Preface

A little over half a century ago, Richard Johnson and Cedric Mims commented that, "it must be explained, for example, why a virus causing no more than a cold sore in one person can produce fatal encephalitis in another". Although their thoughts were directed at herpes simplex virus type 1, this inquiry can be extended to all neuroinvasive alphaherpesviruses. How do these viruses gain access to the nervous system? How do they establish and reactivate from the latent state? And why do these typically benign infections sometimes have severe or lethal outcomes?

Herpesviruses are among the most complex viruses that infect humans and, with genomes in excess of 100 kilobases, their biology remains dauntingly complex. Nonetheless, since Johnson and Mims proffered their insightful question in 1968, a wealth of knowledge on alphaherpesviruses has been gained. In particular, since the last edition of this book in 2011, our understanding of the alphaherpesvirus infectious cycle has been accelerating. With this expanded knowledge comes the recognition that a comprehensive overview of all research, including the clinical and veterinary pathogens belonging to the six recognized alphaherpesvirus genera, is beyond the scope of this text. Instead, in this edition of Alphaherpesviruses: Molecular Biology, Host Interactions and Control, researchers with expertise in different aspects of the host-pathogen interface offer their perspectives on the current state of the field using herpes simplex virus as the focus.

The book begins with the topic of genomics (Chapter 1), which was in its infancy ten years ago and has since yielded new insights into virus diversity and evolution. It then moves on to the early stages of the infectious cycle, a period prior to *de novo* viral gene expression, with chapters dedicated to viral entry into the cell (Chapter 2), genome delivery from the cell periphery to the nucleus (Chapter 3), and the fate of the genome in the nucleus (Chapter 4). With this knowledge in hand, the book then turns to how infection progresses in neurons (Chapter 5) and how latency is established there (Chapter 6). Because latency is tied to host response, the book next covers cellular responses to infection and corresponding viral immune evasion strategies (Chapter 7). Discussion of the infectious cycle concludes with the late stages of infection, including capsid egress from the nucleus (Chapter 8) and subsequent virion assembly in the cytoplasm (Chapter 9). The book ends with the two translational topics, vaccines (Chapter 10) and oncolytic vectors (Chapter 11).

We thank Caister Academic Press for supporting the field of Alphaherpesviruses and especially thank all the contributing authors that dedicated their time and effort to compile this comprehensive overview of the alphaherpesviruses.

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