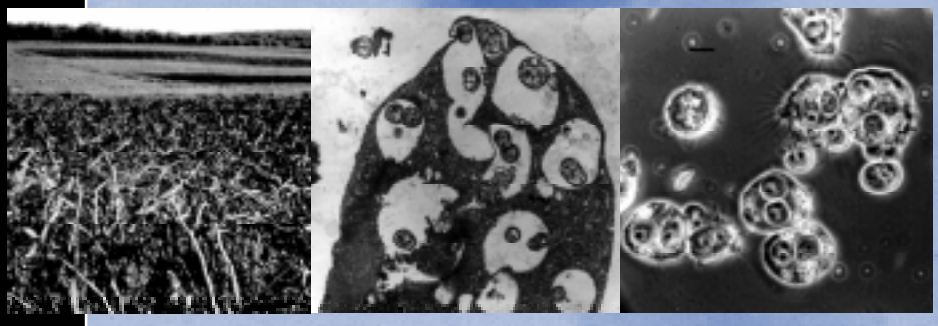
Emerging and Reemerging Infectious Diseases of Animals and Plants



AMERICAN SOCIETY FOR MICROBIOLOGY

Public and Scientific Affairs Board

On April 26, 1996, the Public and Scientific Affairs Board of the American Society for Microbiology sponsored a congressional briefing on new and emerging infectious diseases in plants, animals, and humans. The House Agriculture Committee, chaired by Representative Pat Roberts (R-KS), hosted the briefing. Published in this booklet are the scientific proceedings from the briefing.

Emerging and Reemerging Infectious Diseases of Animals and Plants

Threats to the U.S. economy, food supply and safety

AMERICAN SOCIETY FOR MICROBIOLOGY

Public and Scientific Affairs Board

CONTENTS

Emerging and Reemerging Infectious Diseases

Perspectives on plants, animals, and humans

ANNE K. VIDAVER

Anne K Vidaver is professor and head of the Department of Plant Pathology, University of Nebraska, Lincoln, and chair of the PSAB Committee on Agricultural, Food and Industrial Microbiology.

ASM sponsored a congressional briefing on 26 April 1996 on emerging and reemerging infectious diseases, emphasizing economic impacts on plants, animals, and humans. New findings show similarities among infectious agents that span different taxa and kingdoms (see table), and this trend is bringing together infectious disease specialists who earlier did not consider themselves to have common interests in disease prevention and management. They now agree that broader and better communication, integrated research, and improved public infrastructure would benefit all those dealing with infectious diseases, whether of plants, animals, or humans.

We need to overcome our tendencies to compartmentalize diseases and take a more holistic view when assessing infectious agents and hosts. Such an approach is essential for managing diseases of living organisms that are essential for our survival. As a matter of public policy, there is a compelling need for monitoring and surveillance of nonhuman emerging and reemerging infectious diseases.

Some bacterial pathogens of plants and animals—all gram negative—use similar molecular mechanisms to regulate and secrete their virulence factors (see table). Whether the same principles apply to gram-positive infectious agents, fungi, or viruses remains to be determined. For example, among the positive-strand RNA viruses, which vary greatly in morphology, enzymes involved in replicating and expressing virus genomes contain many conserved sequence motifs. Based on alignments of these motifs, most plant and animal RNA viruses can be grouped into three evolutionary lineages. The conserved nature of these proteins makes it likely that many of the molecular details of plant and animal virus replication are similar. Thus, understanding a virus-plant host interaction within a group should be directly applicable to other combinations within that group.

Further, many plants and animals display similar defenses against infectious agents. These common mechanisms represent tantalizing possibilities for disease prevention and management.

Reasons for Increase in Emerging and Reemerging Infectious Diseases

Many factors contribute to increases in infectious diseases (see box). Economic or military forces that cause population shifts create fertile ground for diseases to emerge and spread. For plants, new hybrids or even new plants not known to a particular region—virtually all that we eat or grow was once "foreign"—can engender susceptibility to pathogens previously unrecognized or recognized elsewhere. This problem is especially acute where monoculture, i.e., the growth of the same plant, such as corn (maize), is extensively practiced.

Increases in population, whether human, animal, or plant, create new microbial exposure conditions. Changes in human behavior, whether in sexual practices or food preferences, affect microbial spread and survival. Changes in agricultural practices, such as minimum tillage, are conducive to the survival of some pathogens, such as the fungi that cause wheat scab and gray leaf spot of corn. Food practices that include handling, cutting, refrigeration, or other treatment to maintain quality also serve to disseminate deleterious microorganisms and select for those that grow under these conditions. And, as population density increases in cities, the dynamics of microbial exposure and evolution increase in humans themselves, as well as in the plants and animals they consume or with which they associate. Urbanization often crowds humans and increases exposure to microorganisms. Crowding of animals, such as cattle in feedlots, poultry in confined mega-houses, and fish in ponds, can result in severe stress that increases the animals' susceptibility to infection. Similarly, crowding of plants decreases air movement, increases humidity and water retention in the soil, and often provides a conducive environment for diseases to become established and spread.

When there are changes in ecology or climate, it should not be surprising to find changes in both beneficial and detrimental organisms. Effects of global

Sequence Similarities of Proteins in Plant and Human Pathogens

PATHOGEN [a]	SEQUENCE (% SIMILARITY) [b]							
X. campestris pv. *vesicatoria* (bacterial spot of tomato)	HrpA1	HrpB6	HrpB3	HrpC1	HrpC2	HrpC3	HrpD1	HrpD2
P. solanacearum (bacterial wilt of tomato, potato)	HrpA (66)	HrpE	HrpI (70)	HrpN (74)	HrpO (81)	HpaP (54)	HrpQ	HrpT
Y. enterocolitica (diarrhea)	YscC (55)		YscJ (56)					
Y. pestis (bubonic plague)	YscC (55)				LcrD (70)		LsaA (52)	LsaB (72)
S. flexneri (bacillary dysentery)	MxiD (50)	Spa47 (65)	MxiJ (57)	Spa40 (55)	MxiA (65)			Spa24 (67)
S. typhimurium (paratyphoid fever)	InvG (52)	SpaL (70)		SpaS (56)	InvA (67)			SpaP (64)
E. coli (gastroenteritis; infections of multiple organs)	β-F1 (53)							FliP (65)

[a] *Xanthomonas campestris* pv. *vesicatoria* and *Pseudomonas solanacearum* are plant pathogens; the *Yersinia* species, *Shigella flexneri*, *Salmonella typhimurium*, and *Escherichia coli* are human pathogens.

[b] Similarities between deduced amino acid sequences of Hrp proteins from *X. campestris* pv. *vesicatoria* and other proteins include conservative amino acid exchanges.

Adapted from U. Bonas, 1994.

warming (*ASM News*, May 1996, p. 238) affect microorganism selection and survival; some of these microorganisms cause diseases in humans, in aquatic organisms, or in land plants and animals.

Microbiologists are all too familiar with the development of resistance to antibiotics used in animal and human medicine. Antibiotic resistance also occurs in bacterial plant pathogens just as resistance to fungicides can be a serious problem among fungal pathogens. A susceptible population, whether plant, animal, or human, can enable infectious disease agents to become established, spread, and evolve virulence factors that enable them to maintain themselves in the population.

One of the paradoxes of modern medicine is that while we are managing and even eradicating some infectious diseases, others are emerging. Immunosuppression, either by another disease agent such as human immunodeficiency virus or by drugs taken upon organ transplants, results in increasingly large numbers of individuals susceptible to new pathogens. In the 20th century, travel by air has obliterated time between exposure and disease outbreak, such that a traveler can spread virtually any human disease in a matter of hours.

Quarantines, isolation sites, and eradication programs can delay disease outbreaks among plants and animals. But with trade barriers being lifted and scientific materials being freely exchanged, virtually all types of microorganisms can be globally dispersed. Furthermore, animal migrations, whether on land or in the seas, take microorganisms from place to place, sometimes thousands of miles. Humans frequently introduce plants, from tulips to trees, and animals, from llamas to wolves, into nonnative environments. Microorganisms accompanying these plants and animals may be pathogenic. Similarly, microorganisms already present in the new site may infect the newcomers.

Meanwhile, the infrastructure for monitoring diseases of humans, animals, wildlife, aquatic life, and plants has been allowed to deteriorate. Even when monitoring indicates a disease outbreak and a response is mounted, it is almost always compartmentalized. Data banks on human diseases are still fragmentary at best. Rarely are animal diseases tracked, much less plant diseases. For instance, the only national data bank on emerging and reemerging plant diseases is maintained voluntarily under the auspices of the American Phytopathological Society and housed at North Carolina State University.

Recognizing Diseases and Dealing with Consequences

Alerting the public to new and reemerging diseases serves several interests, including public safety, environmental stewardship, and stimulating researchers to take action. Reporting of human diseases is more efficient than that of plant or animal infectious diseases for several reasons. A plant or animal disease may be localized or considered minor.

In aquatic systems, infectious diseases typically are difficult to detect. Hence, the focus of attention is on economically important organisms such as seaweed, oysters, shrimp, and saleable fish. Preliminary reports

often generate fear among producers and administrators, who worry because of economic or other possible reprisals, especially if disease will affect prices or international trade. Nonetheless, diseases are a fact of modern life. Whatever the commercial consequences, the public and commercial welfare requires information and technology to prevent or mitigate infectious diseases, including emerging and reemerging diseases.

The question for each disease is simple: what is the economic impact? Social and trade disruptions, hardships, and intangible impacts often make this impact hard to measure. Thus, if a species such as the chestnut is lost, what is the economic and ecological impact? Loss of wildlife, such as eagles, and loss of ornamental trees, such as the dogwood, are difficult to quantify economically. Even when the economic impact can be measured, e.g. corn futures, it is a moving target that may change daily.

Some diseases warrant special attention because of their high incidence, severity, or other factors. For example, of the 118 emerging and reemerging diseases of plants reported in the United States from 1994 to 1996, about 10% are considered severe compared with other chronic and endemic diseases. All these factors are impediments to action.

Reasons for Increase in Emerging and Reemerging Infectious Diseases

- Population shifts
- Population growth
- Changes in human behavior
- Changes in agricultural and food practices
- Urbanization
- Crowding
- Changes in ecology and climate
- Microbial evolution (esp. selection pressure)
- Modern medicine (esp. immunosuppression)
- Modern travel
- Modern trade
- Animal migration
- Animal relocation
- Inadequancy of public infrastructure

Issues and Needs

A more comprehensive strategic approach to surveillance, basic and applied research, and preventive measures makes sense in considering infectious diseases on such a broad basis (e.g. Pro Med, *ASM News*, March 1995, p. 116). In principle, the United States and the United Nations have the technological capacity to better integrate and coordinate information on this subject. For example, agreeing on a standard database for diseases affecting plants, animals, and humans would be a valuable step. Delineating common principles and techniques for disease diagnosis can be useful and may lead to a more cost-effective use of materials for testing. Environmental monitoring of changes in habitats, land use patterns, and mean temperatures can provide a better understanding of disease agents and conditions conducive to disease outbreaks.

The scientific papers contained in this report support improved coordination, communication, and new program development in comparative pathobiology across a broad range of plants and animals as a way of more fully understanding and managing disease agents that infect them. Savings in the public and private sector will accrue from such efforts. Information gained from studying plant pathogens may even prove applicable to human diseases, and vice versa. Anticipated discoveries will lead not only to new insights into the disease agents themselves, but also to disease prevention, interdiction, and understanding of host responses to infection.

A major goal of all governments is to provide a safe and secure public health infrastructure and food systems that are compatible with environmental stewardship. To reach this goal, a more holistic view of infectious diseases is needed, including emerging and reemerging diseases of plants, animals, and humans.

Suggested Reading

Barinaga, M. 1996. A shared strategy for virulence. Science 272: 1261–1263.

Bonas, U. 1994. *hrp* genes of phytopathogenic bacteria, p. 79–97. *In* J. L. Dangl (ed.), Bacterial pathogenesis of plants and animals. Molecular and cellular mechanisms. Springer-Verlag, Berlin.

Dangl, J. L. 1992. The major histocompatibility complex a la carte: are there analogies to plant disease resistance genes on the menu? Plant J. 2:3–11.

Dwyer, S. C., L. Legendree, P. S. Low, and T. L. Leto. 1996. Plant and human neutrophil oxidative burst complexes contain immunologically related proteins. Biochim. Biophys. Acta 1289:231–237.

Koonin, E. V., and V. V. Dolja. 1993. Evolution and taxonomy of positive-strand RNA viruses: implications of comparative analysis of amino acid sequences. Crit. Rev. Biochem. Mol. Biol. 28:375–430.

Rahme, L. G., E. J. Stevens, S. F. Wolfort, J. Shao, R. G. Tompkins, and F. M. Ausubel. 1995. Common virulence factors for bacterial pathogenicity in plants and animals. Science 268:1899–1902.

Rheinheimer, G. 1991. Aquatic microbiology, 4th ed. Wiley-Liss, New York. (Translated from the German by N. Walker).

Walsh, T. J., and A. Pizzo. 1988. Treatment of systemic fungal infections: recent progress and current problems. Eur. J. Clin. Microbiol. Infect. Dis. 7:460–475.

Wharam, S. D., V. Mulholland, and G. P. C. Salmond. 1995. Conserved virulence factor regulation and secretion systems in bacterial pathogens of plants and animals. Eur. J. Plant Pathol. 101:1–13.

Selected Examples of Emerging and Reemerging Infectious Diseases in Animals

Three diseases have significance for both animals and humans

JOHN A. SHADDUCK, RALPH STORTS, and L. GARRY ADAMS

John A. Shadduck (corresponding author) is dean of the College of Veterinary Medicine, Texas A&M University, where he, L. Garry Adams, and Ralph W. Storts are all professors of veterinary pathology.

New diseases emerge and old diseases re-emerge, sometimes in new hosts. Somehow this biological cycle of infectious diseases is embedded in the grand scheme of things, much as we might like it not to be. Here we briefly highlight recent examples of a newly emerging disease, bovine spongiform encephalopathy (BSE); a reemerging disease, tuberculosis (TB); and a disease previously known only in animals but now also found in humans, microsporidiosis.

Each of these diseases illustrates important points about infectious agents. BSE, which is one of several transmissible spongiform encephalopathies (TSEs) that affect humans and animals, is a disease that emerged recently in cattle and seems to have a human counterpart, although this contention has not been proved. Arguments abound as to the origin of BSE in cattle and whether a newly identified variant form of Creutzfeldt-Jakob disease in humans (vCJD) is due to the same agent that causes BSE.

TB is a disease recognized from antiquity. Although still not conquered, the incidence of TB in both humans and animals was markedly reduced during this century but resurged during the late 1980s and early 1990s.

In contrast to TB, microsporidiosis in humans was extraordinarily rare until it emerged as one of several opportunistic infections among AIDS patients. Microsporidosis exemplifies how research on a disease in animals can produce dividends, not only resulting in a better understanding of the animal disease itself, but also because the basic understanding of the organism and its pathogenesis will contribute to a more rapid solution of the human disease.

BSE, a Newly Emerging Disease

BSE refers to the characteristic microscopic vacuolization of neurons that occurs in various areas of the brain. BSE, a progressive degenerative disease, was first recognized in the United Kingdom in 1986. BSE is also known popularly as "mad cow disease," a confusing designation because "mad" has been used to describe the clinical signs of rabies in various mammalian species. BSE belongs to the disease group known as the TSEs, which can be transmitted between animals, at least within a single species. In addition to BSE, this group includes scrapie, a disease of sheep and goats (see figure); transmissible mink encephalopathy, found in farm-raised mink; feline spongiform encephalopathy, in domestically raised cats; and a chronic wasting disease of mule deer and elk. Four similar but rare diseases occur in humans, including kuru and CJD.

The recent emergence of BSE raises three very important concerns. First, in Great Britain it has led to the death of more than 150,000 cattle. Second, sale of British beef in the European Union was significantly curtailed and included an outright ban for several months. Third, some British scientists say that a variant of the BSE agent may be linked to the recent vCJD occurring in Great Britain. However, transmission of the disease from cattle to humans is unproved.

The new vCJD, which was detected in at least 12 persons in Great Britain since the emergence of BSE, differs from classical CJD in three specific ways: age of onset, clinical course, and type of lesions. The typical age of onset of classical CJD is between 50 and 70 years, whereas all patients afflicted with vCJD

were younger than 40 years of age. Also, vCJD is more prolonged than the classical form, and none of the patients afflicted with vCJD had electroencephalogram features associated with classical CJD. Finally, the plaques that form within the brain in victims of vCJD resemble those that typically occur in humans with kuru. Only rarely do vCJD lesions resemble those seen in classical CJD or, for that matter, in cattle with BSE.

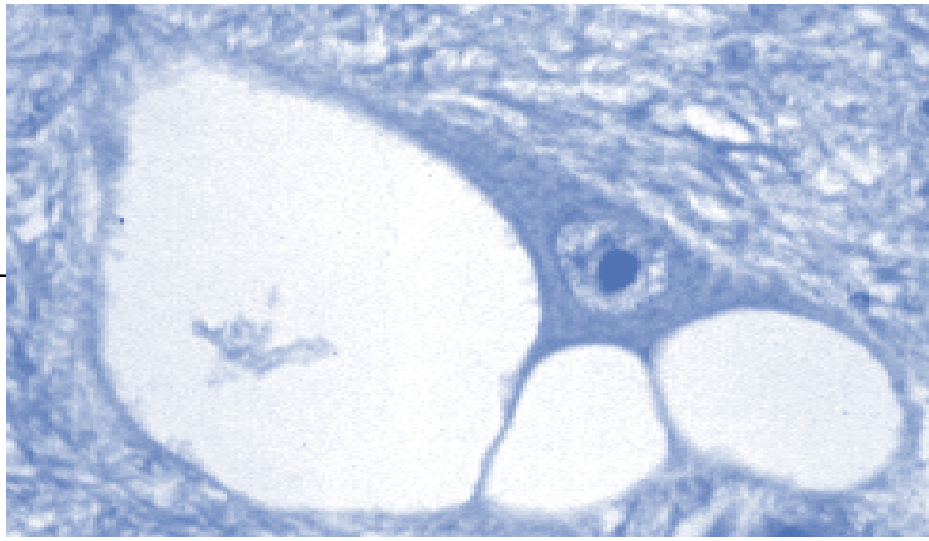
The epidemiologic evidence linking BSE with vCJD is far from definitive. Nonetheless, some British scientists concluded that in the absence of any other explanation, there could be an association between the recent occurrence of vCJD in Great Britain and the emergence of BSE. Several factors make it difficult to determine a cause-and-effect relationship. For example, for both diseases the incubation period can extend over several years, during which time the evidence relating to exposure and infection is obscured. Moreover, individuals in presumably high-exposure occupation groups, such as veterinarians, butchers, and meat packers, who are most likely to contact BSE-infected cattle, scrapie-infected sheep, or materials from such animals, show no increased incidence of TSE. Although there is still great concern over whether people can acquire vCJD by consuming beef, BSE has not been detected in the United States.

Although the agents responsible for different TSEs vary biochemically, they share several basic characteristics. Unlike conventional viruses, the TSE agents resist inactivation by heat, UV and ionizing radiation, and exposure to chemicals such as formaldehyde. The agent appears to consist of a glycoprotein without an associated nucleic acid. This proteinaceous infectious particle, or prion protein (PrP), of 27 to 30 kDa, selectively infects neurons of the central nervous system, causing intracellular membrane injury, vacuolation, and cell death. Healthy neurons and other brain cells produce a version of the prion protein that is noninfectious, varies with regard to amino acid composition from one species to another, and has an unknown function. According to Stanley Prusiner of the University of California, San Francisco, the infectious form of the prion or mutation alters the normal prion protein, resulting in endogenous production of the infectious form of the agent and disease.

A BSE outbreak in the United States would have a devastating, economic impact. Early in 1996, the beef cattle industry in Great Britain lost approximately one-third of its value. In the United States, the cost could easily exceed $50 billion. In Britain, $60 million has been spent on research and $200 million on control of BSE. An aggressive research program is needed to focus on developing tests to detect the disease prior to any clinical signs, characterizing the causative agent of the disease, and determining whether there is a valid link between BSE and vCJD.

Lessons from the emergence of BSE in the United Kingdom include the following:

- Emerging diseases may threaten both animals and humans
- Research is essential for diagnosing, preventing, and controlling new diseases such as BSE
- Research builds upon previous, sometimes esoteric knowledge—for example, about scrapie, which has been a neglected subject in the United States
- The U.S. Department of Agriculture Animal and Plant Health Inspection Service has proved that preventing BSE by aggressive surveillance and regulation is much cheaper than containment and eradication.



Spongiform Lesion in a Sheep with Natural Scrapie

A neuron of the ventral gray matter of the spinal cord contains three prominent cytoplasmic vacuoles that compress the remainder of the cell body, containing a pale nucleus with a dark central nucleolus. Vacuolation of the neuronal processes (e.g., dendrites), which is typical of some other forms of the spongiform encephalopathies, can also occur.

TB, a Reemerging Disease

TB in cattle and deer is a reemerging disease with a serious new manifestation, the increased occurrence of drug-resistant strains. TB-infected cattle can infect other animals, including farmed deer (rarely freeranging deer), in which the incidence of transmission can be quite high. Cattle also can transmit the disease to humans and cause severe illness, although the risk of infection is low, except where unpasteurized milk is consumed.

Bovine TB exerts significant economic effects. For instance, infected animals are poor producers of meat and milk. Moreover, states or countries with bovine TB often are restricted or prohibited from exporting cattle or deer. Bovine epidemics thus are potentially devastating, particularly in Texas, the number one producer of beef cattle in the United States.

A number of factors led to the reemergence in cattle, including an outdated, deficient diagnostic test for cattle; lingering TB infections in a few herds scattered throughout the United States, but especially concentrated in Texas; expanded trade resulting in the importation of animals from areas where bovine tuberculosis is rampant; rapid growth of the farm deer industry; absence of an adequate treatment for tuberculosis in deer; lack of an effective vaccine; state and federal fiscal constraints resulting in a markedly reduced surveillance program; and insufficient appreciation among producers and veterinarians of the importance of bovine TB.

Curtailing the reemergence of this disease has proved difficult. Available diagnostic tests are not particularly accurate, especially for detecting individually infected animals. In addition, there are no approved vaccines for cattle or deer in the United States. Also, problems have been encountered in treating infected animals and humans. The treatment of infected animals is far too costly. Treatment of humans is becoming dangerously ineffective because of drug-resistant TB strains.

Recommendations for addressing these problems include the following:

- Developing improved diagnostic tests for cattle, deer, and other animal species
- Developing effective vaccines
- Selecting TB-resistant animals
- Producing adequate levels of therapeutic products to combat drug-resistant strains of the organism
- Heightening public awareness of this disease.

In addition, regulatory and quarantine procedures should be improved and expanded. However, without better rapid diagnostic tests, implementing such recommendations will be difficult. Meanwhile, modern molecular epidemiologic approaches are facilitating the identification and typing of *M. bovis.*

Microsporidiosis, a New Human Disease Previously Known Only in Animals

Long before it appeared as a human disease, microsporidiosis was well recognized among a group of parasitic diseases in animals. Microsporidia are obligate intracellular protozoan parasites that cause disease among laboratory animals. Because such cases arise spontaneously and, in the past, sometimes were not recognized, they have caused confusion and led to misinterpretation of results involving experimental animals that were inadvertently infected with microsporidial organisms. More recently, with improved methods of detecting and eliminating microsporidial infections, such problems have been curtailed. Infection control in laboratory settings is based on reducing exposure of healthy animals to infected animals by removing them from colonies. Hence, microsporidosis is rarely found in laboratory animal colonies.

Meanwhile, microsporidiosis is occurring more often in humans, primarily in AIDS patients. Some species of microsporidia infect both animals and humans, while others infect only humans. Microsporidiosis is usually an opportunistic infection, and in humans occurs primarily in AIDS patients. The disease in humans can be severe, with enteritis and pneumonia occurring commonly. Clinically silent encephalitis and nephritis are frequently seen in animals. A variety of antimicrobial compounds have been demonstrated to have some effect, but a well-established therapeutic regimen for infected animals or humans is yet to be developed. Even the "effective" antimicrobial agents only inhibit replication of the organism without effectively combating the latent infection, which in all probability is maintained by spores of the parasite.

Microsporidiosis is an excellent example of a new group of human diseases that have well-established animal counterparts. Much that is known about the disease was learned by studying it the spontaneous disease in animals; through these studies, we have

gained an increased understanding of the mechanisms by which the parasites propagate, the importance of sensitized T-cells and activated macrophages in the control of parasite replication, and the pathogenesis of the disease. Exploitation of these and other discoveries should result in rapid advances in the diagnosis, control, and prevention of the disease.

Suggested Reading

Baker, M. D., C. R. Vossbrinck, E. S. Didier, J. V. Maddox, and J. A. Shadduck. 1995. Small subunit ribosomal DNA phylogeny of various microsporidia with emphasis on AIDS related forms. J. Eukaryotic Microbiol. 42:546-570.

Collinge, J., and C. Weissmann (ed.). 1994. Molecular biology of prion diseases. Phil. Trans. R. Soc. Lond. B 343:357-447.

Dankner, W. M., N. J. Waecker, M. A. Essey, K. Moser, M. Thompson, and C. E. Davis. 1993. *Mycobacterium bovis* infections in San Diego: a clinicoepidemiologic study of 73 patients and a historical review of a forgotten pathogen. Medicine 72:11-37.

Didier, E. S., P. J. Didier, D. N. Friedberg, S. M. Stenson, J. M. Orenstein, R. W. Yee, F. O. Tio, R. M. Davis, C. R. Vossbrinck, N. Millichamp, and J. A. Shadduck. 1991. Isolation and characterization of a new human microsporidian, *Encephalitozoon hellem* (n.sp.), from three AIDS patients with keratoconjunctivitis. J. Infect. Dis. 163:617-621.

Essey, M. A., and M. A. Koller. 1994. Status of bovine tuberculosis in North America. Vet. Microbiol. 40:15-22.

International Association of Biological Standardization (ed.). 1993. Transmissible spongiform encephalopathies—impact on animal and human health. Acting editor, F. Brown. Dev. Biol. Standardization 80:1-227.

Liberski, P. P. (ed.). 1993. The enigma of slow viruses: facts and artifacts. Arch. Virol. (Suppl.) 6:1-194.

Perumaalla, V. S., L. G. Adams, J. B. Payeur, J. L. Jarnagin, D. R. Baca, F. Suarez Guemes, and T. A. Ficht. 1996. Molecular epidemiology of *Mycobacterium bovis* in Texas and Mexico. J. Clin. Microbiol. 34:2066-2071.

Shadduck, J. A., and J. M. Orenstein. 1993. Comparative pathology of microsporidiosis. Arch. Pathol. Lab. Med. 117:1215-1219.

Will, R. G., et al. 1996. A new variant of Creutzfeldt-Jakob disease in the UK. Lancet 347:921-925.

Curtailing the reemergence of TB in cattle has proved difficult . . . .Available diagnostic tests are not particularly accurate, especially for detecting individually infected animals.

Reemerging and Emerging Infectious Diseases: Economic and Other Impacts on Wildlife

Transport of animals sometimes spreads infections, while other outbreaks are a mystery

VICTOR F. NETTLES

Victor F. Nettles is the director of the Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, The University of Georgia, Athens.

Reemerging and emerging infectious diseases threaten wildlife species in two key ways. First, an infectious disease that springs up might annihilate a wildlife population. Human interference with a habitat or a species introduction could trigger such events. In addition, normative diseases, when introduced, almost always pose a threat to naive populations. Second, when wildlife species are identified as part of the system that maintains or distributes an infectious agent to livestock, poultry, pets, or people, these species may be targeted for destruction. Thus, if a particular species is perceived to be the carrier of a pathogenic agent that harms humans or their domestic animals, there is pressure to control or eliminate the wild animal culprits. It makes no difference whether a disease agent kills wildlife outright or humans do so to control the disease that species carries; in either case, the resource value will decline.

The National Survey of Fishing, Hunting and Wildlife-Associated Recreation documents the tremendous economic value of fish and wildlife. According to its latest survey, 108 million Americans over 16 years old participate in some form of wildlife-associated recreation and spend more than $59 billion per year. Fish and wildlife-associated recreation represents almost 1% of U.S. economic activity and is part of the $300 billion outdoor recreational package that ranks among the top three industries in 39 of the 50 states. Hunting, the smallest segment in this wildlife use package, generates annual retail sales exceeding $12.2 billion, with a multiplier effect of $35.1 billion and generating $391 million in sales taxes. Moreover, some 410,900 jobs are created, leading to more than $8.8 billion worth of earnings, $98 million in state income taxes, and more than $1.1 billion in federal income taxes. Wildlife resources have undeniable economic value.

Recent instances of emerging and reemerging infectious diseases in wildlife have caused a variety of problems, often contributing to health difficulties for domestic animals and humans.

House Finch Conjunctivitis

In early 1994, observers began noting eye infections in house finches, known as house finch conjunctivitis, nesting in the suburbs of Washington, D.C. Since then, such infections have been noted among finches throughout much of the eastern United States and Canada—in at least 20 states and three Canadian provinces. Although its overall impact on house finch populations is unknown, the mortality rate is high for untreated birds held in captivity. The infection by the bacterium *Mycoplasma gallisepticum* leads to eyelid swelling that, when severe, causes blindness. This new disease syndrome affects a very large taxonomic group of birds, called passerines.

The same bacterial species causes mycoplasmosis, long a serious problem in commercial poultry. The disease in domestic turkeys is called infectious sinusitis; in chickens, air sacculitis. Losses from condemned and downgraded carcasses at slaughter, reduced egg production, poor feed conversion, and medication costs make this one of the most costly diseases confronting the poultry industry. The sudden appearance of *M. gallisepticum* in ubiquitous songbirds such as the house finch adds a new dimension of complexity to an already problematic poultry pathogen.

Molecular studies of house finch-associated isolates of *M. gallisepticum* suggest that they differ from strains commonly infecting domestic poultry. In experiments, house finch isolates of *M. gallisepticum* caused severe disease following direct inoculation of young domestic turkeys and chickens. However, the bacterium is slow to cross from infected house finches to chickens placed in the same pen.

This disease, although largely confined to house finches, affects large percentages of birds in areas involved in outbreaks. Recreational feeding of birds, a pastime that involves 86 million Americans, apparently enhances the spread of this disease. Other factors, such as social and migratory behavior as well as potential egg-to-young transmission, could worsen the problem. Moreover, because this pathogen recently was isolated from goldfinches, there is a serious threat of *M. gallisepticum* spreading into other bird species.

Chronic Wasting Disease

Bovine spongiform encephalopathy (BSE), or "mad cow disease," is a fatal neurologic disease of cattle that recently has received worldwide attention because of suspected infections in humans. BSE belongs to a family of fatal syndromes that cause a slow, spongelike deterioration of the brain; they include scrapie in sheep, transmissible encephalopathy in minks, Creutzfeldt-Jakob disease in humans, and chronic wasting disease in mule deer and elks.

The occurrence of chronic wasting disease, now called spongiform encephalopathy, in mule deer, elk, and white-tailed deer in Colorado and Wyoming leads to progressive weight loss, increased thirst and urination, excessive salivation, and behavioral abnormalities. Most of the more than 100 observed cases have been in captivity-reared research animals. Nevertheless, the disease also occurs in free-ranging elk and deer living near affected penned cervids.

The chronic wasting syndrome caused heavy losses in two research herds and poses a serious threat to game-ranched deer and elk and other free-living ruminants. In January 1996, chronic wasting disease was diagnosed in Saskatchewan in an elk imported 6 years earlier from South Dakota. The prolonged incubation period and uncertainty about how this disease is transmitted could make it impossible to determine the source of this infection or how it spread.

No human health problems are associated with chronic wasting disease in cervids. Even suspicion of such a link, however, could have devastating consequences on wildlife conservation efforts.

Velogenic Newcastle Disease

Velogenic Newcastle disease, a pathogen of poultry that is considered foreign to the United States, recently "found" several new hosts, including double-crested cormorants, white pelicans, and ring-billed gulls. A major outbreak occurred in Canada during the summer of 1995. Newcastle disease virus also caused extensive cormorant losses in three western Canadian provinces in 1990; in 1992, it erupted in four Canadian provinces and seven U.S. states. During the 1992 outbreak, the virus spilled over from wild birds to domestic turkeys in North Dakota, forcing agriculture authorities to destroy all infected and exposed turkeys.

Newcastle disease virus is a significant threat to poultry because it can be extremely costly to eradicate when it enters intensively managed poultry populations. For instance, in 1971 a major outbreak in southern California cost $56 million to eradicate over a 3-year period. Although most of the recent outbreaks in cormorants have occurred in areas remote from poultry, U.S. Department of Agriculture officials are encouraging poultry growers to change biosecurity procedures and eliminate opportunities for wild birds to contact poultry.

Recent instances of emerging and reemerging infectious diseases in wildlife have caused a variety of problems, often contributing to health difficulties for domestic animals and humans.

Cervid TB

For several decades, the United States has waged war against bovine tuberculosis (TB). Only a handful of infected cattle herds remain, and they are under quarantine. Hence, infections in wildlife are deemed especially troublesome.

Concerns arise over two issues. First, infections have reemerged in cervids held on game farms. Since January 1991, *Mycobacterium bovis* infections have been confirmed in at least 31 such cervid herds. After extensive culling, only four herds remain under quarantine at this time. Handling such infections is difficult, and spillover infections from cervids may cause infections in cattle and wild deer.

A second problem erupted recently with the discovery of bovine TB in free-ranging white-tailed deer in Michigan during 1994 and 1995. The deer trace to one 132-square-mile area encompassing private hunting clubs with high-density deer populations. Winter-long supplemental feeding is considered to be an important factor in creating artificial concentrations of deer and favoring high levels of exposure to *M. bovis.* The clubs are being asked to stop feeding deer and to increase the harvest. Although there is no known correlation between TB infections in humans and deer, hunters in Michigan are being advised to take special precautions when field dressing deer and to seek assistance if the viscera appear unusual.

M. bovis appears rarely in free-ranging deer, and most cases have been attributed to "spill-over" from infected cattle. Thus, most specialists thought that bovine TB is not maintained in wild deer populations. Hence this unusual occurrence of TB in wild white-tailed deer in Michigan may be eliminated by halting artificial feeding and reducing wild populations. Otherwise, a new and unwelcome chapter on deer health will need to be written.

Brucellosis in Bison and Elk

The national bovine brucellosis eradication program has nearly reached its goal. Soon, U.S. cows will be free of this abortion-causing bacterium. But the infection has a high prevalence among the 3,000 bison and 90,000 elk in the Yellowstone region of Wyoming, Idaho, and Montana. Herding behavior maintains this infection naturally in bison, but a winter feeding program promotes the infection among elk herds. How will ranchers in these states protect their herds from reinfection? Is there any hope of eradicating brucellosis in these wild animals? Will our foreign trading partners accept the claim that the United States is brucellosis free? These emerging issues are being addressed by the Greater Yellowstone Interagency Brucellosis Committee, and committee members face some difficult decisions.

Opossum Parasite Attacks Horses

Once known as the "wobblers syndrome," equine protozoal myeloencephalitis (EPM) is a severe and often fatal neurologic disease of horses that occurs when *Sarcocystis* protozoans infect the spinal cord. Infected horses become uncoordinated and, in severe cases, paralyzed. There are no vaccines available for EPM, and drug treatments are dependent upon early detection.

Recently, researchers established a link between an opossum parasite and the horse disease. The Virginia opossum is the definitive host for the one-celled intestinal parasite, *Sarcocystis falcutula.* When birds ingest sporocysts in opossum feces, they develop sarcocysts in their muscle tissues. The cycle is completed when opossums eat infected birds. Presumably horses become infected by ingesting sporocysts shed in opossum feces. In horses, the protozoans invade and multiply in the central nervous system.

Because the opossum serves as a reservoir for a parasite that damages horses, this situation provides a perfect example of how an emergent disease influences public opinion about a wild animal species. Certainly there is a much lower tolerance of opossums around horse farms, particularly where expensive horses are involved.

Rabies Relocation

For many years, the same rabies virus was thought to infect the full range of susceptible animals. More recently, researchers learned that specific host-adapted rabies virus strains exist for raccoons, skunks, red foxes, gray foxes, coyotes or dogs, and bats. In a given region, one kind of animal will serve as the rabies reservoir. Other wildlife, domestic animals, and human beings sometimes become infected but do not maintain the viral infectious cycle.

Because rabies virus strains vary, transporting infected wildlife from one region to another can cause rabies outbreaks. For example, raccoon hunters apparently are to blame for the sudden appearance of raccoon strain rabies in West Virginia and Virginia in 1977. Prior to that time, the nearest cases of raccoon rabies were found several hundred miles away in South Carolina. However, the hunters are believed to have imported infected raccoons from a southern state. Since 1977, raccoon rabies has spread from West Virginia and Virginia to 12 northeastern states. Efforts to deal with this problem have cost millions of dollars.

With the rabies experience in mind, experts fear that fox or coyote rabies virus strains could be introduced if such animals are moved across regions to stock hunting enclosures. There have been two close calls. The first happened when a foxhound in Alabama died of a rabies strain previously found only in coyotes and dogs in South Texas. The hound belonged to an enclosure owner who reportedly had imported coyotes from Texas. A year later, the Texas rabies strain was confirmed in five foxhounds in Florida. Again, the hounds had been exposed to coyotes stocked in running enclosures.

Rabies dissemination by wildlife relocation is a small but important example of a very large problem. Such translocations can introduce a wide variety of disease agents to new territories. Because databases on wildlife diseases are poorly developed and regulations covering transport of nondomestic animals are weak, preventing diseases is a serious challenge.

The Emerging Diseases That We Missed

Periodically, unexplained wildlife die-offs occur. For instance, in 1994 several dozen bald eagles nesting near DeGray Lake, about 65 miles southwest of Little Rock, Arkansas, became sick and many died. Although experts examined an exhaustive list of potential agents, the explanation eluded them. Earlier that same year, 73 deer were found dead in Washington County, Maryland; the cause of death is still not known.

Could these mortality deaths be due to new diseases? Considerable information about wildlife diseases has accumulated during the past 30 years and, in several instances, the causes of mysterious wildlife deaths during the 1950s are now well understood. Credit this progress to research, much of which was sponsored by federal dollars.

Wildlife health is significantly linked to public health and economic issues. Knowledge about wildlife diseases is vital for protecting our natural resources, our livestock and poultry industries, and ultimately, human health. Programs to protect wildlife and to train specialists with expertise in wildlife diseases are essential.

Established and Emerging Infectious Diseases of Fish

As fish move, infections move with them

JOHN L. FRYER AND JERRI L. BARTHOLOMEW

J. L. Fryer (corresponding author) is professor of microbiology emeritus and the director of the Center for Salmon Disease Research, and Jerri L. Bartholomew is Assistant Professor of Microbiology and a member of the Center for Salmon Disease Research, at Oregon State University

Formal study of the diseases of fish is a relatively new discipline whose initial focus has been on economically important species. As with many plant and animal diseases, pathogens of aquatic animals often thrive within intensively cultured captive host populations in which the devastating effects of infectious diseases quickly become apparent. Although much remains to be learned about the causes and ecology of diseases among populations of wild fish, concerns for native stocks are bringing this subject increased attention.

A Variety of Pathogens, Parasites Affect Fish

Various bacteria, viruses, protozoans, fungi, and other parasites cause diseases among fish. Many of these organisms are widely recognized as important pathogens, whereas others were recognized more recently, sometimes because of an expanded geographic or host range. Some pathogens disseminate into new regions naturally (for example, among migratory fish), but some are also dispersed when fish are moved to establish recreational fisheries, to develop aquaculture, to control undesirable species, as part of the ornamental fish trade, and following unauthorized or accidental introductions. Many infected eggs or mature fish were moved into different habitats before techniques for detecting specific pathogens were developed and, in some cases, before a particular pathogen or disease was recognized.

Besides serving as vectors for pathogens, fish transferred to new regions often encounter virulent pathogens to which they had not been exposed. The effects of disease on such fish populations is often devastating and may also increase the incidence of infections among the ordinarily less susceptible native population.

Some highly infectious pathogens are transmitted both horizontally and vertically among fish and do not respond to available antimicrobial agents. A wide variety of chemical and antimicrobial agents have been used to treat cultured fish. However, federal restrictions on the use of such agents in aquaculture has reduced the number of drugs available. This has resulted in the reemergence of some pathogens.

For example, the antifungal dye malachite green is no longer available because of its carcinogenic activity in warm-blooded animals. Hence, a water-soluble, nontoxic, inexpensive fungicide is needed for treating eggs and fish. Therapeutic agents for aquaculture include the potentiated sulfonamide Romet, sulfamerazine and oxytetracycline for certain septicemic bacterial infections, and formalin for treating external parasites and fungi. There are no agents available for treating viral infections except for povidone iodine compound to disinfect egg surfaces.

Efficacious vaccines against four major bacterial diseases of fish are available: vibriosis *(Vibrio anguillarum*, *V. ordalii*, and *V. salmonicida*), furunculosis *(Aeromonas salmonicida)*, enteric redmouth disease *(Yersinia ruckeri)*, and enteric septicemia of catfish *(Edwardsiella ictaluri)*. However, this small number of vaccines, the dwindling supply of approved therapeutic agents, and the emergence of drug resistance among some strains of bacterial pathogens (e.g., *Aeromonas salmonicida* and *Flexibacter psychrophilus)* pose major challenges.

Bacterial Diseases

Approximately 50 species of bacteria have been isolated from diseased fish (see table for partial list). Although some of them are only tenuously associated with disease, many are pathogenic. However, in wild populations, it is difficult to obtain information on the prevalence or severity of infections, making it difficult to determine whether these bacteria represent new or emerging disease agents.

Before 1989, rickettsiae were not recognized as important pathogens of fish. However, that year a previously unknown rickettsia (*Piscirickettsia salmonis* gen. nov., sp. nov.) proved responsible for large-scale

Important Bacterial Pathogens of Fish[a]

BACTERIUM	DISEASE
Gram negative	
Vibrio anguillarum[b]	Vibriosis
Vibrio ordalii[b]	Vibriosis
Vibrio salmonicida[b]	Hitra disease
Aeromonas salmonicida[b,d]	Furunculosis
Aeromonas hydrophila[c] septicemia	Motile aeromonad
Pasteurella piscicida	Pasteurellosis
Edwardsiella tarda[c]	Edwardsiellosis
Edwardsiella ictaluri	Enteric septicemia
Yersinia ruckeri disease	Enteric redmouth
Flexibacter psychrophilus disease	Bacterial coldwater
Flexibacter columnaris	Columnaris
Flexibacter maritimus	Flexibacterosis
Flavobacterium branchiophilum	Bacterial gill disease
Piscirickettsia salmonis[b]	Rickettsiosis
Gram positive	
Renibacterium salmoninarum[b]	Bacterial kidney disease

[a] Based on virulence and frequency of isolation.
[b] Considered obligate pathogens of fin fish.
[c] May be associated with human diseases.
[d] Three subspecies.

die-offs of coho salmon *(Oncorhynchus kisutch)* reared in seawater netpens in the waters of southern Chile (Figure 1).

What first appeared to be a disease confined to the waters of southern Chile took on broader significance in 1992-1993 when *P. salmonis* was found in diseased salmonid fish in western Canada, Norway, and Ireland. These agents are now widespread, and the host range includes many salmonids besides coho.

Developing methods of control is a high research priority in Chile, where *P. salmonis* epizootics are severe. Although the bacterium is sensitive to several antibiotics in vitro, its intracellular location seems to protect it. Unlike many other rickettsiae, no vector has been identified for *P. salmonis*, and there is evidence for direct transmission.

Since the appearance of *P. salmonis*, other rickettsiae have been found in ornamental fish and have caused die-offs in cultured tilapia in Taiwan. Because so little is known about these agents, they can be readily disseminated to susceptible hosts.

In contrast to the newly emerging rickettsial agents, *Renibacterium salmoninarum* has proved troublesome to salmonid fish for many years. This pathogen infects salmonids in most areas of the world causing a form of kidney disease that was first recognized in Atlantic salmon in Scotland in 1930 and soon appeared throughout Europe and North America. Pacific salmon appear to be very effective carriers of the infection, causing major epizootics of bacterial kidney disease. Although *R. salmoninarum* has been disseminated by introductions, it has also been detected in feral fish populations with no history of human intervention.

Preventing spread of bacterial kidney disease is difficult because the bacterium is transmitted both vertically and horizontally. Moreover, chronically infected carrier fish are difficult to detect and remove from healthy populations. Treatment with antibiotics is ineffective, and no vaccine is available. Erythromycin is currently the drug of choice, although it is not approved for use in treating fish destined for human consumption.

Viral Infections

In the late 1950s, researchers studying viral pathogens developed fish cell culture lines and techniques. Since then, some 150 cell lines have been used to isolate and characterize at least 88 viral agents from fish—36 DNA and 52 RNA viruses. Another 30 viruses have been observed by electron microscopy but not further characterized. Members of the *Rhabdoviridae* and *Herpesviridae* are among the most important viral pathogens of fish, whereas viruses belonging to the *Reoviridae* seem to be the most numerous.

Viral hemorrhagic septicemia virus (VHSV), a prominent rhabdovirus found in salmonids in Europe for many years, recently was found in North America. The virus is enzootic in Europe, where it is probably maintained commensally in brown trout (*Salmo trutta)* or other native salmonid species. It has been suggested that the disease emerged after susceptible rainbow trout were introduced into Europe.

In 1988, a new strain of this viral agent was isolated from adult salmon in Washington State. In response, severe control measures were introduced, including sacrificing and burying all fish on site, chlorinating water supplies, and sanitizing the affected facilities. When fish surveillance throughout the Pacific

Bar, 1µm.

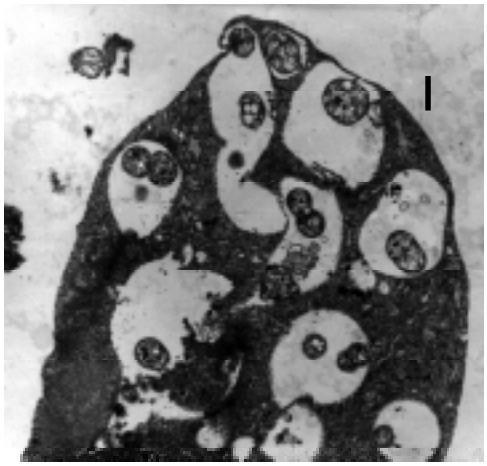


Figure 1. Ultrathin Section of CHSE-214 Cells Showing *P. salmonis* SLGO-94 within Membrane-Bound Cytoplasmic Vacuoles

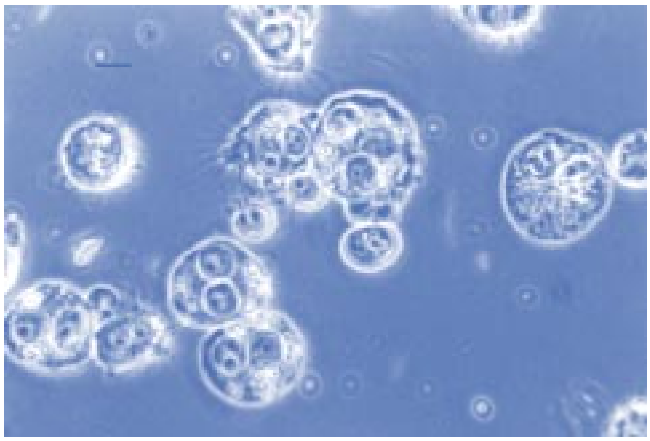


Figure 2. Phase-Contrast Micrograph of *C. shasta* Trophozoites

Northwest was increased, the virus was also detected in lesions on Pacific cod and herring. Molecular studies suggest that this strain is not of European origin and that Pacific herring may be its natural host. Although the Pacific isolate of VHSV is relatively avirulent for salmonids, this virus is highly virulent in Europe, where much effort is directed toward developing appropriate control measures.

The Myxosporean Parasites

Although more than 1,300 species of myxosporea are parasites of fish, the majority cause minimal damage to their hosts. These parasites have complex life cycles and exhibit a high degree of specificity for their fish host, the tissue they infect, and the species of alternate annelid host. Because the geographic distribution of myxosporean parasites depends on the presence of both the host fish and annelid, dissemination to new regions can result from introduction of either host.

Myxosporean parasites can be disseminated when either the host fish or annelid is moved to a new region.

The parasite *Myxobolus cerebralis* coexists with native European brown trout but causes whirling disease in rainbow trout, which were introduced nearly a century ago. The pathologies of whirling disease—tailchasing behavior, black tail condition, and cranial and skeletal deformities—derive from the parasite infecting and digesting the cartilaginous tissues of very young fish. The disease was introduced into the United States in the 1950s and now affects trout and salmon in 21 states.

The parasite is a subject of concern and controversy. For example, the sudden appearance of whirling disease in fish taken from the Madison River in Montana aroused anxiety over rainbow trout and the sport fishing industry these fish support. Although drastic measures were once used at affected hatcheries, current control methods focus on eliminating earthen rearing ponds that provide habitats for the annelid host. However, this strategy cannot be applied in natural ecosystems where whirling disease, if established, seriously threatens resident fish.

In contrast to whirling disease, the distribution of *Ceratomyxa shasta* (Figure 2), a myxosporean parasite of salmon and trout, is limited to California and the Pacific Northwest of the United States and Canada. Unlike whirling disease, which mainly infects young fish, *C. shasta* infects salmonids at all ages and the disease affects both outmigrating juvenile salmon and adults returning to spawn in fresh water. Control methods involve use of pathogen-free water at hatcheries and elimination of aquatic annelid habitats.

A system is needed for developing national policies and procedures and for evaluating risks from transporting fish and fish products. Federal agencies, including the Departments of Interior, Commerce, and Agriculture, need to coordinate their efforts with state fishery departments and organizations in the private sector.

Acknowledgments

We thank Pedro Smith, University of Chile, Santiago, Chile, for providing the SLGO-94 strain of *P. salmonis*.

Support for preparation of this paper was provided by Sea Grant under project no. R/FSD-26, Oregon State University Agricultural Experiment Station Technical Paper no. 11,035.

Suggested Reading

Bartholomew, J. L., J. S. Rohovec, and J. L. Fryer. 1989. *Ceratomyxa shasta*, a myxosporean parasite of salmonids. U.S. Fish & Wildlife Service. Fish Disease Leaflet 80.

Fryer, J. L., and C. N. Lannan. 1993. The history and current status of *Renibacterium salmoniarum*, the causative agent of bacterial kidney disease in Pacific salmon. Fish. Res. 17:15-33.

Fryer, J. L., C. N. Lannan, S. J. Giovannoni, and N. D. Wood. 1992. *Piscirickettsia salmonis* gen. nov., sp. nov., the causative agent of an epizootic disease in salmonid fishes. Int. J. Syst. Bacteriol. 42:120-126.

Ganzhorn, J., J. S. Rohovec, and J. L. Fryer. 1992. Dissemination of microbial pathogens through introductions and transfers of finfish p. 175-192. *In* A. Rosenfeld and R. Mann (ed.), Dispersal of living organisms into aquatic ecosystems. Special publication of Maryland Sea Grant.

Meyers, T. R. and J. R. Winton. 1995. Viral hemorrhagic septicemia virus in North America. Annu. Rev. Fish Dis. 4:3-24.

Wolf, K., and M. E. Markiw. 1984. Biology contravenes taxonomy in the *Myxozoa:* new discoveries show alternation of invertebrate and vertebrate hosts. Science 225:1449-1452.

Emerging and Reemerging Diseases of Plants

Plant diseases can have devastating effects on societies and economies

WILLIAM E. FRY

Plant diseases, although directly harmful to plants, also significantly influence the course of human events. Exotic pathogens have devastating impacts on certain host plant species. Nonetheless, because so many people live in or near cities, they are largely unaware of plant diseases. The large number of plant species and the even larger number of diseases also make it difficult for non-experts to know much about this important subject.

However, most of us are aware of the harmful effects of the late blight disease of potato which caused the Irish potato famine of the mid-19th century. This disease repeatedly destroyed potato crops in Ireland during the late 1840s and subsequently. These crop losses led to the deaths of 1 to 2 million Irish and forced a similar number to emigrate—mainly to North America. The current population of Ireland is still less than in the early 1840s, whereas approximately 47 million Americans now claim some Irish ancestry, with most such families coming because of potato late blight.

Thousands of microbes and viruses cause infectious diseases of plants. In contrast to pathogens of animals and humans, most plant pathogens are fungi, although bacteria, viruses, nematodes, and phytoplasmas also cause problems. Despite the drastic differences in hosts and despite many unique features, plant-infecting and animal-infecting microbes share many common mechanisms of pathogenesis. For example, plant and animal bacterial pathogens use similar pathways to secrete molecules involved in pathogenesis. However, fungal pathogens can destroy perfectly healthy plants, whereas many fungal pathogens of animals are ineffectual except among individuals with impaired defense systems.

Like pathogens of animals and humans, pathogens of plants can be moved around the world, sometimes causing severe epidemics. In the United States and Canada, examples include Dutch elm disease and chestnut blight—both caused by exotic fungi. In both cases, the host was a dominant feature of the ecosystem and indigenous to North America, and the fungal pathogen probably originated in Asia. American chestnuts, once a dominant feature of eastern hardwood forests, are now nearly extinct. American elms are similarly nearly extinct from their previous eastern and midwestern range. These two diseases illustrate the principle that plants that have not coevolved with a pathogen may be particularly susceptible to it. This same principle applies to potatoes and the fungus that causes potato late blight.

Because certain plant pathogens are especially important economically, many countries quarantine them. The quarantines reduce or can even eliminate markets for specific agricultural or forestry products, sometimes when a particular pathogen poses no significant threat to the recipient country. Thus, quarantines also have political implications. For instance, quarantines can be used to protect an industry from competition. The implications of removing a quarantine may include significant economic loss. The seriousness of some quarantines was illustrated in 1995 when a Japanese scientist committed suicide after discovering a plant pathogen in Japan that threatened a sheltered agricultural product.

The American Phytopathological Society began a survey in 1993 of diseases that pose special threats to plants in the United States. Experts were asked about incidents involving emerging or reemerging plant diseases. They identified a series of fungi, bacteria, nematodes, and viruses that are particularly threatening, including those that cause four diseases of major recent importance. Those four diseases are those caused by geminiviruses, Karnal bunt in wheat, reemergent late blight in potatoes, and wheat scab.

William E. Fry is a professor in the Plant Pathology Department, Cornell University, Ithaca, N.Y, and immediate past president (1996-1997), American Phytopathological Society, St. Paul, Minn.

Geminiviruses

For the last decade, geminiviruses transmitted by the whitefly *(Bemisia tabaci)* have been destroying more and more crops in tropical and subtropical regions of the world. In the Old World, geminiviruses cause serious damage to cassava, tomatoes, mung beans, and cotton. By 1987, African cassava mosaic virus was present in all major cassava-growing areas of Africa, and a severe epidemic is occurring in Uganda. In the Middle East, tomato yellow leaf curl virus can quickly wipe out tomato crops. Since 1994, cotton leaf curl virus has caused losses in excess of $2 billion in Pakistan's cotton export trade.

Meanwhile, in the New World, the geminivirus-whitefly complex is affecting several important food crops, including beans and tomatoes. Sometimes entire fields are abandoned because virtually all of the plants are affected. Faced with such losses, a common response is massive overuse of insecticides at considerable cost but without significant benefit. Applications are often made every 2 to 3 days and sometimes even daily.

With such detrimental economic, social, and environmental consequences from vegetable crop losses, the Ministries of Agriculture in Central America declared a regional emergency in 1992. Five hundred tomato growers in Comayagua, Honduras, lost about $4.6 million in tons/ha. In Boaco, Nicaragua, the production of beans decreased from 3.2 to 0.7 metric tons/ha. In areas around Grecia, Costa Rica, average tomato yields have decreased from 35 to 21 metric tons/ha and many growers have lost entire fields. In some cases, governments have compensated growers for their losses.

Similar situations exist in other Central American countries. In Mexico, the geminivirus-whitefly complex is the most important constraint on vegetable production. Tomato yellow leaf curl virus from the Middle East was introduced into the Dominican Republic and Jamaica in the early 1990s, and a nearly complete loss of tomato crop occurred in 1994. This newly introduced virus is also present in Cuba.

Geminiviruses pose a threat to agriculture in southern United States. Geminivirus-caused epidemics have recently occurred on tomatoes and beans in Florida and on peppers in Texas. These viruses are primarily a problem in tropical or subtropical regions, because the whitefly vector is limited by temperature.

Several factors brought about this serious rise in geminivirus-caused epidemics over the last 10 years, including (i) the introduction of the B biotype of the whitefly, which is able to reproduce more effectively than indigenous whiteflies on many different crops; (ii) changes in cropping patterns and agricultural practices; (iii) the introduction of exotic geminiviruses; (iv) difficulties in breeding plants for resistance; (v) the high diversity of geminiviruses and the occurrence of mixed infections; and (vi) the development of whiteflies with tolerance to insecticides.

Management of the geminivirus-whitefly complex remains a challenge. A new systemic insecticide was released recently and is being used extensively to reduce whitefly populations. Emerging technologies, such as rDNA methods for engineering plants with resistance to geminiviruses, show promise. Efforts are also under way to provide better worldwide communication among scientists studying the geminivirus-whitefly complex. These activities include an E-mail network and international conferences. The next international workshop will be in Puerto Rico in the spring or early summer of 1998.

Fungus-Caused Karnal Bunt Is New to U.S. Wheat

Karnal bunt is a disease of wheat caused by a fungus that was not detected in the United States prior to March 1996. Because of a quarantine, wheat from affected areas including the Indian subcontinent (the probable origin of the fungus) and, more recently, Mexico could not be imported. The disease itself rarely reduces yields but in high concentration creates an off flavor.

However, with the recent detection of the pathogen in several states, including Arizona, Texas, and California, the entire U.S. export crop ($5 billion) is at risk. Although in the short term wheat from unaffected locations in the United States is being exported, the future for wheat is uncertain.

The governing council of the American Phytopathological Society has raised questions about plans for a Karnal bunt eradication program. It also called for an international meeting to evaluate strategies for managing diseases such as Karnal bunt and to reevaluate quarantines as a strategy for controlling plant diseases.

Exotic Strains of Late Blight Again Threaten Potatoes

More than 150 years after late blight disease devastated potato crops in the United States and Europe and led to the Irish potato famine, this scourge is again creating a major problem worldwide. Despite decades during which this disease was controlled, recent migrations of exotic strains of *Phytophthora infestans* have caused a worldwide resurgence of late blight disease. The first of the recent migrations probably occurred in the late 1970s and introduced exotic strains into Europe. Additional migrations and secondary migrations have occurred throughout the last quarter of this century.

In the United States and Canada, the effects of exotic strains were not noticed until 1989, when late blight was severe in the Pacific Northwest. Subsequent migrations affected most potato/tomato production areas by 1995. The speed at which these few clonal lineages of *P. infestans* spread surprised growers and scientists alike. The exotic strains represent a more serious threat than did the previous indigenous strains, which the exoties are displacing. The new strains represent both mating types (A1 and A2) and are especially pathogenic on tomatoes, and some appear to be more aggressive on potatoes than were the previous indigenous strains. The exotic strains also are largely resistant to the systemic fungicide metalaxyl.

The most significant long-term impact of the current migrations is that this pathogen reproduces sexually more often than previously. In the United States and Canada, the pathogen had been asexual since the mid-nineteenth century. Sexual reproduction leads rapidly to new gene combinations. Additionally, release of refractory *P. infestans* sexual spores (oospores) will make the disease more difficult to control.

Initial indications are that exotic strains will cause more severe disease outbreaks in the United States and Canada and that epidemiologic patterns also will change. The U.S. Department of Agriculture (USDA) is sponsoring a national workshop on the disease in January 1997, and smaller meetings with growers are occurring all across the United States. Investigations into disease epidemiology, management practices, pathogen genetics, and development of plant varieties with stable resistance to late blight are especially needed.

The reemergence of late blight as a serious disease came as an unwelcome surprise in the United States and Canada. While fungicide resistance was expected, enhanced pathogenicity was not. In their initial response, many growers have increased their use of fungicides to suppress this disease. For example, in Washington and Oregon in 1995, the cost of managing this disease was estimated at $30 million. Costs over the previous 5 years throughout the United States are estimated at well above $500 million.

Fungal Epidemics Surge in Wheat, Other Grains

Wheat scab is caused by fungi in the genus *Fusarium* and causes devastating damage to the heads of wheat and barley. The most common pathogen in the United States, *Fusarium graminearum*, also can infect rye, oat, corn, and other grasses. In corn, the pathogen causes stalk, ear, and root rot. The disease may be found wherever these cereal crops are grown but is most common in more humid areas.

The effects of scab infection are many and severe: reduced yields, reduced seed weights, reduced seed quality, possible production of mycotoxins (chiefly vomitoxin), poor grain quality, and reduced market grade and price. Recent outbreaks in the United States and Canada have caused severe economic losses. Scab outbreaks have been frequent since 1991 in the more humid areas of wheat production. Unparalleled losses due to scab were recorded in the hard red spring wheat and spring barley regions of

More than 150 years after late blight disease devastated potato crops in the United States and Europe and led to the Irish potato famine, this scourge is again creating a major problem worldwide.

North Dakota, Minnesota, South Dakota, and Manitoba in 1993; an estimated 190 million bushels of wheat were lost to the disease, and additional quality losses brought the total loss to over $1 billion. Barley producers and the malting barley industry of the area also reported major losses, primarily because of quality and vomitoxin concerns. Moreover, local communities lost agricultural business and reported reduced income. Severe scab outbreaks also occurred in the central Midwest in 1991 and 1996.

Several factors are contributing to these recent epidemics. For example, in 1993, record rainfall in July in areas of North Dakota, Minnesota, and South Dakota when wheat and barley crops were flowering proved favorable for scab development. Another factor is the increase in acreage under conservation tillage. States such as Iowa, Illinois, Indiana, and Ohio rank high in no-till acres and North Dakota ranks second in acreage under mulch or minimum tillage. The source of the scab fungus is infested surface residue, and with less tillage to bury this residue, the risk of having more scab spores available to infect the crop is high. Lack of crop rotations contributes to the potential for scab epidemics. In some states, there are substantial acreages of corn or wheat and barley with very limited rotations.

Because no one can control the weather and the trends in tillage practice are not likely to reverse, the best strategy for reducing head scab is to develop resistant varieties and to increase crop rotations. Plant breeders now have access to resistant genetic stocks from around the world, and new cooperative efforts have formed to develop resistant varieties.

Perspective

Diseases pose a constant threat to plants used in agriculture and found in rural and suburban landscapes, and exotic diseases are a significant component of that threat. Unfortunately, the effort to address exotic diseases is far from what is needed. Except for occasional technical articles and some mention in general textbooks, plant pathologists have focused little effort on exotic diseases as a group. USDA supports only a small research program at Frederick, Md., but this effort is dwarfed by the magnitude of the problems.

Scientists working on exotic diseases of plants seldom communicate with their colleagues studying the exotic diseases of animals and humans. Although the pathogens may be dramatically different and the hosts are certainly different, there are common issues that need to be explored.

In this period of constantly increasing global commerce and travel, transport of exotic pathogens of all types will also increase. These pathogens will cause significant damage and harm whether the hosts are animals, humans, or plants. Recognizing the threat and taking appropriate action should lessen the eventual harmful impact.

Acknowledgments

Thanks to Marcia McMullen, extension plant pathologist, North Dakota State University, Fargo, for information about wheat scab and to D. P. Maxwell, University of Wisconsin, Madison, for information on geminiviruses.

Public and Scientific Affairs Board

Chair: Gail Cassell, Ph.D.

Staff: Office of Public Affairs
Janet Shoemaker, Director
Amy Melnick, Manager
Linda Murphy, Manager
Heather Garvey, Program Assistant

tel: 202/942-9209
fax: 202/942-9335
email: publicaffairs@asmusa.org

American Society for Microbiology
Office of Public and Scientific Affairs
1325 Massachusetts Avenue, NW
Washington, DC 20005-4171