Special Issue

Marek's Disease Virus

Message from the Guest Editors

Extensive studies in the last few years have identified some of the major viral proteins that contribute directly to the neoplastic transformation and development of tumours, such as the major oncoprotein Meg, MDVencoded microRNAs, the virus-encoded telomerase RNA (vTR) and viral telomeric repeats (TMRs). While these studies have undoubtedly provided insights into the direct determinants of neoplastic transformation, the role of the majority of other viral proteins may also be critical to the understanding of viral pathogenicity; hence, the development of the vaccine development needs to be explored to ensure better protection of chickens from this deadly disease. For this Special Issue, we invite submissions that provide deeper insights into important aspects of MDV infection, lytic replication, latency, transformation, latent to lytic switch, reactivation, pathogenesis, diagnostics, immune response to MD, molecular epidemiology, and the evolution of the field strains.

Guest Editors

Prof. Dr. Sanjay Reddy

Department of Veterinary Pathobiology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX 77943, USA

Dr. Yongxiu Yao

The Pirbright Institute, Pirbright GU24 ONE, UK

Prof. Dr. Blanca Lupiani

Department of Veterinary Pathobiology, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station TX 77943, USA

Deadline for manuscript submissions

closed (31 December 2022)



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About the Journal

Message from the Editor-in-Chief

Viruses (ISSN 1999-4915) is an open access journal which provides an advanced forum for studies of viruses. It publishes reviews, regular research papers, communications, conference reports and short notes. Our aim is to encourage scientists to publish their experimental and theoretical results in as much detail as possible. There is no restriction on the length of the papers. The full experimental details must be provided so that the results can be reproduced. We also encourage the publication of timely reviews and commentaries on topics of interest to the virology community and feature highlights from the virology literature in the 'News and Views' section.

Electronic files or software regarding the full details of the calculation and experimental procedure, if unable to be published in a normal way, can be deposited as supplementary material.

Editor-in-Chief

Dr. Eric O. Freed Director, HIV Dynamics and Replication Program, Center for Cancer Research, National Cancer Institute, Frederick, MD 21702-1201, USA

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