Preface

Perhaps first described in the Bible in Numbers 11:31–34 and later by the poet W.H. Auden:

Little birds with scarlet legs,
Sitting on their speckled eggs,
Eye each flu-infected city

Influenza has over the course of human experience been defined by the suffering, illness, and death rates it caused and causes of epidemics and pandemics worldwide.

Influenza virus belongs to the orthomyxovirus family and comes in three flavors, types A, B, and C, with type A and B viruses being important causes of disease in humans, with the majority of cases due to type A. These viruses have a negative strand sense, segmented RNA genome with eight genes that can code for up to 11 proteins. Because of the segmented genome and permissive infection of several animal species (primarily humans, birds, and pigs) influenza virus can easily reassort differing RNA segments between human and animal viruses. Such an event leads to antigenic shift, and possible new pandemic strains. Further, as with other RNA viruses, influenza's RNA-dependent RNA polymerase is error-prone such that mutations occur frequently, and in the absence of a proofreading frame that eliminates and thus fails to control fit mutated viruses from evolving. The fitness of such mutated virus is suggested by their fidelity during replication. They undergo further selection because of antibody responses and immune escape, termed antigenic drift.

This work consisting of two volumes explores influenza pathogenesis and control from basic structure, binding, entry, replication, and release of influenza virus to its spread, the results of its interaction with animal models, the innate and adaptive immune systems, and current epidemiologic efforts at rapid recognition and antiviral, anti-host cytokine storm therapies as well as vaccine strategies to control and prevent influenza virus infections.

Volume I provides overviews of current information on molecular determinants of viral pathogenicity, virus entry and cell tropism, pandemic risk assessment, transmission and pathogenesis in animal species, viral evolution, ecology, and

antigenic variation. Leading investigators who provide current information on these topics include Hans-Dieter Klenk and colleagues on the influenza hemagglutinin, Guelsah Gabriel and Ervin Fodor on the polymerase complex, David Steinhauer and John McCauley on receptor binding, and Charles Russell on membrane fusion activity of the hemagglutinin protein. Nancy Cox and colleagues describe the influenza risk assessment tool (IRAT) to evaluate pandemic potential of novel influenza viruses. Yoshi Kawaoka, Ron Fouchier, Anice Lowen, John Steel, Hualan Chen, Juergen Richt, and their colleagues provide overviews of studies on viral transmission in animal hosts. Monique Franca, Jacqueline Katz, Ian York, Terrence Tumpey, Amy Vincent, and colleagues review studies on pathogenesis in avian and mammalian hosts. Amber Smith and Jon McCullers describe the significance of secondary bacterial infection in viral pathogenesis. Viral ecology, evolution, and antigenic variation are discussed in chapters by Sun-Woo Yoon, Robert Webster, Richard Webby, Anice Lowen, John Steel, and Ruben Donis.

The second volume in this series, Volume II is concerned with innate immunity and adaptive immunity, vaccines, and antivirals. Experts in these various areas including John Teijaro, Adolfo Garcia-Sastre, Bali Pulendran, Stacey Shultz-Cherry, Paul Thomas, and colleagues have contributed chapters on specific aspects of innate immunity, while Tom Braciale, Rafi Ahmed, Donna Farber, and colleagues describe B and T cell adaptive immunity. Chapters on vaccines and vaccination include those contributed by Rino Rappuoli, Ian Wilson, Peter Palese, John Steel, Kanta Subbarao, Daniel Perez, Philip Dormitzer, Hongquan Wan, Maryna Eichelberger, Hiroshi Kida, Hideki Hasegawa, Richard Compans, Ioanna Skountzou, and their colleagues. Lastly, Ralph Tripp and S. Mark Tompkins discuss new antiviral discoveries, while John Teijaro writes about the use of sphingosine-1-phosphate receptor 1 agonist to control the resultant cytokine storm caused by influenza virus infection.

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