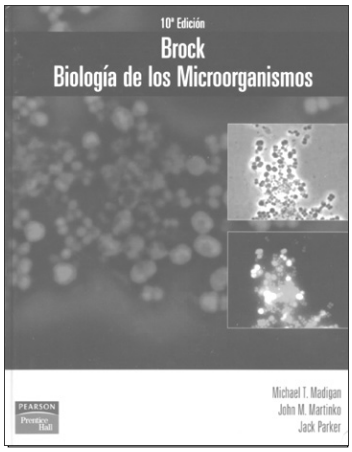
INTERNATIONAL MICROBIOLOGY (2004) 7:77–80

INTERNATIONAL MICROBIOLOGY




# Brock. Biología de los microorganismos (10ª ed.)*

MICHAEL T. MADIGAN, JOHN M. MARTINKO, JACK PARKER (eds)

2003. Pearson-Prentice Hall, Madrid, Spain
1012 pp, plus Glossary and Indexes. 27.8 × 22 cm
Price: 77.90 €
ISBN 84-205-3679-2

*Translation of the book "Brock Biology of Microorganisms" (10th ed.). Prentice Hall-Pearson Education Inc., Upper Saddle River, NJ 07458, USA. 2003. ISBN 0-13-066271-2

"One of the most satisfying activities of my career has been the success of my book, *Biology of Microorganisms* (BOM). I signed the contract for this book with Prentice Hall on January 11, 1967. Although I had already published two books, they were both modest in scope, whereas the new book was to be a major effort". So wrote Thomas D. Brock in his memoir for *Annual Review of Microbiology* [1]. The book, which was first published in 1970, has already reached its 10th edition. At the beginning, BOM emphasized general concepts and tried not to compete with *The Microbial World* by Stanier, Doudoroff, and Adelberg, which had also been released by Prentice Hall. When the publisher decided to no longer publish *The Microbial World*, Brock was working on the second edition of the BOM. He thus decided to widen his book's scope to include those topics previously covered by Stanier's et al. book. Michael T. Madigan, who was a Ph.D. student of Brock in 1973–1976, joined him in the preparation of the BOM's 4th edition and has coauthored the book ever since. John M. Martinko and Jack Parker joined Madigan and Brock in writing the 7th edition. Since the 8th edition, Madigan, Martinko, and Parker have taken over as authors of the book. As a tribute to its early author, the book has incorporated his name in the title, which now is *Brock Biology of Microorganisms* (BBOM).

Sergei Winogradsky (1856–1953) and Martinus Beijerink (1851–1931)—each working independently—applied the principle of enrichment culture to isolate specific microorganisms, explore the microbial world, and thereby prove both the practically ubiquitous occurrence of microorganisms on earth and the amazing diversity of their nutritional requirements [2]. Even though Winogradsky's and Beijerink's approach established the foundations of microbial ecology, Brock is considered to be the modern father of this subfield of microbiology. This may have led some of BBOM's potential readers to believe that the book is devoted solely to microbial ecology. As a matter of fact, microbial ecology is only a part of the book even if its perspective pervades the whole work. The book comprises six main units: "Principles of microbiology", "Evolutionary microbiology and microbial diversity", "Metabolic diversity and microbial ecology", "Immunology, pathogenicity and host responses", "Microbial diseases," and "Microorganisms as tools for industry and research".

"Principles of microbiology" (Chapters 1–10) could serve as the core of an introductory course on general microbiology, since the unit contains basic information. As a novelty, this edition has introduced a chapter (Chapter 2, "An overview of microbial life") that provides a general view of microbial diversity, including the main microbial groups and their evolutionary relationships. Chapters 9 ("Essentials of virology") and 10 ("Bacterial genetics") have been both restructured and reduced. Chapter 9 focuses on essential concepts of virology, and Chapter 10 discusses bacterial genetics as a mixture of both in vivo and in vitro science.

"Evolutionary microbiology and microbial diversity" (Chapters 11–16) deals with several topics crucial to understanding the origin and evolution of life and the diversity of microbial life, which is the basis for all biodiversity. It also provides an introduction to the new field of genomics (Chapter 15, "Microbial genomics"), which entails more than finding out the number of genes of a given organism and their functions. Comparative genomics allows evolutionary relationships among different organisms to be reconstructed and early forms of life to be distinguished from those more evolved. Chapter 16 ("Bacterial, plant an animal viruses") focuses on viral diversity and viral strategies for replication, which is the basis for the classification of viruses.

"Metabolic diversity and microbial ecology" (Chapters 17–19) are discussed in the same unit, which is an excellent compendium of current microbial ecology. "Metabolic diversity" (Chapter 17) lays the foundations for understanding nutrient cycles and other essential microbial activities dealt with in the two following chapters. Phototrophy,

chemolithotrophy, anaerobic metabolism, aerobic degradation of hydrocarbons, polysaccharides, fats and other substances, as well as nitrogen fixation are widely discussed and updated. Chapter 18 ("Methods in microbial ecology") discusses the different approaches used in microbial ecology studies. These include enrichment and isolation, cell-staining methods, gene isolation and characterization, and measuring the activities of microorganisms in situ. Chapter 19 ("Microbial habitats, nutrient cycles, and interactions with plants and animals") explores what microorganisms do in their habitats and how they interact with other organisms and with the ecosystem itself.

Starting with Chapter 20, the book focuses on the relationships and interactions between microorganisms and humans. The unit "Immunology, pathogenicity and host responses" (Chapters 20–24) includes new chapters on immunology, discussing both the basic principles (Chapter 22, "Essentials of immunology") and the molecular details, including proteins and their genetics, as well as the intercellular interactions (Chapter 23, Molecular immunology), that trigger and control the immune response. This unit also provides insights into practical aspects of clinical microbiology, such as control of microbial growth (Chapter 20) and diagnostic methods based on immunology and molecular biology (Chapter 24, "Clinical microbiology and immunology").

The 10th BBOM edition devotes more attention to medical microbiology than previous editions did. In addition to the topics already covered in the previous units, the unit "Microbial diseases" (Chapters 25–29) discusses epidemiology and the different kinds of microbial infections, and devotes an entire chapter to the prevention of waterborne microbial diseases and another to food-borne microbial diseases. The last unit, "Microorganisms as tools for industry and research" (Chapters 30 and 31), discusses another aspect of relationships between humans and microorganisms. It deals with the use of microbes to obtain valuable commercial products or carry out major chemical transformations. Industrial microbiology and the techniques and applications of genetic engineering are widely discussed.

Translating works of this kind, with more than 1000 pages and specialized terminology, is a very hard task that requires a great deal of time and effort. This explains why translations are often published when a new edition of the original book is already in preparation or has even been published. In the case of the BBOM 10th edition, however, the Spanish version has been released quickly, and students from Spain and Latin American countries can use it knowing that the same edition in English is still the latest one available. The collaboration of several translators, all of them microbiology experts in Spanish universities, has made it possible to accelerate the translation process. Expertise in science, however, does not mean expertise in language, and there are irregularities in the quality of the different chapters of the Spanish BBOM translation. Some chapters contain errors that are frequently found in scientific textbooks translated into Spanish. In addition, this BBOM translation reflects that politically correct language is not yet common in Spain. In fact, in some chapters the term *hombre* (man) is used where the original uses "humans".

The Spanish BBOM is a major microbiology textbook for students in fields such as biology, medicine, biochemistry, food sciences, veterinary medicine, environmental sciences, and biotechnology. It succeeds in making microbiology understandable to anyone with a basic knowledge of biology and biochemistry, as it is written extremely clearly. In addition, pedagogic aids, such as high-quality figures, review tools, and glossaries, not to mention online media tutorials and the popular virtual exam, help students to understand fundamental concepts and to check their knowledge of the subject. Moreover, the BBOM is not just a textbook that makes learning microbiology fun; it is also an updated reference book on microbiology for professionals interested in catching up on biochemistry, microbiology, and genetics.

In his above-mentioned memoir, Brock wrote: "In reviewing my life and career, I can see that my broad interests have carried me into quite a few fields. This has been both a strength and a weakness […]. I have occasionally thought how nice it would have been to have specialized in one narrow area." [1]. Current microbiologists should be greatly indebted to Brock for the choice he made. Apart from his findings as a researcher, Brock's exploration of both the many facets of microbiology and the history of this science has made it possible that not only his contemporaries but also later generations of microbiologists have been able to enjoy books such as *Principles of microbial ecology*, *Milestones of microbiology*, and *Biology of Microorganisms*, of which the BBOM is the natural outcome. Thank you, Prof. Brock, for this wonderful legacy!

## References

1. Brock TD (1995) The road to Yellowstone—and beyond. Annu Rev Microbiol 49:1–28
2. Kluyver AJ, van Niel GB (1956) The microbe's contribution to biology. Harvard University Press, Cambridge, MA, pp 1-30

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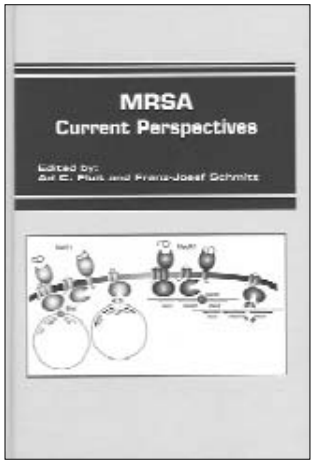
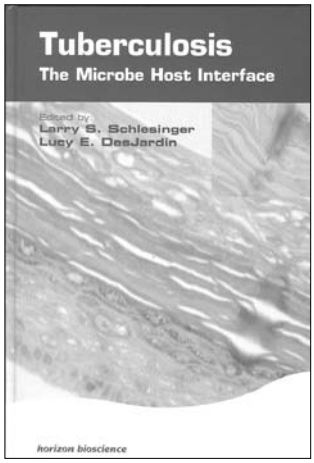



## MRSA Current Perspectives

AD C. FLUIT, FRANZ-JOSEF SCHMITZ (eds.)

2003. Caiser Academic Press, Norfolk, UK
365 pp, 16 × 24 cm
Price: £ 90.00
ISBN: 0-9542464-5-4




## Tuberculosis. The Microbe Host Interface

LARRY S. SCHLESINGER, LUCY E. DESJARDIN (eds.)

2003. Horizon Scientific Press, Norfolk, UK
280 pp, 16 × 24 cm
Price: £ 90.00
ISBN: 0-9545232-1-0

It is the great diversity among microbes, manifested by their structural diversity, modes of reproduction, ecological relationships with other species, and evolutionary history, that make the subject of human exposure to them so challenging. Groups of bacteria, viruses and protists could cause most infectious diseases. The incredible diversity among these microbes results in a great variety of modes by which they meet humans. Two recent books, MRSA *Current Perspectives* [MRSA means methicillin-resistant *Staphylococcus aureus*] and *Tuberculosis. The Microbe Host Interface*, discuss the behaviors of two pathogens whose different strategies have enormous implications for human health.

Imagine that you could become a microbe for one day. Which microbe would you prefer, *Staphylococcus* or *Mycobacterium*? Before answering this crucial question, I highly recommend that you read these two books. They provide a state-of-the-art study of two human pathogens by integrating genetic, microbiological, and immunological approaches to elucidate the mechanisms of pathogenesis. At the same time, they offer interesting reading for microbiologists, molecular biologists, and clinicians.

Humans live in a dynamic state of coexistence with a myriad of microbial life forms. The human body is the habitat of a community of microbes that mutually interact; indeed, our body may be considered as an ecosystem. Some microorganisms are usually mutually beneficial (they keep us healthy), whereas others can be adverse effects. We humans are still here because the pathogens that have attacked us have an "interest in our survival". The death of the host is not the microbe's primary objective, rather it is an accident of "collateral damage". In this context, humans survive because the elimination of its hosts is not advantageous for the pathogen. Instead, the aim of pathogens, the driver of their natural selection, is to domesticate their host. The most successful pathogens are those persisting in a healthy or near-healthy host, and promoting and exploiting their own behavior for two principal goals: (1) to ensure continued maintenance and viability, and (2) to promote efficient dissemination to other hosts. This explains why morbidity is a more prevalent outcome of disease than mortality.

*Staphylococcus aureus* has remained a significant cause of nosocomial (hospital-related) morbidity and mortality. For almost four decades, the increasing prevalence of MRSA has presented unique challenges to clinical microbiologists. In addition, strains of community-acquired *S. aureus*, causing a wide range of acute infections, have also become common. The disease spectrum includes bacteremia, endocarditis, central nervous system infections, osteomyelitis, abscesses, pneumonia, urinary tract infections, and other syndromes caused by exotoxins, including food poisoning, scalded skin syndrome, and toxic shock syndrome. An interesting property of staphylococci is their ability to colonize different habitats. They can, for example, colonize human hosts as "commensal microbiota" with a frequency of about 25–30%, and nearly 90% of the human population is transiently colonized with *S. aureus* at some time. Very little is known about how *S. aureus* exists in the carrier state and how it avoids both the innate immune defenses of the host and mucosal immunity.

During the nineteenth and twentieth centuries, a single disease, tuberculosis (the "white plague"), was responsible for the death of approximately a thousand million persons. *Mycobacterium tuberculosis* remains one of the most successful human pathogens, and the cause of one of the greatest medical struggles in human history. But tuberculosis is much more than a medical affliction. It has inspired great works of literature (*The Magic Mountain*, Thomas Mann), painting (Simonetta Vespucci, painted after her death by Piero di Cosimo—she was first the model for Botticelli's Venus), and opera (*La Traviata*, Giuseppe Verdi). It has also robbed humanity of many famous people (Tutankhamon, Frederick Chopin, Franz Kafka, Miguel Hernández, George Orwell, etc.). Humans are the only natural hosts for *M. tuber-*

*culosis* infection. The global infectious reservoir is immense, with over one-third of the world population infected. Maintenance of the infectious cycle among humans depends on the transmission of *M. tuberculosis* from patients with active pulmonary tuberculosis to immunologically naïve individuals. The alveolar macrophages are the primary cell type targeted by *M. tuberculosis*.

Inside a given ecosystem there is a "constant state of interaction" between all of its living species. Each species in an ecosystem directly or indirectly drives the evolution of every other species. As populations of different species coevolve in the same evolving ecosystem, the nature of the relationship between any two cohabiting species—for example, between a pathogen and its host—will continuously change, sometimes to the advantage of one species over the other. Prolonged interaction between human hosts and infectious organisms, carried on for many generations and among suitably numerous populations on each side, has created a pattern of mutual adaptation allowing both to survive. A disease organism that kills its host quickly creates a crisis for itself, since a new host must somehow be found fast enough to keep the pathogen's own chain of generations going. This apparent initial advantage will typically lead to a pyrrhic victory. Conversely, a human body that resists infection so completely that the would-be parasite cannot find any lodgment obviously creates another kind of crisis of survival for the infectious organism. Unlike bacterial pathogens that proliferate rapidly within a host (e.g. *S. aureus*), causing acute infections of relatively short duration and producing virulence determinants, such as toxins or other compounds that interfere with normal host function, the hallmark of *M. tuberculosis* is the ability to survive and grow slowly inside its obligate human host. *M. tuberculosis* can grow as an intracellular parasite in non-activated macrophages, persist for decades in a quiescent state in the granuloma, and survive in an aerosol as it is passed from one host to another by coughing, singing, or talking.

MRSA was discovered shortly after the methicillin entered into clinical use in 1961. MRSA mediates clinically inadequate responsiveness to all currently available β-lactam antibiotics. In addition, MRSA is typically resistant to several other antimicrobial agents, including aminoglycosides, chloramphenicol, clindamycin, fluoroquinolones, and macrolides. The ability of *S. aureus* to colonize and multiply in the host and its capacity to acquire and exchange genetic information are elements that contribute to its success as a pathogen. Virulence factors can be generally separated into two main classes: (1) surface-associated factors facilitating adherence, and (2) secreted factors causing tissue destruction and allowing immune system evasion. It has frequently been suggested that MRSA are more pathogenic than MSSA (methicillin-susceptible *S. aureus*). However, despite a large volume of research, it is not clear whether MRSA are more pathogenic than their methicillin-susceptible counterpart. One finding favors the hypothesis that some MRSA are more virulent: all cases of necrotizing pneumonia in children are caused by a leukotoxin encoded by methicillin-resistant strains of *S. aureus*.

*M. tuberculosis* has acquired a number of mechanisms to interfere with the activation of both innate and adaptive host immune systems. The bacterium inhibits phagosomal acidification of macrophages, resists killing by oxygen radicals through production of superoxide dismutase, and induces macrophages to produce inhibitory cytokines such as interleukin (IL)-10 and transforming growth factor (TGF)-β. These molecules, in actively replicating tuberculosis (such as during primary infection and upon reactivation), inhibit the effects of pro-inflammatory cytokines such as interferon (IFN)-γ and tumor necrosis factor (TNF)-α. In addition, active *M. tuberculosis* infection is associated with increased apoptosis of mycobacterial-antigen-specific T cells. Molecular epidemiologic studies have revealed several critical features of the pathogenesis of tuberculosis. Some strains are much more widely distributed than others, suggesting that they have phenotypic characteristics that favor dissemination. Drug-resistant strains may be less transmissible than drug-susceptible ones, suggesting that expression of some of the genes encoding drug resistance also reduces fitness.

As Louis Pasteur wrote: "I have taken my drop of water from the immensity of creation, and I have taken it full of the elements appropriate to the development of inferior being. And I wait, I watch, I question it, begging it to recommence for me the beautiful spectacle of the first creation. But it is dumb, dumb since these experiments were begun several years ago; it is dumb because I have kept it from the only thing man cannot produce, from the germs which float in the air, from Life, for Life is a germ and a germ is Life".

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