

Prions: The New Biology of Proteins

Claudio Soto

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Prions are believed to be the causative agents of a group of rapidly progressive neurodegenerative diseases called transmissible spongiform encephalopathies, or prion diseases. They are infectious isoforms of a host-encoded cellular protein known as the prion protein. Prion diseases affect humans and animals and are uniformly fatal. The most common prion disease in humans is Creutzfeldt-Jakob disease (CJD), which occurs as a sporadic disease in most patients and as a familial or iatrogenic disease in some patients. Whether prions are infectious proteins that act alone to cause prion diseases remains a matter of scientific debate. However, mounting experimental evidence and lack of a plausible alternative explanation for the occurrence of prion diseases as both infectious and inherited has led to the widespread acceptance of the prion hypothesis.

Interest in prion disease research dramatically increased after the identification in the 1980s of a large international outbreak of bovine spongiform encephalopathy (BSE, also known as mad cow disease) in cattle and after accumulating scientific evidence indicated the zoonotic transmission of BSE to humans causing variant CJD. In recent years, secondary bloodborne transmission of variant CJD has been reported in the United Kingdom.

Prions: The New Biology of Proteins describes the current state of knowledge about the enigmatic world of prion diseases. The book is organized into 12 mostly brief chapters, which nicely summarize the various types of prion diseases and the challenges associated with their diagnosis

and treatment. These sections review the biology of prions, the underlying hypotheses for prion replication, and the biochemical basis for strain diversity. Chapters 2 through 5 describe the various characteristic features of prions, including the historical evolution of the prion hypothesis, a detailed description of the possible mechanisms by which the normal prion protein is converted into the pathogenic form, and the cellular biology and putative functions of the normal prion protein. The author's lucid descriptions of the various topics are supported by diagrams and key references. Subsequent chapters describe prion disease laboratory diagnostic tools that are available or under development. Chapter 9 succinctly summarizes the most likely target sites, from the formation of the infectious agent to its effects on neurodegeneration, which can be exploited for likely therapeutic development. The same chapter describes the various anti-prion compounds that have been or are being tested as therapeutic interventions for prion diseases.

The book is unusual because its entire content was exclusively authored by 1 person, resulting in a paucity of in-depth information in some areas, which may have been provided by multiple authors. However, all things considered, the book can be a valuable resource for scientists beginning to understand the world of prion diseases, the underlying biochemical mechanism of disease occurrence, and the challenges associated with the diagnosis and treatment of prion diseases.

Ermias D. Belay*

*Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Address for correspondence: Ermias D. Belay, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, Mailstop A-39, Atlanta, GA 30333, USA; email: ebelay@cdc.gov

Battle of the Genomes: The Struggle for Survival in a Microbial World

H.M. Lachman

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Although this book's title promises the excitement of a 21st-century computer game, the cover photograph of Robert Koch in 1883 provides a better clue to the contents. The general plan is a survey of 20th-century genetics, illustrated by insights into human coevolution with microbial pathogens. Early chapters focus on familiar examples, including G6PD deficiency and sickle cell trait as adaptations to malaria, as evidence for pathogen-driven natural selection. Later chapters discuss more recent research findings, varying from female preference for the scent of males with dissimilar human leukocyte antigen types to the role of human CFTR membrane protein in infection with *Salmonella* Typhi. All of these are such good stories that science writer Matt Ridley included briefer versions in Chapter 9 of his popular book *Genome: The Autobiography of a Species in 23 Chapters (1)*.

Battle of the Genomes: The Struggle for Survival in a Microbial World discusses in some detail how catastrophic epidemics of cholera, bubonic plague, and smallpox could explain the emergence of certain common human genetic mutations. Some of these mutations are deleterious; for example, CFTR $\Delta F508$, which reduces the risk for typhoid, causes cystic fibrosis in persons who inherit 2 copies. Other mutations are beneficial, such as CCR5 $\Delta 32$, which may have protected carriers from smallpox and now reduces the risk for HIV infection. In general, the author's review