RIG-I Promotes Protective Type I Interferon Production by Human Glial Cells During Bacterial Meningitis.

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Globally, 1.2 million cases of meningitis are attributed to bacterial agents, contributing to 250,000 mortalities annually. Meningitis is infection of the meninges which provides protection to the brain and spinal cord. During bacterial meningitis, potent inflammatory responses lead to life-threatening injuries and permanent neurological damage. Previous data indicates that resident brain cells recognize bacterial motifs through host receptors, leading to the production of immune mediators. Recent data in peripheral cell types demonstrates a novel role for retinoic acid-inducible gene I (RIG-I) in identifying bacteria and stimulating type I interferons (IFNs). However, the role of RIG-I in initiating IFN production during bacterial CNS infection has not been explored. In this study, we demonstrate upregulation of RIG-I during infection by relevant pathogens including *Neisseria meningitidis* and *Streptococcus pneumoniae*. Additionally, we observe significant IFN production and subsequent increases of interferon-stimulated genes (ISGs) by infected microglia. We have attributed these responses, in part, to RIG-I as knockdown of this receptor resulted in reduced IFN levels. Importantly, we show that RIG-I-dependent-IFN responses contribute to reduced bacterial burden, promoting increased host cell viability. Collectively, these findings indicate a protective role for RIG-I during bacterial meningitis, suggesting its potential as a novel target for therapeutic intervention.

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