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0521832489 - Molecular Pathogenesis of Virus Infections

Edited by P. Digard, A. A. Nash and R. E. Randall

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Molecular pathogenesis of virus infections

Virus and prion diseases remain a major public health threat, in both developed and developing countries. The worldwide HIV pandemic is but one example of a newly emerged virus disease; other potential threats come from exotic viruses such as SARS, Ebola and Hantaan viruses. Older human viruses such as influenza, papilloma, herpes and the hepatitis viruses still cause major health problems. Furthermore, as well as causing acute infections, some viruses may also establish persistent infections which can lead to the development of chronic diseases, including cancer. This symposium book covers central factors that influence the pathogenicity of virus and prion infections. Topics range from innate and adaptive immune responses and virus evasion of host defences to details of selected virus–host interactions, including those involving dengue virus, HIV, influenza viruses, coronaviruses, hepatitis C virus, herpesviruses, papillomaviruses, African swine fever virus and poxviruses.

Paul Digard is a Lecturer in Virology in the Department of Pathology at the University of Cambridge, UK.

Anthony A. Nash is Professor of Veterinary Pathology in the Division of Veterinary Biomedical Sciences at the University of Edinburgh, UK.

R. E. Randall is Professor of Molecular Virology in the School of Biology at the University of St Andrews, UK.

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Front cover illustration: Coloured scanning electron micrograph of a cluster of
coronavirus particles. Eye of Science / Science Photo Library.

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CONTRIBUTORS

Alcami, A.

Department of Medicine, University of Cambridge, Addenbrooke's Hospital, Cambridge, UK, and Department of Molecular and Cellular Biology, Centro Nacional de Biotecnología (CSIC), Campus Universidad Autónoma, Cantoblanco 28049 Madrid, Spain

Alejo, A.

Department of Medicine, University of Cambridge, Addenbrooke's Hospital, Cambridge, UK, and Department of Molecular and Cellular Biology, Centro Nacional de Biotecnología (CSIC), Campus Universidad Autónoma, Cantoblanco 28049 Madrid, Spain

Andino, R.

Department of Microbiology and Immunology, University of California, San Francisco, CA 94143-2280, USA

Barron, R. M.

Institute for Animal Health, Neuropathogenesis Unit, Ogston Building, West Mains Road, Edinburgh EH9 3JF, UK

Barry, G.

Centre for Infectious Diseases, College of Medicine and Veterinary Medicine, University of Edinburgh, Edinburgh EH9 1QH, UK

Borrow, P.

Viral Immunology Group, The Edward Jenner Institute for Vaccine Research, Compton, Newbury RG20 7NN, UK

Breakwell, L.

Centre for Infectious Diseases, College of Medicine and Veterinary Medicine, University of Edinburgh, Edinburgh EH9 1QH, UK

Decman, V.

Department of Ophthalmology and Graduate Program in Immunology, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213, USA

Dixon, C. M.

Centre for Infectious Diseases, College of Medicine and Veterinary Medicine, University of Edinburgh, Edinburgh EH9 1QH, UK

Dixon, L. K.

Institute for Animal Health, Pirbright Laboratory, Ash Road, Pirbright, Woking GU24 0NF, UK

Dye, C.

Department of Molecular and Cellular Medicine, University of Bristol, Bristol BS8 1TD, UK

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viii Contributors

Ebrahimi, B.

Centre for Comparative Infectious Diseases, University of Liverpool, Duncan Building, Daulby Street, Liverpool L69 3GA, UK

Fazakerley, J. K.

Centre for Infectious Diseases, College of Medicine and Veterinary Medicine, University of Edinburgh, Edinburgh EH9 1QH, UK

Freeman, M. L.

Department of Ophthalmology and Graduate Program in Molecular Virology and Microbiology, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213, USA

Gray, L.

Bute Medical School, University of St Andrews, Bute Medical Buildings, Westburn Lane, St Andrews, Fife KY16 9TS, UK

Haller, O.

Abteilung Virologie, Institut für Medizinische Mikrobiologie und Hygiene, Universität Freiburg, D-79008 Freiburg, Germany

Hartman, A. L.

Special Pathogens Branch, Division of Viral and Rickettsial Diseases, Centers for Disease Control and Prevention, Atlanta, GA 30306, USA

Hendricks, R. L.

Departments of Ophthalmology, Immunology and Molecular Genetics and Biochemistry, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213, USA

Herrington, C. S.

Bute Medical School, University of St Andrews, Bute Medical Buildings, Westburn Lane, St Andrews, Fife KY16 9TS, UK

Hoffmann, E.

Division of Virology, Department of Infectious Diseases, St. Jude Children's Research Hospital, 332 North Lauderdale St, Memphis, TN 38105, USA

Hughes, D.

Centre for Comparative Infectious Diseases, University of Liverpool, Duncan Building, Daulby Street, Liverpool L69 3GA, UK

Jolly, C.

Bute Medical School, University of St Andrews, Bute Medical Buildings, Westburn Lane, St Andrews, Fife KY16 9TS, UK

Kochs, G.

Abteilung Virologie, Institut für Medizinische Mikrobiologie und Hygiene, Universität Freiburg, D-79008 Freiburg, Germany

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Lemon, S. M.

Department of Microbiology & Immunology, Institute for Human Infections & Immunity,
University of Texas Medical Branch, Galveston, TX 77555-0428, USA

Li, K.

Department of Microbiology & Immunology, Institute for Human Infections & Immunity,
University of Texas Medical Branch, Galveston, TX 77555-0428, USA

Lipatov, A. S.

Division of Virology, Department of Infectious Diseases, St. Jude Children's Research
Hospital, 332 North Lauderdale St, Memphis, TN 38105, USA

Manson, J. C.

Institute for Animal Health, Neuropathogenesis Unit, Ougston Building, West Mains Road,
Edinburgh EH9 3JF, UK

Mongkolsapaya, J.

Department of Immunology, Hammersmith Hospital, Imperial College, Du Cane Road,
London W12 0NN, UK

Nichol, S.

Special Pathogens Branch, Division of Viral and Rickettsial Diseases, Centers for Disease
Control and Prevention, Atlanta, GA 30306, USA

Roaden, L.

Centre for Comparative Infectious Diseases, University of Liverpool, Duncan Building,
Daulby Street, Liverpool L69 3GA, UK

Ruiz-Argüello, M. B.

Department of Medicine, University of Cambridge, Addenbrooke's Hospital, Cambridge,
UK, and Centro de Investigación en Sanidad Animal (INIA), Valdeolmos, Madrid, Spain

Screaton, G.

Department of Immunology, Hammersmith Hospital, Imperial College, Du Cane Road,
London W12 0NN, UK

Siddell, S.

Department of Molecular and Cellular Medicine, University of Bristol, Bristol BS8 1TD,
UK

Stewart, J. P.

Centre for Comparative Infectious Diseases, University of Liverpool, Duncan Building,
Daulby Street, Liverpool L69 3GA, UK

Towner, J. S.

Special Pathogens Branch, Division of Viral and Rickettsial Diseases, Centers for Disease
Control and Prevention, Atlanta, GA 30306, USA

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x Contributors

Turnbull, E.

Viral Immunology Group, The Edward Jenner Institute for Vaccine Research, Compton, Newbury RG20 7NN, UK

van Rij, R. P.

Department of Microbiology and Immunology, University of California, San Francisco, CA 94143-2280, USA

Weber, F.

Abteilung Virologie, Institut für Medizinische Mikrobiologie und Hygiene, Universität Freiburg, D-79008 Freiburg, Germany

Webster, R. G.

Division of Virology, Department of Infectious Diseases, St. Jude Children's Research Hospital, 332 North Lauderdale St, Memphis, TN 38105, USA

Whitton, J. L.

Department of Neuropharmacology, CVN-9, The Scripps Research Institute, 10550 N Torrey Pines Rd, La Jolla, CA 92037, USA

The molecular mechanisms of replication as well as the pathogenesis of several coronaviruses have been actively studied since the 1970s. Some of the animal viruses, such as porcine transmissible gastroenteritis virus (TGEV), bovine coronavirus (BCoV), and avian infectious bronchitis viruses (IBV), are of veterinary importance. The extensive studies of the pathogenesis of MHV and the resulting host immune response have been reviewed (206, 214, 242). Infection of macrophages then leads to viremia and systemic spread of the virus, including inflammation of the abdominal and thoracic cavities and causing occasional ocular and neurological disorders (1, 71). A complication of FIPV infection involves immune-mediated pathology (138). Regarding the molecular pathogenesis, the hemagglutinin protein (H) plays a crucial role both in the antigenic recognition and the viral interaction with SLAM and nectin-4, the host cell's receptors. These cellular receptors have been studied widely as CDV receptors in vitro in different cellular models. chimeric viruses that are to be employed in different fields such as pathogenesis studies, vaccine development, and gene therapy vectors [13]. As with other members of the Paramyxoviridae family, the H glycoprotein facilitates the virus binding to the host cell membrane and the F protein achieves the viral and the host membrane's fusion, enabling the viral RNP's entrance into the cytoplasm [12]. CDV proteins have a specific activity regarding virus replication and in the infection cycle. Epstein-Barr virus (EBV)-infected humanized mice reproduce cardinal features of EBV-associated B-cell lymphoproliferative disease and EBV-associated hemophagocytic lymphohistiocytosis (HLH). Erosive arthritis morphologically resembling rheumatoid arthritis (RA) has also been recapitulated in these mice. Low-dose EBV infection of humanized mice results in asymptomatic, persistent infection. In this review, recent findings on the recapitulation of human EBV infection and pathogenesis in these mouse models, as well as their application to preclinical studies of experimental anti-EBV therapies, are described. Infections with Epstein-Barr virus (EBV) can be initiated in mice by adding components of the human immune system. @article{Horzinek1987MolecularPO, title={Molecular pathogenesis of virus infections}, author={Marian C. Horzinek}, journal={Experientia}, year={1987}, volume={43}, pages={1193-1196} }. Marian C. Horzinek. Although a very wide range of viral diseases exists in vertebrates, certain generalizations can be made regarding pathogenetic pathways on the molecular level. The presentation will focus on interactions of virions and their components with target cells. Using coronaviruses as examples the changes in virulence have been traced back to single mutational events; recombination, however, is likel