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Wildlife Disease Management: An Insurmountable Challenge?

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16 Wildlife Disease Management: An Insurmountable Challenge?

Scott E. Henke, Alan M. Fedynich, and Tyler A. Campbell

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Wildlife biologists are increasingly being thrust onto the frontlines of wildlife disease management and surveillance in the United States. For example, biologists now routinely engage in oral vaccination of wildlife [e.g., Oral Rabies Vaccination Programs (ORVPs)] and collect specimens for both diagnostic purposes and disease surveillance or monitoring (e.g., avian influenza in migratory birds). For many, these responsibilities are novel compared to the more traditional roles of population estimation, habitat manipulation, and formulation of harvest recommendations. Wildlife disease investigation and management, in reality, are in their infancy compared with disciplines of human and domestic animal disease management. The recent focus on wildlife diseases is attributable to (1) the emergence of zoonoses that have a clear wildlife component (e.g., Lyme disease), (2) the recognition that wildlife can serve as reservoirs for diseases important to domestic animals (e.g., pseudorabies), (3) the increase in game farming and the associated risk of disease transmission to free-living wildlife (e.g., chronic wasting disease), (4) recognition of risks associated with the translocation of wildlife, and (5) intensified management for species at risk of extinction (Wobeser 2002). Clearly, interest in wildlife diseases and their management will continue to expand as humans, livestock, and wildlife come into closer contact as a direct or indirect result of the burgeoning human population.

In the context of “*Linking Ecological Theory and Management Applications*,” we believe it appropriate and timely to examine historic and current wildlife disease management activities aimed at preventing, controlling, or eradicating a particular disease-causing agent. Although some of these management activities have been highly successful at achieving their objectives, others

have been replete with challenges. Nevertheless, we do not believe wildlife disease management is an insurmountable challenge. Successful wildlife disease management is possible when scientists (e.g., ecologists, biologists, modelers, pathologists, virologists, and toxicologists) and practitioners (e.g., managers, agriculturalists, and veterinarians) participate together in formulating and implementing management plans with clearly stated goals and objectives (Wobeser 2006), and with the benefit of sufficient resources. We conclude this chapter with how we think the profession of wildlife disease management is evolving.

DISEASE MANAGEMENT: PRE-1900

Generally, few substantive regulations were passed during the eras of wildlife abundance (1600–1849) and overexploitation (1850–99) aimed at conserving wildlife (Taber and Payne 2003), and notions of wildlife disease management were largely nonexistent. Early concepts pertaining to infectious disease agents were not yet developed, primarily because of uncertainty of what actually caused disease. Not until the invention of a sufficiently powerful microscope in 1674 by Anton van Leeuwenhoek were scientists able to observe organisms as small as protozoans. The ability to observe microbes and microscopically examine diseased tissues dramatically advanced concepts pertaining to human disease agents and provided the foundation for advancing science-based human disease management and epidemiological theory. Advances in understanding human diseases provided the foundation from which domestic animal and wildlife disease management would emerge. This allowed for progress to be made in describing and classifying disease agents, assigning clinical signs to the disease, developing concepts pertaining to disease agent transmission, and characterizing mortality events within the context of causative agents.

During the infancy of domestic animal and wildlife disease management, scientists and practitioners began to recognize the ability and need to manage several diseases important to livestock. In 1884, the Bureau of Animal Industry (BAI) was established specifically to eradicate the infectious cattle disease, contagious bovine pleuropneumonia (CBPP) (Walton 2000). The sweeping success of the CBPP eradication program (i.e., accomplished in only 8 years through quarantine of contact animals, slaughter of infected animals, and disinfection of infected premises) ensured the continuation of this new, yet important Bureau within the United States Department of Agriculture (USDA). The BAI was the forerunner of the USDA Animal and Plant Health Inspection Service (APHIS), the agency currently charged with “Protecting America’s Agriculture,” including wildlife damage and disease-related issues. Though not the intended purpose, many USDA APHIS activities in managing selected livestock diseases (Table 16.1) altered the dynamics of diseases as they relate to wildlife hosts as well.

DISEASE MANAGEMENT: 1900–1949

Wildlife management during the first half of the twentieth century was characterized by eras of protection (1900–1929) and game management (1930–65) (Taber and Payne 2003). Aldo Leopold’s (1933) landmark text *Game Management* shaped wildlife management theory and practices through the later years of this period and into the present. Concurrently, scientists began foundational investigations involving diseases of wildlife. Some of these early studies included determining the vulnerability of small mammals to plague (McCoy 1911), describing tularemia in rodents (McCoy and Chapin 1912), and characterizing avian botulism in waterfowl (Kalmbach and Gunderson 1934).

Establishment of The Wildlife Society and its affiliated scientific publication *Journal of Wildlife Management* in 1937 marked the beginning of formalized study in wildlife management. The importance of wildlife diseases was recognized and emphasized from the journal’s inception. For example, in Volume 1 there was a call for cooperation between parasitologists and wildlife biologists (Van Cleave 1937); Volume 2 included articles concerning leucocytozoonosis in ruffed grouse

TABLE 16.1**Partial List of Events, Occurrences, Milestones, or Accomplishments of USDA's Animal and Plant Health Inspection Service (APHIS)**

Disease	Year	Event, occurrence, milestone, or accomplishment
Classical swine fever (CSF)	1833	First reported in southern Ohio and along the Wabash River in Indiana
	1903	Discovered that CSF is caused by a virus
	1906	Serum-virus method of immunizing swine developed
	1913	First license issued for production of anti-CSF serum
	1951	First use of modified live virus vaccines for CSF
	1961	Congress authorizes CSF eradication program
	1965	All states are enrolled in a four-phase CSF eradication program
	1969	Government bans the use of modified live virus CSF vaccines
	1970	Task force approach first used to eradicate a CSF outbreak
	1972	Secretary of Agriculture declares CSF emergency, providing additional funding
	1973	First CSF-free month in more than 100 years
Foot-and-mouth disease (FMD)	1870	First known outbreak of FMD in the United States
	1914	Largest outbreak of FMD occurs in the United States; more than 3,500 livestock herds are infected
	1924	United States Army kills 22,000 deer in containing FMD outbreak
	1929	Last of 9 FMD outbreaks occurs in the United States (1870, 1880, 1884, 1902, 1908, 1914, 1924 [2], and 1929) is eradicated
	1930	Tariff Act of 1930 prohibits imports of animals or animal products from countries infected with FMD
	1979	APHIS establishes FMD "vaccine bank" with Canada and Mexico
Bovine tuberculosis (BTB)	1892	First tuberculin test of a herd was made in the United States; of the 79 animals tested, 30 were reactors
	1900	Tuberculin test is required for all imported cattle
	1917	Congress appropriates \$75,000 to begin efforts against bovine TB; 5% of the nation's cattle are infected
	1936	Bovine TB reactor rate drops to new low of 1%
	1940	All states are Modified Accredited Bovine TB areas (cattle infection rate <0.5%)
	1974	Bovine TB reactor rate drops to new low of 0.03%
	1984	Bovine TB reactor rate drops to new low of 0.003%
	1991	Bovine TB recognized as a serious problem in deer and elk
Brucellosis	1905	Brucellosis discovered in imported goats
	1910	<i>Brucella abortus</i> first isolated from cattle in the United States
	1916	Committee on Contagious Abortion formed by the United States Livestock Sanitary Association to encourage control of brucellosis
	1918	Discovery of the relationship between brucellosis organisms that cause the disease in cattle, swine, and goats
	1930	Discovery of Strain 19 vaccine for brucellosis
	1934	Efforts against bovine brucellosis begin as part of a "cattle reduction plan" caused by severe drought conditions
	1936	National brucellosis herd infection rate estimated at 14–15%
	1940	Vaccination with Strain 19 becomes part of the brucellosis control program
	1942	North Carolina becomes the first Modified-Certified Brucellosis Area (infection in <5% of herds and 1% of cattle)
	1947	First Uniform Methods and Rules (UM&R) are adopted for brucellosis program
	1952	Brucellosis milk ring test adopted as a surveillance tool in dairy herds

TABLE 16.1
Continued

Disease	Year	Event, occurrence, milestone, or accomplishment
Brucellosis (Continued)	1954	Accelerated brucellosis eradication program begins; an estimated 124,000 herds are infected nationwide
	1960	Market cattle testing program adopted as a surveillance tool for brucellosis in beef cattle
	1961	Swine brucellosis eradication program begins
	1978	Brucellosis Technical Commission reports its findings that "control leading to eradication is biologically feasible"
	1979	New standards adopted for brucellosis eradication program
	1982	New state classifications become effective for brucellosis eradication program
	1988	<i>Ad hoc</i> committee on brucellosis in Yellowstone National Park is formed
	1989	APHIS begins "Rapid Completion Plan" to finish its brucellosis eradication program
	1990	Number of cattle herds quarantined for brucellosis drops below 1,000 for the first time
	1992	Number of cattle herds quarantined for brucellosis drops below 500 for the first time
	1995	Number of cattle herds quarantined for brucellosis drops below 100 for the first time
Screwworm	1938	The theory of using laboratory-reared sterilized insects to control and eradicate pest populations is conceived
	1951	Procedures developed for sterilizing screwworms, enabling control of this pest and others with sterile insect technology
	1954	Screwworms successfully eradicated from the Island of Curacao after 4 months using the sterile insect technique
	1958	Screwworm eradication begins in the southeastern United States using the sterile insect technique; eradication is accomplished in 1959 and a quarantine line is established at the Mississippi River to prevent reinfestation
	1962	Screwworm eradication program begins in the southwestern United States
	1966	Overwintering screwworm populations are eradicated in the United States
	1981	APHIS closes screwworm production plant in Mission, Texas
Pseudorabies virus (PRV)	1983	Five states (Iowa, Illinois, North Carolina, Pennsylvania, and Wisconsin) partake in pilot projects against PRV
	1986	First genetically engineered vaccine is licensed by APHIS (for PRV in swine)
	1989	APHIS adopts standards for 5-stage State-Federal-industry program to eradicate PRV in swine
	1995	All states in stage 2 or higher; 14 states have "free" status
Rabies	1991	Field trial successfully completed on Parramore Island, Virginia, for the first genetically engineered oral raccoon rabies vaccine
	1995	APHIS launches campaign against coyote rabies in Texas using genetically engineered oral vaccine

Note: Includes predecessor organizations in managing selected livestock diseases important to wildlife from 1833 to 1995 (modified from <http://permanent.access.gpo.gov/lps3025/history.html>)

(*Bonasa umbellus*) (Clarke 1938), waterfowl parasites (Gower 1938), and coccidiosis in muskrats (*Ondatra zibethica*) (Shillinger 1938); and Volume 3 contains papers involving blood parasites of deer (Dougherty 1939; Whitlock 1939a), *Echinococcus* infections in wildlife (Riley 1939), larval tapeworms in cottontail rabbits (*Sylvilagus floridanus*) (Whitlock 1939b), *Plasmodium* infections in wild birds (Wetmore 1939), and a description of "a freak deer head" with cutaneous fibromas (Honest 1939). This interest in wildlife pathogens and diseases set the stage for other emerging professional organizations such as the Wildlife Disease Association, which was formed in 1951 and consists

mostly of wildlife disease specialists. Volume 1 of the Bulletin of the Wildlife Disease Association was published in 1965 and became *Journal of Wildlife Diseases* in 1970.

A vast majority of the scientific studies conducted from 1900 to 1950 involving wildlife diseases were descriptive in nature, aimed at identifying the disease-causing agent, suitable hosts, and disease presence. Our understanding of most wildlife diseases had not yet advanced to the point where management recommendations could be made with a healthy degree of scientific rigor. In attempts to eradicate the large liver fluke (*Fascioloides magna*), for example, broadcast application of copper sulfate was used to kill aquatic snails, the fluke's intermediate host (Swales 1935). This management practice was successful in eradicating the liver fluke, but at the expense of beneficial snails (Pybus 1990). The unintended consequences of such disease management activities pointed to the need to develop a more thorough ecosystem-based theoretical approach to disease management.

PRESENT DISEASE MANAGEMENT

This period has seen movement away from preoccupation with large mortality events often focused upon by biologists in earlier time periods and toward adoption of a theoretical based approach to disease and disease management. Emphasis is being directed on the role that diseases actually play in wildlife populations, understanding the disease cycle to find the weakest link, and identifying the underlying mechanisms that promote stable and unstable host-pathogen systems (Anderson and May 1978; May and Anderson 1978).

There are two general or overarching strategies employed in present disease management. The first option is the "hands-off" approach, which is applicable to infectious disease agents. Its foundation is established in ecological theory, in which all organisms are components of the ecosystem and the concept that ecological systems evolve toward equilibrium states. In this view, the host and the pathogen are in a continuous battle of survival: The pathogen seeks to maintain a viable population within a susceptible host without "damaging" the host population to the extent that it causes the pathogen's own extinction. Simultaneously, selective pressures on the host result in behavioral modifications to avoid the pathogen or enhancements in immunological resistance. When the pathogen causes a significant disruption in the host population, a locally unstable situation arises until the equilibrium state is re-established, thereby permitting coexistence of host and pathogen. In situations where there are large, healthy wildlife populations, human intervention is often considered ineffective and economically costly, because an equilibrium state will establish itself naturally.

Equilibrium states that occur between pathogens and hosts have been demonstrated in several long-term field studies. The seminal study by van Riper et al. (1986) and van Riper (1991) on the impact of the introduced malarial parasite *Plasmodium relictum* on Hawaiian avifauna found that natural selection eliminated individuals that slept with their head and legs exposed to mosquito vectors. They also found that the daily bird movement patterns in some species were modified to avoid mosquito vectors. Ultimately, some avian species adapted in response to the exotic pathogen, whereas others did not and their populations declined. Thus, the host-pathogen system reached equilibrium, but host species composition and abundance was substantially different from before the introduction of the pathogen (regionally unstable equilibrium state). In another study, Pence and Windberg (1994) followed a disease cycle involving an outbreak of sarcoptic mange in a coyote (*Canis latrans*) population in southern Texas. Although there was about 80% mortality in the coyote population at the peak of the epizootic, at the end of the 10-year cycle, coyote abundance was approximately the same as prior to the epizootic, which suggested both locally and regionally stable equilibriums for this host-pathogen system.

Although the hands-off approach may apply to wildlife populations that are large and widely distributed, it does not seem applicable in three situations: (1) species that are threatened or endangered,

for example, an outbreak of avian cholera in the Nebraska Rainwater Basin during peak migration of whooping cranes (*Grus americana*) would be more risky to the cranes than to waterfowl co-occurring in the same area; (2) game species that reach a new equilibrium with a pathogen in which host density is substantially lower would not be viewed favorably by hunters; and (3) host–pathogen systems that involve zoonotic potential.

If the hands-off approach is not applicable, intervention can be an option. Some diseases can be prevented or reduced. This option can be particularly effective for certain noninfectious and infectious diseases that are caused or facilitated by human activities. Development of disease transmission theory is essential for the success of this approach, as it is necessary to determine where the weakest link is in the disease cycle and focus efforts there to have the highest probability of success.

Instances in which humans have aided in exposing wildlife to disease agents are numerous, including selenium poisoning of waterfowl and shore birds in California because of irrigation runoff (Ohlendorf et al. 1988), lead poisoning in waterfowl and upland game birds from the use of lead shot for hunting (Deuel 1985), introduction of avian malaria to Hawaii (van Riper et al. 1986), and translocation of raccoons (*Procyon lotor*), and subsequently rabies, from Florida to mid-Atlantic coast states (Nettles et al. 1979). The first two examples highlight human activities — irrigation for agriculture and hunting, which resulted in environmental contamination and wildlife diseases. The latter two examples involved translocation of animals for human purposes, which inadvertently translocated the animals' disease agents (i.e., viruses, bacteria, helminths, etc.). In the case of avian malaria, the mosquito vector was brought to Hawaii through ballast water that contained larvae (Warner 1968). Malarial parasites (*P. relictum*) were later introduced to Hawaii with importation of exotic birds (van Riper et al. 1982). Now, avian malaria is well established in Hawaii, and it has negatively affected the native avifauna. Raccoons were transported and released by hunting clubs who wished to increase raccoon density, but by doing so, also introduced protozoa, helminths (Schaffer et al. 1978), and rabies (Nettles et al. 1979) to the eastern United States. In addition, urban and suburban sprawl has led to habitat loss, resulting in the crowding of wildlife into smaller tracts of land, thereby promoting disease transmission. In each case, human activities have created or exacerbated a disease scenario, and thus, it is human responsibility to circumvent wildlife disease problems.

There are three reasons why humans consider intervention wildlife disease management: (1) disease can be a health risk to humans, (2) disease can be deleterious to domestic livestock, and (3) disease can negatively affect wildlife considered beneficial to humans. It is rare for a disease management program to evolve and not to be justified by one or more of these reasons. For example, the oral rabies vaccine program, which has placed millions of vaccine-laden baits for raccoons along the eastern coast of the United States (Hanlon et al. 1989) and for coyotes and gray foxes (*Urocyon cinereoargenteus*) in Texas (Texas Department of Health 1994; Farry et al. 1998a), has a central objective to reduce the risk of rabies to humans. Without this focus on human health, it is likely, little effort and financial resources would be expended on eradicating rabies from wildlife populations.

EXAMPLES OF DISEASE MANAGEMENT TO AID LIVESTOCK INDUSTRY

The following are examples in which concepts pertaining to disease transmission theory were used to identify the weakest link and focus control efforts to break the disease cycle.

Swine Brucellosis: Swine brucellosis is a good example of disease management to aid the livestock industry. The brucellosis eradication program primarily is concerned with bovine brucellosis (*Brucella abortus*). At present, Idaho, Wyoming, and Texas are Class A (not disease-free) for *B. abortus*. For swine brucellosis, only Texas remains at Stage 2 (not disease-free). Swine brucellosis is caused

by *Brucella suis*, a small Gram-negative bacterium. Infected animals may be asymptomatic, or have chronic clinical signs including abortion, fetal reabsorption, infertility in sows, orchitis (inflammation of the testes) in boars, lameness, and a high-mortality rate in piglets (Tessaro 1990; Davidson and Nettles 1997). Transmission occurs by oral and venereal routes, and the bacteria localize in lymph nodes with an incubation period from 2 weeks to several months (Davidson and Nettles 1997; Conger et al. 1999). A fully effective vaccine has not yet been developed, and there is no known cure for the disease. In the United States, brucellosis has been found in Alabama (Davidson and Nettles 1997), Arkansas (Zygmunt et al. 1982), California (Sweitzer et al. 1996; Davidson and Nettles 1997), Florida (Zygmunt et al. 1982; Belden 1993; van der Leek et al. 1993a; Davidson and Nettles 1997), Georgia (Hanson and Karstad 1950; Zygmunt et al. 1982; Davidson and Nettles 1997), Hawaii (Davidson and Nettles 1997), Louisiana (Zygmunt et al. 1982; Davidson and Nettles 1997), Oklahoma (Davidson and Nettles 1997), South Carolina (Wood et al. 1976; Zygmunt et al. 1982; Davidson and Nettles 1997; Gresham et al. 2002), and Texas (Randhawa et al. 1977; Corn et al. 1986; Davidson and Nettles 1997). Prevalence in feral swine populations can range from 0 to 44% (Dees 1999). Due to the potential spread of brucellosis from feral hogs to domestic pigs, it is recommended that domestic swine facilities be double fenced to reduce the chances of direct contact between feral and domestic swine. In addition, periodic disease testing of domestic swineherds is advisable.

Pseudorabies: Pseudorabies virus (PRV, Aujeszky's disease, Mad Itch) is an alphaherpes virus (suid herpesvirus 1) that occurs in swine, but can be lethal to nonswine species that contract the virus (Kocan 1990). When infection occurs, the virus travels along peripheral sensory nerves toward neurons in ganglia, where the virus maintains its latent status until reactivated during periods of host stress (Romero et al. 2003). In swine, the disease ranges from asymptomatic to fatal in young animals, and depends on strain of the disease and age of the infected animal (Davidson and Nettles 1997). Clinical signs include fever, respiratory infection, loss of coordination, abortion, mummified fetuses, stunted growth, and high mortality in piglets less than four weeks old (Kocan 1990; Davidson and Nettles 1997). A current theory is that modes of transmission differ in feral pigs versus domestic pigs due to different ganglionic sites of latency. The virus settles in the sacral (most common in feral pigs) and trigeminal ganglia (most common in domestic pigs) of the nervous system tissues, and can be isolated from the tonsil (Romero et al. 2003). In feral hogs, because the virus is in sacral ganglia, venereal transmission has the highest frequency (Romero et al. 1997, 2001, 2003), unlike that in domestic pigs where the virus is predominantly transmitted through exchange of oral and nasal fluids. However, PRV has occurred by aerosol transmission (Schoenbaum et al. 1990; Christensen et al. 1993), infected meat, and contaminated food and water (Kocan 1990; Hahn et al. 1997; Kluge et al. 1999). The wild-type of PRV, found in feral swine, appears to be attenuated, with lower pathogenicity than those found in domestic herds. Therefore, it may not manifest similar symptoms, making it difficult to recognize the virus in domestic herds (Romero et al. 1997).

Feral hogs, as a disease reservoir, can be economically significant. The U.S. pork industry is valued at \$30 billion annually, employs over 600,000 people, and produces 10% of the world's pork supply (APHIS 2003; Witmer et al. 2003), giving the industry valid concern when it comes to disease management. Since 1989, the domestic pork industry has participated in a USDA-coordinated national campaign to eradicate PRV. PRV alone costs the national pork industry an estimated \$40 million annually, not including loss of market opportunity internationally (NIAA: www.animalagriculture.com). The PRV program has five stages: stage I is preparation, stage II is control, stage III is mandatory cleanup of all pseudorabies-infected herds, stage IV is surveillance to verify no infection remains, and stage V status is when all herds are pseudorabies-free for 1 year or more. As of late 2004, all states, Puerto Rico, and the Virgin Islands were at stage V (NIAA: www.animalagriculture.com). The threat of reintroduction of these diseases to uninfected domestic herds by diseased feral populations has been considered in the scientific literature, and only recently has a disease management program been initiated (Wyckoff et al. 2005).

Pseudorabies appears to be well established in feral populations throughout the United States, and persists in populations through time (Gresham et al. 2002; Corn et al. 2004). Infected populations have been found in Florida (van der Leek et al. 1993a,b), Georgia (Pirtle et al. 1989), Oklahoma (Davidson and Nettles 1997), South Carolina (Wood et al. 1992; Gresham et al. 2002), Texas (Corn et al. 1986), and in 12 unlisted states (Miller 1993). Rates of infection have varied from not present to 70% (Pirtle et al. 1989; van der Leek et al. 1993a; Sweitzer et al. 1996; Hahn et al. 1999; Gresham et al. 2002; Corn et al. 2004). Rates of infection seemingly depend on location, season of sampling, and age structure of the sampled population (Romero et al. 1997). Similar management recommendations as suggested for brucellosis have been offered for pseudorabies.

Screwworm Eradication: Control of the blowfly *Callitroga hominivorax*, implemented under the federal Screwworm Eradication Program, provides an example for a disease management strategy to aid wildlife considered beneficial to humans, although the initial reason for the program was to aid the livestock industry. This fly lays its eggs in homeotherms, and historically cattle and white-tailed deer (*Odocoileus virginianus*) were preferred hosts. The larvae feed on living tissue, debilitating the host. In Texas, mortality of white-tailed deer fawns from screwworm infections reached 80% before control programs were implemented (Wobeser 1994). Control of this parasitic infection was based on behavioral biology of the fly; the fly breeds only once each year. Massive numbers of irradiated, and thus sterile, adult male flies were released. Female flies mated with sterile males and viable eggs were not produced, thus reducing parasitic infection in cattle and deer. Suppression resulted for several years, but occasional outbreaks have occurred (Richardson et al. 1982). Efforts are now underway to eradicate the screwworm throughout Americas.

PRESENT STRATEGIES OF WILDLIFE DISEASE MANAGEMENT

Disease management can take three forms: prevention, control, and eradication. Most disease management programs involve components of prevention, control, and eradication.

Prevention is designed to keep a disease from entering an unaffected area. Prevention typically involves restrictions on importation or translocation of certain animals. Prevention often is the easiest and most economic method of disease management. Consequences from accidental introduction of a disease can be disastrous. Zebu cattle from India were introduced into Africa and, consequently, rinderpest swept across the continent. Mortality rates for wild ruminants exceeded 90% for some species (Henderson 1982).

Disease control is designed to reduce the frequency of disease to some tolerable level. Control implies that the disease will persist in the host population and its environment, but at a level that will produce negligible effects to humans or human interests. The ORVP is a control program to reduce the infection rate of rabies in wild animals [i.e., raccoons, coyotes, gray fox, and striped skunks (*Mephitis mephitis*)]. The ORVP involved many stages, from vaccine development (Rupprecht et al. 1988), bait development (Farry et al. 1998b; Steelman et al. 1998), baiting strategies (Farry et al. 1998a), and program assessment.

Based on disease transmission theory, control can be attempted by manipulating four basic factors: the disease agent, the host population, the environment, and human activities, or by a combination of these factors. These factors have been incorporated into various theoretical models developed by R. M. Anderson and R. M. May. Management of infectious disease agents is complicated by replication of the disease agent and by transmission to other susceptible individuals in the host population (Anderson and May 1979). Reproduction rate of a disease, R , is the average number of secondary infections caused by a single infected individual that was introduced into a completely susceptible population (Fine et al. 1982). In other words, an intrinsic reproductive rate of 8 means that, on average, each infected individual resulted in the infection of eight susceptible individuals. Anderson (1982) defined the reproductive rate (R) of a disease as the density of susceptible individuals

in the population (X) divided by the threshold density for disease persistence (N_T), or $R = X/N_T$. The proportion of susceptible individuals in a population to a disease is reduced by immunization; therefore, immunization reduces R . If $R = 1$, then the disease agent is just able to maintain itself in a population. If $R < 1$, then the disease agent cannot sustain itself and will eventually become extinct from the population. Only when $R > 1$ will a disease agent become obvious in a host population. Therefore, the objective of many disease control programs is to depress the reproductive rate of a disease agent below unity. However, a disease agent with a high rate of reproduction is more difficult to control than one with a low rate (Anderson 1982). The proportion of a population (p) that must be immunized to eradicate a disease must exceed $1 - 1/R$ (Anderson 1982). For example, for reproductive rates greater than 5, more than 80% of the susceptible population must be vaccinated to eradicate the disease. It becomes obvious that diseases with high reproductive rates are extremely difficult to manage through immunization because of the large proportion of the population that must be immunized. Consider a population with a threshold density of 1 animal/km² and a population density of 2, 4, and 8 animals/km². Under these conditions, the proportion of the population that must be immunized to eliminate a disease would be 50, 75, and 88%, respectively. Another obstacle with immunization that must be considered is the average age of a susceptible host when it is exposed to the disease. For an immunization program to be effective, animals must be immunized prior to their exposure to disease. Therefore, animals that are exposed to disease at an early age are more difficult to control by immunization.

Disease eradication involves complete elimination of a disease from an area for an indefinite period. Such programs typically are large in scale and require a large investment of time and money. When foot-and-mouth disease was accidentally introduced into California in 1923, a deer eradication program was successfully initiated (Brooksby 1968).

The most direct way to manage a disease is by manipulating the disease agent. It is often easier to manipulate a noninfectious disease agent, such as a toxicant, than an infectious disease, such as a parasite. A biological or synthesized toxin eventually degrades with time, so its effects lessen as long as no new toxin is added to the environment. However, infectious agents can replicate themselves without new additions. The pesticide DDT (dichlorodiphenyltrichloroethane) was once utilized throughout the world. DDT and its metabolites DDE (dichlorodiphenyldichloroethylene) and DDD (dichlorodiphenyldichloroethane) have high stability and persistence within the environment. This organochlorine pesticide was implicated as the causative agent for eggshell thinning of birds, and thus the reason for reproductive failure in many bird species (Spitzer et al. 1978). Once highlighted as a mortality problem, efforts were made to stop the use of DDT in the United States. Upon cessation, reproductive success of birds improved, but not as dramatically as might have been expected. This pesticide and its metabolites are still being found in tissues of birds (Mora 1995; Wainwright et al. 2001). Screwworm (previously discussed) is a parasitic infection that plagued cattle and white-tailed deer. However, manipulation of the disease agent, the blowfly *C. hominivorax*, reduced the incidence of disease in wild cervids.

Disease management through manipulation of the host population has been attempted. Dispersion of animals to manage a disease can be useful if there is no chance of spreading the disease agent to a new area. Manipulation of host populations has included culling diseased animals, test and slaughter programs, and reducing population density. Selective culling can only work if infected animals are easily identified and if the disease is slow to spread through a population. Test and slaughter programs are of limited use for wildlife because of the difficulty of capturing, holding in captive facilities, and testing all individuals of a population. Reducing host population density on a local or regional scale has been attempted, but the effort to do so is typically intensive, and the results are temporary because of animal repopulation of the control area (Henke and Bryant 1999; Henke et al. 2002). Reduction of host population density through reproductive control of the host has been suggested, but does not appear to have been attempted till date. Creation of a barrier (e.g., geographic area in which animals were vaccinated to the disease) to restrict disease spread has been attempted for striped skunks to reduce the spread of rabies in Alberta (Gunson et al. 1978), for red fox (*Vulpes vulpes*)

to reduce rabies spread in Europe (Wandeler et al. 1974), and for African buffalo (*Syncerus caffer*) to reduce the spread of rinderpest in Uganda (Anonymous 1953). Depopulation has been attempted for American coots (*Fulica americana*) in Virginia to control avian cholera (Purseglove et al. 1976), for ground squirrels (*Spermophilus* sp.) in Colorado to control plague (Waltermire 1982), and for European badgers (*Meles meles*) in England to control tuberculosis (Henderson 1982).

Immunization of the host population has potential as a disease control manipulation because immunization reduces the proportion of susceptible individuals in a population, thus reducing *R*. Vaccines to immunize animals are generally available. The problem typically lies with how to deliver the vaccine to wild animals, and whether the methodology to do so is feasible and economical. The ORVP (previously described) for coyotes in Texas provides an example of a successful immunization program to control disease in a wild animal population (Texas Department of Health 1994).

Disease management through manipulation of the environment can be used to reduce the causative agent, the host population, populations of other species involved in the disease, and other factors involved in disease occurrence. Environmental manipulation typically does not provide quick results in disease management, but it does usually provide long-lasting results. Avian botulism in waterfowl provides a good example of habitat manipulation (Wobeser 1994). The bacterium, *Clostridium botulinum* type C, is the causative agent of avian botulism. This organism is found as a resistant spore within the soils of wetlands. Under anaerobic conditions, the bacteria produce a toxin. Waterfowl become poisoned when they consume vegetation or invertebrates containing the toxin. The bacteria use decaying animal matter, more so than decaying vegetative matter, as a substrate upon which to grow. The concept here is to reduce the amount of substrate, which reduces the number of bacterial spores, and thus reduces the level of toxin. Invertebrates that die because of changing water depths can provide suitable substrate for the bacteria, and thus the toxin. Occurrence of botulism can be reduced by maintaining consistent water depths via water control devices.

Wildlife disease management usually involves people management. Public support of a disease management program is necessary to get compliance with the program and financial support to conduct the program. Education programs for the public are aimed at acquiring this support. The public must understand the biology of the disease (i.e., risk to people from zoonoses), justification for disease management, and how the program will be conducted. Often, human activities must be altered to reduce risk of disease to humans and to wildlife. A rabies education program was initiated in southern Texas during the ORVP for coyotes when it was discovered that most of the public did not vaccinate their family pets against rabies (Kresta and Henke 2000). An education program explained how rabies was transmitted, the risk of exposure to humans, the effects of the disease, and how to reduce risk of exposure.

Lead poisoning of waterfowl illustrates another example of altering human activities. The effect of ingesting spent shotgun pellets was described by Westmore (1919) early in the history of wildlife management. Zwank et al. (1985) searching two lakes in Louisiana during 3 months found 783 of 1171 sick or dead ducks (67%) caused by lead poisoning. Other management techniques, such as hazing birds from areas of heavy pellet deposition and plowing areas in an attempt to bury lead shot deep in the soil, have been attempted (Wobeser 1994).

Use of nontoxic shot, beginning with first generation steel shot, was promoted as the solution to reduce lead poisoning in waterfowl. To force this change from lead shot to nontoxic shot, the U.S. Fish and Wildlife Service initiated regulations that systematically eliminated lead shot for hunting waterfowl beginning with hot-spots and eventually ending with a U.S.-wide ban in 1991 (USFWS 2004). However, the perception of additional federal regulation of hunting activity, coupled with poor ballistic performance of steel shot (compared with lead), and the much higher price per shell were not looked upon favorably by the waterfowl hunting community. With advances in nontoxic shot alternatives (bismuth–tin, tungsten–bronze, tungsten–iron, tungsten–matrix, tungsten–nickel–iron, tungsten–polymer, tungsten–tin–bismuth, and tungsten–tin–iron–nickel) allowed by the U.S. Fish and Wildlife Service during the 2005 waterfowl hunting season (<http://www.wildlifedepartment.com/approvednontoxic.htm>) more ballistically effective loads are

now available for hunting waterfowl and upland game birds. The conversion from lead shot to non-toxic shot has been a slow process, and education efforts were not the most efficient. Such difficulties in education and acceptance point to the need for focusing on information transfer to stakeholders and for effective public relations personnel in state and federal wildlife agencies.

FUTURE DIRECTIONS

Clearly, the wildlife disease profession has come a long way from its earliest endeavors of monitoring and assessing disease impacts in the early part of the century to the present where intervention strategies are being adopted and implemented. Wobeser (2002) suggested that the desire to actively manage infectious disease in wild animals is a relatively recent phenomenon, compared to disease management strategies for humans and domestic animals. Therefore, with greater scientist and general public awareness of the effect of wildlife disease agents, what does wildlife disease management hold for the future? At least four areas of focus will likely emerge. These include (1) increased global cooperation in surveillance and management of wildlife diseases, (2) increased communication with the public and people management, (3) developing sophisticated tools for solving complex disease issues, and (4) continued focus on basic and applied research.

INCREASED GLOBAL COOPERATION

Based on the recent past, emerging and reemerging diseases will likely be center-stage, particularly those infectious agents that have pandemic potential and can rapidly spread among continents. For example, the foot-and-mouth disease virus was responsible for an explosive pandemic affecting Asia, Africa, and Europe during 1998–2001 (Knowles et al. 2005). West Nile virus and avian flu are other recent examples of diseases that have spread across continents rapidly. Additionally, there is the possibility of bioterrorism using militarized infectious agents and exotic zoonotic agents. Militarized zoonotic disease agents such as anthrax have recently captured the attention of the media, public, and U.S. Homeland Security Agency. Other agents such as cattle plague, foot-and-mouth disease, African swine fever, classical swine fever (hog cholera), and avian influenza have been identified as potential agents for agricultural terrorism (Committee on Confronting Terrorism in Russia 2002). Such disease agents have the potential to disrupt many globally linked economies. For example, control measures and economic sanctions imposed during the 2001 foot-and-mouth disease outbreak in the United Kingdom had a direct loss to agriculture and its associated food chain of approximately \$3.1 billion (Thompson et al. 2002). Consequently, there clearly is a need for significant collaboration among global stakeholders. Governments will increasingly realize the need to form cooperative linkages to provide better disease surveillance, sharing of information, and database management. Benefits of such cooperation will include advanced disease detection, which should aid in control and prevention before agents reach epizootic or panzootic proportions. Both the United States and Canada have recognized the need for collaborative activities and incorporated them into their disease management strategies (USGS 2004; Ministry of Natural Resources 2005). However, increased knowledge from sharing information about country-specific disease agents will likely lead to increased controls on importation, exportation, and movement of wildlife among countries and, for zoonotic agents, potential restrictions on human travel.

INCREASED COMMUNICATION WITH THE PUBLIC

To implement an effective wildlife disease management plan, it is critical to inform the public. Many past problems in disease management were not necessarily related to the plan of action involving control or eradication, but to the ability to convince the public that the plan was appropriate (e.g., chronic wasting disease in Wisconsin, fowl plague in California). Such a lack of support can often be traced to lack of public awareness about the problem or its effect on wildlife, domestic animals, and humans.

Consequently, there will be an increasing need to have highly trained public relations personnel who can bridge information gaps between researchers and the general public and “sell” the disease management plan. Additionally, Daszak et al. (2000) suggested that it might become increasingly important to include the potential of wildlife disease impacts in Environmental Impact Statements so that the public is aware that disease issues are being considered.

As multimedia technology advances, new communication tools can be used. These tools may include Internet newsgroups, which provide rapid dissemination of credible information (Daszak et al. 2000), and pod-casts and other rapidly advancing technological outlets that can be used for quick and accurate information transfer by public relations personnel in state and federal agencies. Unfortunately, with such advances in communication technology, various interest groups can and will present biased information to advance their agendas. Consequently, the public could be swayed by inaccurate information. The importance of providing reliable and trustworthy information will become paramount.

DEVELOPING NEW SCIENTIFIC TOOLS

Innovative and cutting-edge science will be needed to control increasingly complex disease issues. Greater emphasis is needed in developing improved modeling and quantitative tools. Development of such capability will provide insights regarding transmission and spread of diseases and aid in evaluating management actions used in specific control or eradication programs (USGS 2003). Additionally, there is a need to develop models of the economics of managing infectious wildlife diseases (Horan and Wolf 2005).

Advanced technologies, such as Geographic Information Systems (GIS) and Global Positioning Systems (GPS), which incorporate satellite imaging, weather maps, habitat and land use features, and geographic distributions of disease carriers, reservoirs, and vectors, will become increasingly important in monitoring enzootic diseases and spotting early stages of emerging diseases. Studies at the Caesar Kleberg Wildlife Research Institute are incorporating GIS and GPS technology to monitor feral pig movements across the landscape to develop better control strategies. Another study is using GIS to examine relationships between *Baylisascaris procyonis*, raccoons, and human populations. Use of such technological resources is clearly part of future efforts to manage wildlife diseases.

EMPHASIS ON MORE RESEARCH

Emerging zoonotic diseases have recently been the primary focus of disease management, particularly chronic wasting disease, avian flu, and West Nile virus. There will be an increasing need to understand the dynamics of emerging diseases to develop effective management strategies (USGS 2003). Basic information will be required for monitoring and surveillance, which will provide information on which types of diseases are present, incidence of disease, patterns within susceptible populations, and associated risks to humans and livestock (Duff 2003). Additionally, more sophisticated assessments of wildlife diseases will be needed including disease dynamics, risk analysis, and development of more effective sampling techniques (USGS 2004). Focus of disease research will increasingly be on ecology, pathology, and population biology of host-pathogen systems approached from individual, population, and environmental perspectives. Thus, disease research will necessitate a multidisciplinary approach to identify causes of disease outbreaks and develop effective control measures (Daszak et al. 2000). Additionally, there is a need for studies focusing on economic costs of disease management strategies. Wobeser in his Carlton Herman Founders Fund Lecture at the 2006 Wildlife Disease Association meeting stressed the need to move from observational and descriptive studies to experimental studies in which biotic and abiotic factors are manipulated to assess impacts of disease agents on hosts under experimental scenarios. He also indicated that the large, long-term disease and mortality datasets being compiled by various state and federal agencies need to

be examined using epizootiological approaches to understand population level dynamics of disease agents.

Looking to the future, it seems that epizootiologists will encounter increased complexity. However, with multinational cooperation, greater public awareness, and the development of advanced scientific tools, wildlife disease managers can continue their efforts in moving from the theoretical realm to the applied in bettering the management of wildlife populations well into the twenty-first century.

REFERENCES

- Anderson, R. M. 1982. Transmission dynamics of indirectly transmitted disease agents: The vector component. In *Population Biology of Infectious Diseases*, R. M. Anderson, and R. M. May (eds). Berlin: Springer-Verlag, p. 149.
- Anderson, R. M., and R. M. May. 1978. Regulation and stability of host-parasite population interactions. I. Regulatory processes. *J. Anim. Ecol.* 47:219.
- Anderson, R. M., and R. M. May. 1979. Population biology of infectious disease. *Nature* 280:361.
- Anonymous. 1953. Rinderpest-buffalo free zone, Uganda. *Bull. Epiz. Dis. Afr.* 1:46.
- APHIS. 2003. Pseudorabies, www.aphis.usda.gov/vs/nahps/pseudorabies/q-a.html (accessed December 19, 2003).
- Belden, R. C. 1993. Feral hogs: The Florida experience. In *Proceedings on Feral Swine: A Compendium for Resource Managers*, D. Rollins (ed.). Austin: Texas Animal Health Commission, p. 101.
- Brooksby, J. B. 1968. Wild animals and the epizootiology of foot-and-mouth disease. *Symp. Zool. Soc. Lond.* 24:1.
- Christensen, L. S., S. Mortensen, A. Botner, B. S. Strandbygaard, L. Ronsholt, C. A. Henriksen, and J. B. Andersen. 1993. Further evidence of long distance airborne transmission of Aujeszky's disease (pseudorabies) virus. *Vet. Rec.* 132:317.
- Clarke, C. H. D. 1938. Organisms of a malarial type in ruffed grouse, with a description of the schizogony of *Leucocytozoon bonasae*. *J. Wildl. Manage.* 2:146.
- Committee on Confronting Terrorism in Russia. 2002. High-impact terrorism: Proceedings of a Russian-American Workshop, National Academy Press. <http://darwin.nap.edu/books/0309082706/html/207.html> (accessed April 12, 2006).
- Conger, T. H., E. Young, and R. A. Heckmann. 1999. *Brucella suis* in feral swine. *Proc. First Nat. Feral Swine Conf.* 1:98.
- Corn, J. L., P. K. Swideren, B. O. Blackburn, G. A. Erickson, A. B. Thierman, and V. F. Nettles. 1986. Survey of selected disease in wild swine in Texas. *J. Amer. Vet. Med. Assoc.* 189:1029.
- Corn, J. L., D. E. Stallknecht, N. M. Mechlin, M. P. Luttrell, and J. R. Fischer. 2004. Persistence of pseudorabies virus in feral swine populations. *J. Wildl. Dis.* 40:307.
- Daszak, P., A. A. Cunningham, and A. D. Hyatt. 2000. Emerging infectious diseases of wildlife — Threats to biodiversity and human health. *Science* 287:443.
- Davidson, W. R., and V. F. Nettles. 1997. *Field Manual of Wildlife Diseases in the Southeastern United States*, 2nd edn. Southeastern Cooperative Wildlife Disease Study, Athens: University of Georgia.
- Dees, T. 1999. Feral/wild swine surveillance for foreign animal diseases and some field study projects on brucellosis and pseudorabies in the southeastern USA. *Proc. First Nat. Feral Swine Conf.* 1:121.
- Deuel, B. 1985. Experimental lead dosing of northern pintails in California. *Calif. Fish Game* 71:125.
- Dougherty, R. W. 1939. Sickle cells in the blood of western deer. *J. Wildl. Manage.* 3:17.
- Duff, P. 2003. Wildlife disease surveillance by the veterinary laboratories agency. *Micro. Today* 30:157.
- Farry, S. C., S. E. Henke, A. M. Anderson, and M. G. Fearneyhough. 1998a. Efficacy of bait distributional strategies to deliver canine rabies vaccines to coyotes in southern Texas. *J. Wildl. Dis.* 34:23.
- Farry, S. C., S. E. Henke, A. M. Anderson, and M. G. Fearneyhough. 1998b. Responses of captive and free-ranging coyotes to simulated oral rabies vaccine baits. *J. Wildl. Dis.* 34:13.
- Fine, P. E. M., J. L. Aron, J. Berger, D. J. Bradley, H. J. Burger, E. G. Knox, H. P. R. Seeliger, C. E. G. Smith, K. W. Ulm, and P. Yekutieli. 1982. The control of infectious disease group report. In *Population Biology of Infectious Diseases*, R. M. Anderson, and R. M. May (eds). Berlin: Springer-Verlag, p. 121.
- Gower, W. C. 1938. Seasonal abundance of some parasites of wild ducks. *J. Wildl. Manage.* 2:223.

- Gresham, C. S., C. A. Gresham, M. J. Duffy, C. T. Faulkner, and S. Patton. 2002. Increased prevalence of *Brucella suis* and pseudorabies virus antibodies in adults of an isolated feral swine population in coastal South Carolina. *J. Wildl. Dis.* 38:653.
- Gunson, J. R., W. J. Dorward, and D. B. Schowalter. 1978. An evaluation of rabies control in skunks in Alberta. *Can. Vet. J.* 19:214.
- Hahn E. C., G. R. Page, P. S. Hahn, K. D. Gillis, C. Romero, J. A. Anelli, and E. P. J. Gibbs. 1997. Mechanisms of transmission of Aujeszky's disease virus originating from feral swine in the USA. *Vet. Microbiol.* 55:123.
- Hahn, N., C. Hsu, and B. Paszkiet. 1999. Research on PRV in feral swine: Past, present and future direction. *Proc. First Nat. Feral Swine Conf.* 1:75.
- Hanlon, C. L., D. E. Hayes, A. N. Hamir, D. E. Snyder, S. Jenkins, C. P. Hable, and C. E. Rupprecht. 1989. Proposed field evaluation of a rabies recombinant vaccine for raccoons (*Procyon lotor*): Site selection, target species characteristics, and placebo baiting trials. *J. Wildl. Dis.* 25:555.
- Hanson, R. P., and L. Karstad. 1950. Feral swine in the southeastern United States. *J. Wildl. Manage.* 23:64.
- Henderson, W. M. 1982. The control of disease in wildlife when a threat to man and farm livestock. In *Animal Disease in Relation to Animal Conservation*, M. A. Edwards, and U. McDonell (eds). London: Academic Press, p. 287.
- Henke, S. E., and F. C. Bryant. 1999. Effects of coyote removal on the faunal community in western Texas. *J. Wildl. Manage.* 63:1066.
- Henke, S. E., D. B. Pence, and F. C. Bryant. 2002. Effect of short-term coyote removal on populations of coyote helminths. *J. Wildl. Dis.* 38:54.
- Honess, R. F. 1939. A freak deer head. *J. Wildl. Manage.* 3:360.
- Horan, R. D., and C. A. Wolf. 2005. The economics of managing infectious wildlife disease. *Amer. J. Agr. Econ.* 87:537.
- Kalmbach, E. R., and M. F. Gunderson. 1934. Western duck sickness — A form of botulism. Technical Bulletin, No. 411, United States Department of Agriculture, Washington, DC.
- Kluge, J. P., G. W. Beran, H. T. Hill, and K. B. Platt. 1999. Pseudorabies (Aujeszky's Disease). In *Diseases of Swine*, B. E. Straw, et al. (eds). Ames: Iowa State University Press, p. 233.
- Knowles, N. J., A. R. Samuel, P. R. Davies, R. J. Midgley, and J. Valarcher. 2005. Pandemic strain of foot-and-mouth disease virus serotype O. *Emerg. Inf. Dis.* 11:1887.
- Kocan, A. 1990. Pseudorabies. In *Review of Wildlife Disease Status in Game Animals in North America*, R. Lind (ed.). Saskatchewan, Canada: ADF-Saskatchewan Agriculture Development Fund, p. 43.
- Kresta, A. E., and S. E. Henke. 2000. Attitudes towards rabies in southern Texas: A need for public education. In *Proceedings of the Nineteenth Vertebrate Pest Conference*, D. A. Whisson, and R. M. Timm (eds). San Diego, p. 113.
- Leopold, A. 1933. *Game Management*. New York: Charles Scribner's Sons.
- May, R. M., and R. M. Anderson. 1978. Regulation and stability of host-parasite population interactions. II. Destabilizing processes. *J. Anim. Ecol.* 47:249.
- McCoy, G. W. 1911. The susceptibility to plague of the weasel, the chipmunk, and the pocket gopher. *J. Infect. Dis.* 8:42.
- McCoy, G. W., and C. W. Chapin. 1912. Bacterium tularense the cause of a plague-like disease of rodents. *U.S. Public Health Marine Hosp. Bull.* 53:17.
- Miller, J. E. 1993. A national perspective on feral swine. In *Proceedings of Feral Swine: A Compendium for Resource Managers*, D. Rollins (ed.). Austin: Texas Animal Health Commission, p. 9.
- Ministry of Natural Resources. 2005. *Our Sustainable Future*. Ontario: Ontario Ministry of Natural Resources Strategic Directions.
- Mora, M. A. 1995. Residues and trends of organochlorine pesticide and polychlorinated biphenyls in birds from Texas, 1965-88. U.S. Department Interior, National Biological Service, Fish Wildlife Research 14, Washington, DC.
- Nettles, V. F., J. H. Shaddock, R. K. Sikes, and C. R. Reyes. 1979. Rabies in translocated raccoons. *Am. J. Publ. Health* 69:601.
- Ohlendorf, H. M., A. W. Kilness, J. L. Simmons, R. K. Stroud, D. J. Hoffman, and J. F. Moore. 1988. Selenium toxicosis in wild aquatic birds. *J. Toxicol. Environ. Health* 24:67.
- Pence, D. B., and L. A. Windberg. 1994. Impact of a sarcoptic mange epizootic on a coyote population. *J. Wildl. Manage.* 58:624.

- Pirtle, E. C., J. M. Sacks, V. F. Nettles, and E. A. Rollor, III. 1989. Prevalence and transmission of pseudorabies virus in an isolated population of feral swine. *J. Wildl. Dis.* 25:605.
- Purseglove, S. R., Jr., D. F. Holland, F. H. Settle, and D. G. Gnegy. 1976. Control of a fowl cholera outbreak among coots in Virginia. *Proc. Ann. Conf. S. E. Assoc. Fish Wildl. Agencies* 30:602.
- Pybus, M. J. 1990. Survey of hepatic and pulmonary helminths of wild cervids in Alberta, Canada. *J. Wildl. Dis.* 26:453.
- Randhawa, A. S., V. P. Kelly, and E. F. Baker, Jr. 1977. Agglutinins to *Coxiella burnetii* and *Brucella* spp., with particular reference to *Brucella canis* in wild animals of southern Texas. *J. Amer. Vet. Med. Assoc.* 171:939.
- Richardson, R. H., J. R. Ellison, and W. W. Averhoff. 1982. Autocidal control of screwworms in North America. *Science* 215:361.
- Riley, W. A. 1939. The need for data relative to the occurrence of hydatids and of *Echinococcus granulosus* in wildlife. *J. Wildl. Manage.* 3:255.
- Romero, C. H., P. Meade, J. Santagata, K. Gillis, G. Lollis, E. C. Hahn, and E. P. J. Gibbs. 1997. Genital infection and transmission of pseudorabies virus in feral swine in Florida, USA. *Vet. Microbiol.* 55:131.
- Romero, C. H., P. N. Meade, J. E. Shultz, H. Y. Chung, E. P. Gibbs, E. C. Hahn, and G. Lollis. 2001. Venereal transmission of pseudorabies viruses indigenous to feral swine. *J. Wildl. Dis.* 37:289.
- Romero, C. H., P. N. Meade, B. L. Homer, J. E. Shultz, and G. Lollis. 2003. Potential sites of virus latency associated with indigenous pseudorabies viruses in feral swine. *J. Wildl. Dis.* 39:567.
- Rupprecht, C. E., A. N. Hamir, D. H. Johnston, and H. Koprowski. 1988. Efficacy of vaccinia rabies glycoprotein recombinant virus vaccine in raccoons (*Procyon lotor*). *Rev. Infect. Dis.* 10:S803.
- Schaffer, G. D., W. L. Hanson, W. R. Davidson, and V. F. Nettles. 1978. Hematotropic parasites of translocated raccoons in the southeast. *J. Am. Med. Assoc.* 173:1148.
- Schoenbaum, M. A., J. J. Zimmerman, G. W. Beran, and D. P. Murphy. 1990. Survival of pseudorabies virus in aerosol. *Amer. J. Vet. Res.* 51:331.
- Shillinger, J. E. 1938. Coccidiosis in muskrats influenced by water levels. *J. Wildl. Manage.* 2:233.
- Spitzer, P. R., R. W. Risebrough, W. Walker, R. Henderson, A. Poole, D. Puleston, and I. C. T. Nisbet. 1978. Productivity of ospreys in Connecticut-Long Island increases in DDE residues decline. *Science* 202:333.
- Steelman, H. G., S. E. Henke, and G. M. Moore. 1998. Gray fox response to baits and attractants for oral rabies vaccination. *J. Wildl. Dis.* 34:764.
- Swales, W. E. 1935. The life cycle of *Fascioloides magna* (Bassi, 1875), the large liver fluke of ruminants, in Canada. *Can. J. Res.* 12:177.
- Sweitzer, S., I. A. Gardener, B. J. Gonzales, D. Van Uren, and W. M. Boyce. 1996. Population densities and disease surveys of wild pigs in the coast ranges of central and northern California. *Proc. Vertebrate Pest Conf.* 7:75.
- Taber, R. D., and N. F. Payne. 2003. *Wildlife, Conservation, and Human Welfare: A United States and Canadian Perspective*. Malabar: Krieger Publishing.
- Tessaro, S. V. 1990. Brucellosis caused by *Brucella suis*. In *Review of Wildlife Disease Status in Game Animals in North America*, R. Lind (ed.). Saskatchewan, Canada: Saskatchewan Agriculture Development Fund, p. 105.
- Texas Department of Health. 1994. Gray fox rabies in Texas: A status report. 20 June 1994, Texas Department of Health, Austin.
- Thompson, D., P. Muriel, D. Russell, P. Osborne, A. Bromley, M. Rowland, S. Creigh-Tyte, and C. Brown. 2002. Economic costs of the foot-and-mouth disease outbreak in the United Kingdom in 2001. *Rev. Sci. Tech.* 21:675.
- USFWS. 2004. *Waterfowl Population Status, 2004*. Washington, DC: U.S. Dep. Inter.
- USGS. 2003. Helping to combat chronic wasting disease. U.S. Geological Survey — National Wildlife Health Center Information Sheet.
- USGS. 2004. Wildlife health: Thirty years of science. U.S. Department of the Interior, U.S. Geological Survey.
- Van Cleave, H. J. 1937. Worm parasites in their relations to wildlife investigations. *J. Wildl. Manage.* 1:21.
- van der Leek, M. L., H. N. Becker, E. C. Pirtle, P. Humphrey, C. L. Adams, B. P. All, G. A. Erickson, R. C. Belden, W. B. Frankenberger, and E. P. J. Gibbs. 1993a. Prevalence of pseudorabies (Aujeszky's Disease) virus antibodies in feral swine in Florida. *J. Wildl. Dis.* 29:403.

- van der Leek, M. L., H. N. Becker, P. Humphrey, C. L. Adams, R. C. Belden, W. B. Frankenberger, and P. L. Nicoletti. 1993b. Prevalence of *Brucella* sp. antibodies in feral swine in Florida. *J. Wildl. Dis.* 29:410.
- van Riper, C., III. 1991. The impact of introduced vectors and avian malaria on insular passeriform bird populations in Hawaii. *Bull. Soc. Vector Ecol.* 16:59.
- van Riper, C., III, S. G. van Riper, M. L. Goff, and M. Laird. 1982. The impact of malaria on birds in Hawaiian Volcanoes National Park. Technical Report 47, CPSU/UH 022, University of Hawaii at Manoa, Honolulu.
- van Riper, C., III, S. G. van Riper, M. L. Goff, and M. Laird. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecol. Monogr.* 56:327.
- Wainwright, S. E., M. A. Mora, J. L. Sericano, and P. Thomas. 2001. Chlorinated hydrocarbons and biomarkers of exposure in wading birds and fish of the Lower Rio Grande Valley, Texas. *Arch. Environ. Contam. Toxicol.* 40:101.
- Waltermire, R. G. 1982. Analysis of small mammal control in a Colorado campground. MS Thesis, Colorado State University, Ft Collins.
- Walton, T. E. 2000. The impact of diseases on the importation of animals and animal products. *Ann. New York Acad. Sci.* 916:36.
- Wandeler, A. J. Muller, G. Wachendorfer, W. Schale, U. Forster, and F. Steck. 1974. Rabies in wild carnivores in central Europe: Ecology and biology of the fox in relation to control operations. *Z. Vet. Med. B* 21:765.
- Warner, R. E. 1968. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. *Condor* 70:101.
- Westmore, A. 1919. Lead poisoning in waterfowl. U.S. Dept. Agric. Bull. No. 793, Washington, DC.
- Wetmore, P. W. 1939. A species of *Plasmodium* for the sharp-tailed grouse infected to other birds. *J. Wildl. Manage.* 3:361.
- Whitlock, S. C. 1939a. Studies of the blood of white-tailed deer. *J. Wildl. Manage.* 3:14.
- Whitlock, S. C. 1939b. Infection of cottontail rabbits by *Cysticercus pisiformis* (*Taenia pisiformis*). *J. Wildl. Manage.* 3:258.
- Witmer, G. W., R. B. Sanders, and A. C. Taft. 2003. Feral swine — Are they a disease threat to livestock in the United States. In *Proceedings of the 10th Wildlife Damage Management Conference*, K. A. Fagerstone, and G. W. Witmer (eds). Fort Collins: Wildlife Damage Management Working Group of The Wildlife Society, p. 316.
- Wobeser, G. A. 1994. *Investigation and Management of Disease in Wild Animals*. New York: Plenum Press, p. 265.
- Wobeser, G. 2002. Disease management strategies for wildlife. *Rev. Sci. Off. Int. Epiz.* 21:159.
- Wobeser, G. 2006. *Essentials of Disease in Wild Animals*, Chap. 13. Ames: Blackwell.
- Wood, G. W., J. B. Hendricks, and D. E. Goodman. 1976. Brucellosis in feral swine. *J. Wildl. Dis.* 12:579.
- Wood, G. W., L. A. Woodward, D. C. Mathews, and J. R. Sweeney. 1992. Feral hog control efforts on a coastal South Carolina plantation. *Ann. Conf. Southeast. Assoc. Fish Wildl. Agencies* 45:167.
- Wyckoff, A. C., S. E. Henke, T. Campbell, D. G. Hewitt, and K. VerCauteren. 2005. Preliminary serologic survey of selected diseases and movements of feral swine in Texas. In *Proceedings of the 11th Wildlife Damage Management Conference*, K. A. Fagerstone (ed.). Ft Collins: Wildlife Damage Management Working Group of The Wildlife Society.
- Zwank, P. J., V. L. Wright, P. M. Shealy, and J. D. Newsom. 1985. Lead toxicosis in waterfowl on two major wintering areas in Louisiana. *Wildl. Soc. Bull.* 13:17.
- Zygmunt, S. M., V. F. Nettles, E. B. Shotts Jr., W. A. Carment, and B. O. Blackburn. 1982. Brucellosis in wild swine: A serologic and bacteriologic survey in the southeastern United States and Hawaii. *J. Am. Vet. Med. Assoc.* 181:1285.