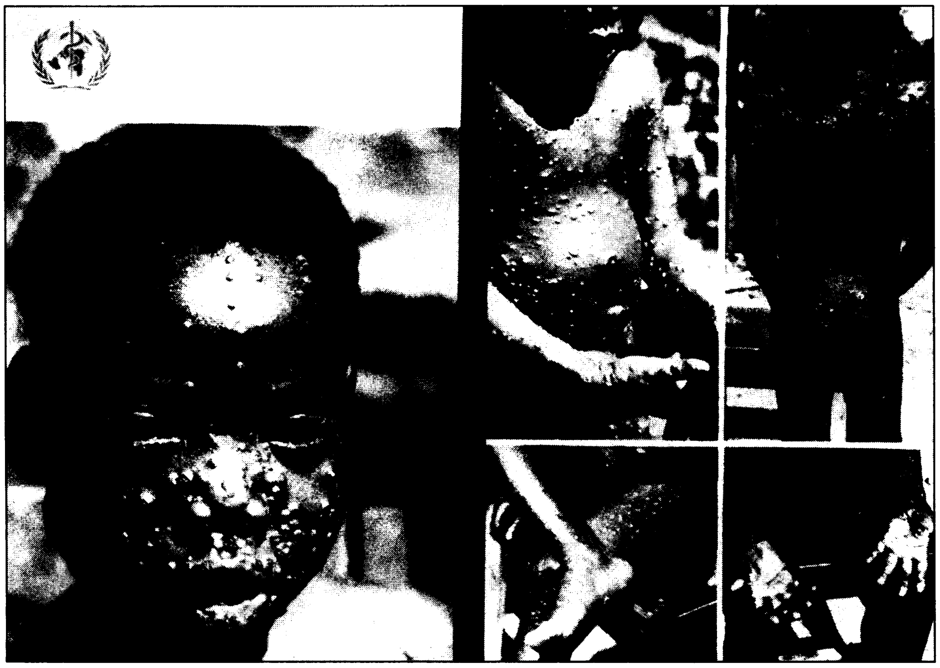
disease. As Morse points out in chapter 2, viruses may emerge by any of three basic mechanisms: introduction to a species from another species, evolution of a new variant, or dissemination from a smaller to a larger population. Although many people assume that new diseases are the result of viral evolution (as with influenza), in recent times they have more often been due to existing viruses that have gained access to new hosts. The four-corners virus, for example, which appears to be endemic in deer mice, has probably caused sporadic disease among humans in the past. The recent outbreak is very likely a result of some change in ecological conditions that has led to more frequent interaction between the rodent hosts and humans. Argentine hemorrhagic fever, Bolivian hemorrhagic fever, and hemorrhagic fever with renal syndrome are described in the book as examples of diseases that emerged owing to changes in agricultural practices that facilitated transmission of arenaviruses from the rodent hosts to agricultural workers. Rift Valley fever in Africa and Oropouche in South America are cited as examples of disease outbreaks that occurred because of the development of ecological conditions that favored an increase in the number of insect vectors.

Human immunodeficiency virus is another example of a virus that probably is not a new variant but has gained access to new populations through medical technology (blood transfusion), sexual transmission, and contaminated hypodermic needles. Describing the AIDS pandemic in chapter 20, Temin remarks, "With the changes in urbanization, enormous population increases in Africa, freer lifestyles in North America, and jet travel, a major new pandemic occurred. If anything the surprise might be that there has been only one new pandemic" since World War II.

In "Are we prepared for a viral epidemic emergency?," based on discussions at a meeting of the American Society of Tropical Medicine and Hygiene in Honolulu in December 1989, Legters *et al.* describe a hypothetical "super-Ebola" pandemic to which the United States and the global public health community are ill-prepared to respond. A series of fictitious interviews clearly lays out the basic reasons for the pandemic and our failure to detect the virus earlier. This meeting and the conference on which this book is primarily based were major stimulants for a study on emerging diseases undertaken by the Institute of Medicine. The resulting 1992 report, "Emerging Infections: Microbial Threats to Health in the United States," recognizes our vulnerability to new or emerging bacterial, parasitic, and viral diseases and makes specific recommendations for the development and implementation of strategies that would strengthen state and federal efforts in national and international surveillance.



"Monkeypox recognition card. Adapted from the smallpox recognition card used for surveillance during the Intensified Smallpox Eradication Programme of the World Health Organization, this card, in color, illustrates the clinical features of a typical case. It helped greatly in surveillance for cases of human monkeypox in Zaire." [From F. Fenner's paper in *Emerging Viruses*; courtesy of the World Health Organization]

Joint planning is now under way by the CDC, the National Institutes of Health, the Food and Drug Administration, the Department of Defense, and other state and federal agencies.

Implementation of these plans will be a major challenge. Budgeting for events in the future is far more difficult than budgeting for situations that are facing us now. Yet *Emerging Viruses* provides strong justification for improving our national and international capabilities for detecting and responding to emerging infectious diseases before they become public health crises.

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Extracellular Organizers

Molecular and Cellular Aspects of Basement Membranes. DAVID H. ROHRBACH and RUPERT TIMPL, Eds. Academic Press, San Diego, CA, 1993. xx, 448 pp., illus. $115 or £87. Cell Biology.

Once viewed as merely a histologic curiosity, the basement membrane is now appreciated as a dynamic extracellular organizer with diverse and highly complex functions. Basement membranes provide a directional anchor for cells and a scaffolding that guides tissue morphology. They lay out a migration pathway for development and healing and constitute a selective filter essential for hemodynamic and renal function. More than just making up a continuous connective-tissue sheath separating organ parenchyma cells from the stroma, they act as an autocrine solid-phase differentiation agent, a breadboard for axonal network regeneration, and a storage depot for soluble factors. Basement membranes may even play a role in transfer of information across tissue boundaries. *Molecular and Cellular Aspects of Basement Membranes* weaves material on cell biology and physiology with information on the expanding catalogue of basement membrane–associated proteins. With the synthesis of structure and function that is reflected in this book we can begin to understand a number of pathologic conditions that can now be considered diseases of the basement membrane.

Populations of cells in tissues are organized into specialized communities through two major regulatory forces: cell-cell interactions and cell-matrix interactions. The extracellular matrix that stays in direct contact with the formative cells is made up of basement membranes. Found in virtually all members of the animal kingdom, basement membrane proteins first appear at the two-cell embryo stage. Branching epithelial morphogenesis, which establishes the architecture of many embryonic tissues, is tightly regulated by basement membranes, which can be produced by cells derived from all three embryonic layers. In the adult organism, basement membranes ensheath nerves and muscles and underlie epithelium, mesothelium, and endothelium. Separate chapters in the book summarize the unique characteristics and specialized functions of basement membranes in skin, nerve, and kidney.

In the first chapter, Martin and Piez credit Todd and Bowman with providing, in 1957, the first description of the basement membrane: " 'We have found this epithelium to rest immediately on a continuous transparent basement membrane of excessive tenuity.' " With the advent of electron microscopy, basement membranes were found to exist as uniformly thin (less than 500-nanometer), dense condensations of the extracellular matrix. Their laminated appearance in cross section could be divided into three zones: lamina lucida, the pale layer adjacent to the cell membrane; lamina densa, the dark layer below; and lamina fibroreticularis, the outer region containing anchoring fibrils that blend with the underlying stroma. The relative thickness and appearance of the three zones vary in different tissues. The recurring question in basement membrane research over the last 30 years has been, how is this highly organized extracellular structure generated? The rather surprising current answer, according to Yurchenco and O'Rear (chapter 2), is that "functional basement membrane molecular architectures are created directly through mass action-driven self-assembly." That is, the participating cells secrete a family of basement membrane proteins that automatically assemble into a polarized functional unit. Cell templates and adenosine triphosphate–dependent energetic processes are not considered essential. Once basement membrane–associated proteins were isolated and characterized, insight into the structure of basement membranes progressed rapidly. Investigators have combined purified basement membrane proteins to create a reconstituted basement membrane in vitro that directly or indirectly has many of the properties of natural basement membranes, providing support for the self-assembly thesis.

A critical mass of information has now been reached in the field. Analysis of the individual components of basement membranes has progressed to the stage of detailed domain models. With this molecular foundation, investigators are poised to develop transgenic or gene knock-out models to evaluate regulation and function in vivo. One could conclude that all of the major collagenous, glycoprotein, and proteoglycan components of the basement membrane have already been identified. However, researchers are encountering an unexpected number of variants and isoforms of proteins such as laminin and type IV collagen, many with tissue-specific distributions. Furthermore, several new proteins with putative or proven basement membrane association have been identified. The ultimate repertoire of domain interactions will undoubtedly be complex and informative.

The editors of this volume state in their preface, "It is a testament to the importance of basement membranes that subtle changes in basement membrane composition of protein structure can cause dramatic pathology." In chapter 20 Tryggvason *et al.* describe the recent discovery of Alport syndrome as the first disease known to be caused by a mutation in a gene for a structural basement membrane component. In the majority of cases this syndrome is X-linked dominant and results in progressive renal failure, often accompanied by familial hearing loss and ocular lesions. As noted by Kasinath and Kanwar in chapter 5, ultrafiltration of plasma in the kidney glomerular basement membrane (GBM) is the first step in the formation of urine. The GBM in Alport syndrome patients has been found through electron microscopic examination to be abnormal, with areas of splitting and thickening of the lamina densa. On the basis of this observation it was suggested that the affected gene might encode for one of the chains of type IV collagen, a type known since the early work of Kefalides and Spiro in 1967 to be found predominantly in basement membranes. This hypothesis proved correct in the X-linked form, where more than 50 different mutations in the alpha 5 (IV) collagen chain gene (X q22) have been identified in Alport kindreds. The alpha 5 (IV) chain seems to be expressed predominantly in the kidney GBM and in Descemet's membrane in the eye. This restricted distribution of the mutated collagen chain may explain why the pathologic manifestations are limited to specific organs. This discovery may result in a range of advances in the identification and treatment of Alport syndrome, from an improved diagnostic test to gene therapy. Since prevention of renal failure may not require correction of both kidneys, the defective gene could conceivably be replaced in one kidney by isolated perfusion or ex vivo treatment.

Several other inherited diseases may also be caused by molecular defects in the basement membrane. These include polycystic kidney disease, familial thin basement membrane nephropathy, congenital nephrotic syndrome of the Finnish type, nail-patella syndrome, certain corneal endothelial dystrophies, and hereditary blistering disorders of the skin such as epidermolysis bullosa. Furthermore, the pathologic basis of many other inherited or acquired diseases may be indirectly related to molecular alterations of the basement membrane. Basement membrane thickening in diabetes, discussed by Rohrbach and Murrah in chapter 19, may be due in part to nonenzymatic glycosylation of basement membrane components. Goodpasture's syndrome is an autoimmune disease defined by linear localization of immunoglobulin G along the basement membrane. A major antigenic determinant for Goodpasture's antibodies has been localized to the noncollagenous domain of type IV collagen. Cancer, in an important sense, is also a disease related to basement membranes. The transition from in situ to invasive carcinoma and metastasis formation is defined as focal and generalized

Vignettes: Arthropods of Britain

Casterbridge [Dorchester] was the complement of the rural life around; not its urban opposite. Bees and butterflies in the corn-fields at the top of the town, who desired to get to the meads at the bottom, took no circuitous course, but flew straight down High Street without any apparent consciousness that they were traversing strange latitudes.

—*Thomas Hardy*, in *The Mayor of Casterbridge* (1886)

Early in this century several investigators estimated the densities of spiders in assorted terrestrial ecosystems . . . . Perhaps the most imaginative estimate is 11,000 spiders per acre of woodland, derived from a count of nine spiders in a 4 square foot patch of forest. Bristowe (1971) claims that during some seasons a particular field in Sussex had over 2,000,000 spiders per acre. A few quick calculations lead him to conclude that the weight of insects consumed by the entire British spider fauna in a year exceeds the combined weights of all the humans in Britain. The full implications of this finding are yet to be determined.

—*David H. Wise*, in *Spiders in Ecological Webs* (Cambridge University Press) breaches in the basement membrane at the point of microinvasion. These defects may be the consequence of altered regulation of tissue boundaries and the inappropriate proteolytic breakdown of basement membrane barriers, which permit the migration of carcinoma cells out of the epithelial compartment, as discussed by Goodman in chapter 17. Tumor neoangiogenesis is also directly linked to endothelial modification of basement membranes regulated by angiogenic growth factors, a topic explored by Vlodavsky *et al.* in chapter 16. Damage to basement membranes prevents functional reconstitution and proper healing of cardiac muscle following infarction and of nerves following mechanical injury. For all these disease states, strategies for reconstitution of defective basement membranes or protection from proteolytic damage could serve as the basis for prevention and therapy.

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