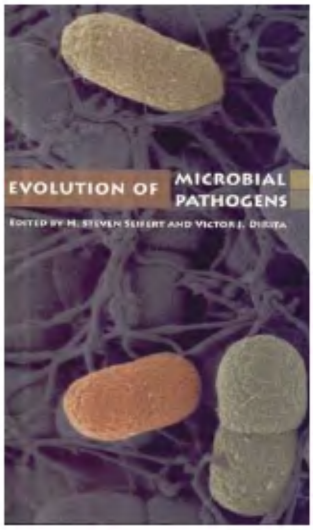

Evolution of Microbial Pathogens. H. Steven Seifert and Victor J. Dirita (eds). ASM Press, 1752 N. Street, NW, Washington DC, USA. 2006. xiii + 355 pp. Price: US$ 119.95.

The emergence of the new pathogenic microbes, our understanding of the molecular mechanisms that underlie genetic change in microbes in the last two decades and their nexus with myriads of viruses present in the natural habitats seem to have a sobering effect on infectious diseases specialists. The mood seems to be that it would be prudent to explore thoroughly the basic biology of pathogens in our endeavours to devise new antimicrobials and vaccines for their control. The book under review is an attempt to understand one important aspect (evolution) of the complex biology of pathogens.

The book has been divided into three sections. Section I (General concepts of microbial evolution) is devoted to discussing how evolution is studied in microbes and has six chapters. In an overview (chapter 1) of this section, J. J. Mekalanos, a well-known expert in the area of microbial molecular genetics, opines that although evolution has shaped the traits of pathogenic microbes, less research efforts have been devoted to this than to other aspects of microbial pathogens. Chapter 2 (Studying evolution using genome sequence data by J. G. Lawrence) brings out how large-scale sequencing of microbial genomes has generated much interest in understanding the evolution of pathogens, and how in this backdrop certain questions can now be addressed for the first time. It is startling to know that slipped-strand mispairing during DNA replication constitutes the major force for generating strain diversity. The evolution of certain pathogens, which are highly fastidious for growth *in vitro* such as *Borrelia burgdorferi* and the obligate endosymbiont *Buchnera aphidicola* can now be gleaned from complete genome sequences. Chapter 3 (Population dynamics of bacterial pathogens by Martin Maiden and R. Unvin) shows how bacterial populations are both dynamic and diverse, exhibiting a range of population structures, which may be studied by multilocus enzyme electrophoresis (MLEE) and multilocus sequence typing (MLST). These studies are germane to understanding evolution and epidemiology, and their application to public health. Chapter 4 (The study of microbial adaptations by long-term experimental evolution by V. S. Cooper) is an account of principles and observations on the evolution of microbial pathogens by the use of long-term laboratory cultures of *E. coli* that were established in 1988 and must have evolved ≈20,000 generations by now. Obviously such evolution is not affected by events like horizontal gene transfer (HGT) and niche adaptation. This approach is unconventional, beset with its own limitations but nevertheless holds promise for understanding evolution of many pathogens. Chapter 5 (The contribution of pathogenicity islands to evolution of bacterial pathogens by Jorg Hacker and colleagues) shows how acquisition of pathogenicity islands (PAIs) confers new traits on pathogenic microbes. The transfer of PAIs into new host genomes is followed by modifications to indiginize and form stable association, a process called 'homing'. The authors tabulate a large number of PAI and PAI-like elements identified in pathogenic enterobacteria. Chapter 6 (Black holes and antivirulence genes: selection of gene loss as part of evolution of bacterial pathogens by W. A. Day and Anthony Maurelli) brings out how gene loss can play role in the evolution of pathogens. The expression of certain genes called antivirulence genes is detrimental to pathogenic lifestyle of microbes. Modifications, mutation or loss of antivirulence genes allow emergence of new pathogens. This has been illustrated by the loss of lysine decarboxylase encoded by *cad*A gene in *Shigella*. This paradigm has been extended to other genes and pathogens namely *Bacillus anthracis* and *Burkholderia mallei*.

Section II (Environment and the evolution of microbial pathogens) also has six chapters and brings out the common themes of microbial pathogenicity in evolutionary terms.

An overview (chapter 7) of this section by R. Kotler and D. Hogan brings out that the take home message from this section is that many determinants that help a microbe to convert into a pathogen probably evolve in environmental settings, microbial communities and in mutualistic associations. Exploration of this viewpoint, which seems slightly unconventional at this moment, may open new vistas to gain insights into the evolution of pathogens and their control. Chapter 8 (Evolution of pathogens in soil by R. Muir and Man-Wah Tan) interestingly avers that soil is an important niche where pathogenic microbes evolve. The factors that drive evolution in soil are low nutrient availability and predation. How traits such as acquisition of iron, osmoadaptation and ability to survive within amoebae developed during stay in the environment help non-pathogens to develop into pathogens has been highlighted. Although there is no doubt that there is parallelism between the stresses (abiotic and biotic) encountered by bacteria in the environment and while colonizing the animal/plant host; the veracity of this parallelism needs an in-depth analysis. Chapter 9 (Experimental models of symbiotic host–microbial relationships: understanding the underpinnings of beneficence and the origin of pathogenesis by M. J. McFall Ngai and J. I. Gordon) is in the same vein as that in the previous chapter. These authors suggest dissecting beneficial host–microbe interactions such as legume–rhizobia and interactions of complex microbial consortia in different regions of the alimentary canal (oral cavity, stomach and intestine) as these are likely to reveal features critical and/or common to evolutionary patterns of pathogenic and non-pathogenic microbes. Chapter 10 (Evolution of bacterial toxins by O. Colin Stine and J. Nataro) shows how toxin genes represent an exquisite model for studying evolution of pathogens as these genes are acquired through HGT using multiple mechanisms. Detailed discussion on shiga-like toxins, heat-stable toxins, RTX-pore forming toxins, tetanus and botulinum toxins, and autotransporter toxins illustrate the evo-