Infectious disease is merely a disagreeable instance of a widely prevalent tendency of all living creatures to save themselves the bother of building, by their own efforts, the things they require. Whenever they find it possible to take advantage of the constructive labors of others, this is the path of least resistance. The plant does the work with its roots and its green leaves. The cow eats the plant. Man eats both of them; and bacteria (or investment bankers) eat the man. (Hans Zinsser, 1935 from Rats, Lice and History)

This edition of Microbiology Australia is addressing the issue of microbes and chronic disease. Many of the microbes dealt with in this edition are viruses, partly due to interest, but also due to the nature of viral infection, which is of a host cell parasite, often of limited genomic means. Viruses only replicate by infecting cells, and the essence of viral infection of the cell is manipulation of the cellular processes, presumably in order to increase viral replication and allow viral persistence for the duration of infection. Even viruses with shorter genomes (such as RNA viruses typically with genomes in the order of seven to 10 kilobases in length) have considerable parts of their genome dedicated to non-structural, and in some cases structural, proteins that alter cellular processes. Through encoding different messenger RNAs, and different proteins, virus families have been able to occupy distinct ecological niches, allowing them to infect the human host.

The nature of chronic disease induced by viruses, bacteria, and other organisms often appears as a side-effect, or by product of the infection. It may be that the association of human rhinoviruses with asthma exacerbations is purely fortuitous. Conversely, it appears that in other cases the chronic disease may enhance acute infection and persistence through manipulating specific cells to allow a suitable environment for the viruses, bacteria or other microbes persistence. A good example of this is the herpes virus EBV (discussed by Professor Rajiv Khanna) where infection of the B-cell lineage can result in tumorigenesis, and where infection of such cells is a necessary part of the viral life cycle. In the case of EBV, B cell infection is critical for persistence, where the virus persists in a latent, non-replicating phase. It is harder to see the importance of bacteria in inflammatory bowel disease from the microbes point of view, discussed by Professor Stephen Riordan. However, it is important to keep an open mind, as there may be as yet unclear ecological advantages for the organism.

Microbes are evolving continuously, and in many ways at a globally faster rate than high eukaryotes such as humans. This edition provides an insight into how such co-evolution of microbes with humans has resulted in what are in many cases unexpected untoward effects. Although some of these may seem unrelated, it is likely that better understanding of the organism/host relationship that results in these chronic diseases will further our understanding of both the human disease and the microbe. Through such enhanced understanding, we hope to improve our means of controlling, and perhaps curing, some of these diseases.

Biography
Professor William Rawlinson is a Medical Virologist and is head of the Division of Virology, in the Department of Microbiology SEALS, with a conjoint position in the Department of Infectious Diseases, Prince of Wales Hospital. He holds a conjoint academic position as Professor in the School of Medical Science and the School of Biotechnology and Biomolecular Sciences at The University of New South Wales, currently supervising PhD, Masters and science Honours students. His major research interest is in human cytomegalovirus (CMV) infection of mothers and babies, particularly mechanisms of transplacental virus transmission. The research group that he heads studies congenital infections, enteroviruses, hepatitis viruses, new antivirals and antiviral resistance of herpesviruses.