI: 10.1016/j.cellsig.2023.110659	2. The results of this study show that ZIKV NS1 compromises the Blood brain barrier through miR-29b/DNMT3b/MMP9 signaling pathway
9.	Pandey N, Rastogi M, **Singh SK** (2021), Chandipura virus dysregulates the expression of hsa-miR-21-5p to activate NF-κB in human microglial cells. ***J Biomed Sci***. doi: 10.1186/s12929-021-00748-0.	1. **The study by Pandey *et al* elucidates the miRNA-mediated mechanisms underlying Chandipura virus-induced neuroinflammation.** 2. This study, for the first time, provides crucial insights to the miRNA-mediated regulatory mechanisms of inflammatory pathways during the Chandipura virus (CHPV) infection in human microglial cells. 3. The study shows that CHPV infection induces miR-21, which in turn promotes activation of NF-κB through PI3K/Akt pathway resulting in increased pro-inflammatory state.
10.	Pandey N, **Singh SK** (2023), MicroRNA-155 triggers a cellular antiviral immune response against Chandipura virus in human microglial cells, ***Microbes and Infection***. 2023 Jun 14;105173. doi: 10.1016/j.micinf.2023.105173	1. **This study provides insights into the miR-155 mediated activation of antiviral state against CHPV by modulating the type-I interferon signaling.** 2. The study shows that the expression of miR-155 increases during CHPV infection creating a antiviral state by activating interferon stimulated genes (ISG56 and ISG54) through SOCS1/STAT1 pathway 3. The findings of this study provide a clue to how Chandipura virus infection acts as a double-edged sword leading to CNS damage.