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Editorial: Interaction between virus and host cell: how do viruses cause cellular dysregulation?

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Editorial on the Research Topic

Interaction between virus and host cell: how do viruses cause cellular dysregulation?

Viruses are opportunistic pathogens that hijack host cell machinery, ensuring that host cell cycle, apoptosis, immunity, and essential cellular signaling cascades are employed only for their own replication. Other viruses, namely oncogenic viruses, control and dysregulate cellular oncogenic pathways, leading directly or indirectly to the development of carcinoma or malignancy. These viruses include hepatitis B virus (HBV, DNA virus), human papillomavirus (HPV, DNA virus), human herpesvirus-8 (HHV-8, DNA virus), Merkel cell polyomavirus (MCPyV, DNA virus), Epstein-Barr Virus (EBV, DNA virus), hepatitis C virus (HCV, RNA virus), and human T-cell lymphotropic virus-1 (HTLV-1, RNA virus). The high prevalence and extended epidemiology of these oncogenic viral infections in developing countries such as those in Sub-Saharan Africa (Diakite et al.) is alarming. It is important to explore early and affordable precancer molecular markers with high reliability and predictive value in these populations. For their use in studying early diagnoses of these viral infections and understanding the role cellular factors play in establishing viral infections and their long-term consequences (e.g. carcinoma), transcriptomic and proteomic studies of virus-infected cells and single-cell analysis have been attracting more attention. These studies could globally revolutionize the approach taken to translational research. Interestingly, the viral infection consequences may extend beyond virus clearance due to treatment with direct-acting antivirals. To this point, Elbahrawy et al. emphasized the importance of following up on pretreatment and posttreatment predictors of hepatocellular carcinoma (HCC) and recommended HCC surveillance in recovered HCV patients with compensated advanced chronic liver disease. The post-treatment complications in HCV patients might be caused by remaining HCV-induced epigenetic alterations, immune dysregulations, and hepatic parenchymal injuries, leading to *de novo* HCC occurrence.

This Research Topic also gathers contributions that focused on the intracellular trafficking networks of human-cytomegalovirus (HCMV) and elucidate the strategies that HCMV employs to hijack and dysregulate the intracellular trafficking networks to permit viral fitness and replication (Mosher et al.). Cell infection with viruses has always been associated with the intracellular release of different types of RNAs including long non-coding RNA (IncRNA), MicroRNA (miRNA), protein-coding mRNA, and other RNAs. These RNA species play important roles in virus–host interactions, viral pathogenicity, and regulating innate antiviral responses. In the minireview article by Nangare et al., the authors highlighted the importance of secreted miRNA species during Chikungunya viral infections and their possible role in developing candidate cellular miRNA-based therapies against this virus.

This Research Topic brings to focus the need to intensify research efforts and funding to understand the cellular networks that are induced following viral infections and to define regulated and dysregulated cellular elements/pathways during viral infections. This could enable these elements to be applied as markers of virus-induced complications such as malignancy or provide novel approaches to use them as druggable targets.

Author contributions

AM: Conceptualization, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing.

Conflict of interest

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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