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IMPLICATIONS OF INFECTIOUS DISEASES FOR CAPTIVE PROPAGATION AND INTRODUCTION PROGRAMS OF THREATENED/ENDANGERED REPTILES

Elliott R. Jacobson, D.V.M., Ph.D.

Abstract: Health and disease are becoming extremely relevant issues for the conservation biology of members of all major groups of vertebrates. The importance of disease in captive propagation, relocation, repatriation, and translocation (RRT) programs is just being appreciated. All of us experience multiple diseases in our lifetimes. Reptiles are not different. They are susceptible to the range of infectious agents known to occur in other vertebrates. The causes of disease in captive reptiles are better understood than those in their wild counterparts. However, several important diseases have recently been documented in wild chelonians. One of these, upper respiratory tract disease in the desert tortoise (*Gopherus agassizii*) is thought to have been introduced into populations of tortoises in the Mojave Desert in the southwestern USA by release of ill captive desert tortoises. A similar situation appears to exist for certain populations of the gopher tortoise (*Gopherus polyphemus*) in Florida, USA. Although conservation strategies such as RRT programs have been implemented for a number of threatened/endangered reptiles, results indicate that the success rate is rather low. Because of this low success rate and the recent awareness of the possible introduction of exotic pathogens acquired in captivity, release programs should be scrutinized more closely.

Key words: Infectious disease, propagation, release, reptiles.

INTRODUCTION

There are approximately 6,000 extant species of reptiles. Currently, 34 are listed as threatened/endangered in the United States, and 78 are listed as threatened/endangered internationally.[10] These numbers probably will continue to grow into the next century. Of those listed as threatened or endangered, the American Association of Zoological Parks and Aquariums has developed Species Survival Plans for the following five reptiles: Aruba Island rattlesnake (*Crotalus unicolor*), Virgin Island boa (*Epicrates monensis gratis*), Dumeril's ground boa (*Acrantophis dumerili*), Chinese alligator (*Alligator sinensis*), and radiated tortoise (*Geochelone radiata*). Far larger numbers of reptiles that are not listed as threatened or endangered are being bred in zoos and by the private sector throughout the world.[33] Although no data are currently available for the fate of these captive-bred reptiles, a number of captive-bred reptiles have been released to the wild in areas where these species naturally occur (pers. obs.). Other reptiles, such as the red-eared slider (*Trachemys scripta elegans*), have been shipped all over the world and have established themselves well beyond their historic range.

Relocation, repatriation, and translocation (RRT) programs involving reptiles have become an extremely popular conservation strategy to mitigate the loss of habitat or of individuals or populations in areas that have experienced declines or extirpations.[9] Although some of these programs have been successful, such as the introduction of captive-reared gharials (*Gavialis gangeticus*) to areas where they had been reduced or eliminated,[4] many of these programs either have been unsuccessful or have not been followed to determine the outcome.[9] For example, although numerous relocations of large numbers of gopher tortoises (*Gopherus polyphemus*) have occurred in Florida, USA, outcomes of these relocations have been poorly studied.

Although the effect of disease has been poorly studied and seldom reported in free-ranging populations of reptiles, epizootics

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are well documented in captive reptiles. As in birds and mammals, reptiles are susceptible to infection with a wide range of pathogens, including viruses, bacteria, fungi, and parasites. Of these, ophidian paramyxovirus infection,[5,24,25,27] a retroviruslike infection of boid snakes,[36] mycoplasmosis of tortoises,[23] herpesvirus infections of tortoises[6,21,32] and other chelonians,[13,26,35] and a recently described epizootic of chlamydiosis in green turtles in aquaculture[19] are examples of the more problematic diseases of reptiles. These agents probably represent the tip of the iceberg. Without knowing the prevalence or location of these agents in free-ranging populations of reptiles, the potential exists for introducing new pathogens into naive populations through release of captive-bred, captive-reared, and captive-held reptiles. The numbers of pathogens that can be transmitted among species in captivity is largely unknown. Also, reptiles that are free of the infectious agents found in free-ranging populations will be at risk if exposed to these agents following release.

In this paper, those significant diseases of captive and free-ranging reptiles which occur as epizootics are reviewed. The difficulties of assessing reptile health and the consequences of releasing reptiles infected with either natural or exotic pathogens are also discussed.

EMERGING AND HISTORICALLY IMPORTANT INFECTIOUS DISEASES OF REPTILES

Ophidian paramyxovirus (OMPV) infections

In 1976, a respiratory epizootic spread through a collection of fer-de-lance (*Bothrops moojeni*) in a snake farm in Switzerland.[11] Although *Pseudomonas* and *Aeromonas* were isolated from the respiratory tracts of dead snakes, a virus (FDLV) with ultrastructural properties similar to those of the myxoviruses was identified.[5] In the first reported die-off in the United States from which OPMV was recovered, eight of nine rock rattlesnakes (*Crotalus lepidus*) died with clinical signs of central nervous system disease.[25] In another report, 8% of the total viperid collection in a zoo in Louisiana died during a 2–3-mo period; the affected genera were *Crotalus, Vipera, Bothrops, Trimeresurus,* and *Bitis.*[25] In 1987, OPMV was recovered from the lungs of two dead Ottoman vipers (*Vipera xanthena xanthena*) from a zoological collection in Tennessee[34] and subsequently from several viperids that died in a zoological collection in Louisiana.[24]

Over the last 7 yr, OPMV-associated die-offs have been identified in a variety of private and zoologic collections in the USA and Europe. In some collections, although multiple species of viperid snakes were maintained, only one or two species per collection were severely affected. Certain species of *Crotalus* are particularly susceptible to infection. In epizootics of OPMV, if *C. durissus* and *C. basiliscus* are present, in most situations these will be the first species to die. In 1988, OPMV was isolated from a black mamba (*Dendroaspis polylepis*) in a serpentarium experiencing an epizootic in viperids, elapids, boids, and colubrids. In 1988, OPMV was also isolated from clinically ill corn snakes (*Elaphe guttata*), beauty snakes (*E. taeniurus*), and Molendorff's ratsnakes (*E. moellendorffi*).[22] In Germany, a myxoviruslike agent was recovered from a red-tailed ratsnake (*E. oxycephala*), gaboon viper (*Bitis gabonica*), and carpet python (*Morelia spilotes*).[1,2] Thus, the range of species that become ill and die extends beyond the family Viperidae. A nonviperid may be the ultimate source of infection.

Clinical signs of illness are variable. In some cases, the signs are extremely subtle; the snake is found dead in its cage without suspicion of a problem. In other cases, snakes may simply go off feed, regurgitate, or be polyuric.[11] A series of stages were described in a colony of fer-de-lance. In stage 1, snakes had a loss of muscle tone; affected snakes exhibited a "stretched out" linear

posture with the head slightly elevated. During stage 2, which lasted 1–2 days, snakes showed abnormal activity. Affected snakes crawled about restlessly and kept their mouths partially opened. Their tongues were incompletely withdrawn into the sheaths, and their pupils were extremely dilated. Stage 3 was seen from several hours to 1 day preceding death. The mouth was kept completely open, and the snakes expelled a purulent material from the glottis. Stage 4 was seen from several minutes to 1 hr preceding death. The mouth was kept maximally opened, the pupils were dilated, and snakes were excessively active.

In an epizootic involving rock rattlesnakes, a new breeder male was introduced into an established collection without having been quarantined.[25] Ultimately, this snake was in contact with eight other rock rattlesnakes, seven of which died. On day 3 following introduction, the new snake developed head tremors and loss of equilibrium; it died on day 14. Over the next 2.5 mo, four females and three males died after manifesting clinical signs. Only one rattlesnake remained healthy and survived.

In an epizootic in a zoological park in Louisiana, clinical signs included a sudden gaping of the mouth followed within 1 day by violent convulsions, causing the entire body to spiral.[24] Frequently, there would be an expulsion of brownish fluid from the glottis. Several snakes regurgitated ingested mice or passed greenish mucoid feces from 1 to 3 wk prior to death.

Presumptive diagnosis of OPMV infection can be made upon finding characteristic light microscopic changes in the lung. The most significant lung lesion is proliferation of epithelial cells lining the airways, with or without interstitial inflammation. In some affected rattlesnakes, an enlarged pancreas with hyperplasia of acinar cells has been seen.[27]

OPMV has been isolated in a wide variety of cell types of reptilian and mammalian origin, including gecko embryo, rattlesnake fibroma, rattlesnake spleen, viper heart, and baby hamster kidney cells.[5] Application of serologic methods has been useful in determining exposure of snakes to OPMV. The hemagglutination inhibition assay has been most useful because of its relative simplicity and rapid turn-around time.[14]

In an epizootic in a zoological collection in Louisiana, snakes with initially higher titers to OPMV had significant declines within 6 mo following exposure.[27] Some snakes will maintain high titers for over 6 mo following initial testing (pers. obs.). Why some snakes maintain high titers and others do not is unknown. Snakes with high titers may serve as carriers and shedders of OPMV. Research is this area is necessary to determine the shedding potential of persistently seropositive snakes.

There is little information available about the presence of or exposure to OPMV in wild populations of snakes. In a survey of Aruba Island rattlesnakes on Aruba, all snakes evaluated were seronegative (Odum, pers. comm.). Serologic surveys should be conducted on snakes in the wild to determine the prevalence of exposure to OPMV in wild populations.

Inclusion body disease of boid snakes

This disease has been seen in a variety of snakes in the family Boidae, including boa constrictor (*Boa constrictor*), anaconda (*Eunectes murinus*), Haitian boa (*Epicrates striatus*), Burmese python (*Python molurus bivittatus*), Indian python (*P. m. molurus*), reticulated python (*P. reticulatus*), and ball python (*P. regius*).[36] In boa constrictors, the first clinical sign manifested is regurgitation of food within several days of feeding. Although some snakes die within several weeks of first manifesting illness, others may survive for months. Some of the snakes may eventually show signs of central nervous system (CNS) disease, exhibiting disorientation, head tilting, and opisthotonos. In Burmese pythons, pathologic changes are primarily seen in the CNS; several snakes

with flaccid paralysis have been seen. Regurgitation is seldom seen in Burmese pythons.

The major histologic finding, which appears characteristic of this disease, is the presence of eosinophilic intracytoplasmic inclusions within hematoxylin and eosin-stained sections of a variety of tissues. In boa constrictors, the inclusions are most prominent in the kidney, pancreas, liver, and brain. In Burmese pythons, the inclusions are mostly found within neurons in the CNS. The inclusions are generally associated with an encephalitis, which is often more severe in pythons than in boa constrictors. An RNA virus that morphologically resembles members of the family Retroviridae has been identified in tissues of affected snakes and has been grown in primary cultured kidney cells of affected snakes. Although the route of transmission is unknown, in many situations affected snakes are from collections with severe mite infestations. The snake mite, *Ophionyssus natricis,* may be involved in the transmission.

Boid inclusion body disease is extremely insidious. The incubation period prior to manifestation of clinical signs is unknown. The range of species susceptible to this virus also is unknown. No information is available on the occurrence of inclusion body disease in wild populations of snakes. Because this agent has been difficult to isolate, a serologic test has not been developed. My laboratory is focusing on developing a cell line continuously infected with this virus so that virus characterization can be accomplished and a serologic test developed. Once a test is available, a large serologic survey of snakes in zoological collections can be conducted.

Herpesvirus infections of tortoises

The first report of a herpesviruslike agent associated with a lesion in a tortoise is that of a case involving a desert tortoise.[15] A 6-yr-old cachectic desert tortoise, which had been in captivity since hatching, had a pharyngeal abscess. Histologic examination revealed intranuclear inclusions in superficial epithelial cells of the palatine mucosa. Electron microscopic examination revealed various developmental stages of a virus morphologically compatible with members of the family Herpetoviridae.

In a second report, 1,200 of 2,200 recently imported Argentine tortoises (*Geochelone chilensis*) died over a 3-mo period; red-footed tortoises (*G. carbonaria*) imported with and housed with the Argentine tortoises remained clinically healthy.[21] At necropsy, the dominant lesion was necrosis of the oral mucosa, with accumulations of necrotic cellular debris around the glottis, the roof of the oral cavity, and internal nares. Light microscopic examination revealed desquamated degenerating epithelial cells containing eosinophilic intranuclear inclusions. Electron microscopic examination revealed inclusions consisting of viral particles with an electron-dense core. Particles consistent with herpesvirus were seen enveloping from cell membranes, and mature enveloped particles of approximately 125 nm were seen in the cytoplasm. Viral isolation attempts in green sea turtle embryo fibroblasts were negative.

Of 13 spur-thighed tortoises (*Testudo graeca*) from two private colonies, herpes-like particles were detected by electron microscopy in two animals with stomatitis.[6] Initially, swabs taken from the oral lesions resulted in the isolation of a variety of microorganisms, however treatment with a number of systemic and local antibiotics had no effect on the course of the disease. Eventually, virus particles were demonstrated by electron microscopy within bronchial and palatine mucosal epithelium. Treatment of subsequent cases with 5% acyclovir ointment was described as encouraging.

In a preliminary report that described viral epidemics in pet trade Mediterranean tortoises (*Testudo* spp.) and detailed 300 case histories derived from the Tortoise Trust in England, the author concluded that a virus

was the responsible agent.[17] This conclusion was based upon findings of viral inclusion bodies in the livers of diseased tortoises in Germany. The reservoir species was considered to be the pet trade-collected Turkish *T. ibera* group. However, detailed pathologic evaluations were not reported, and until a virus is isolated and Koch's postulates fulfilled, this report can only be considered anecdotal.

In an examination of 16 Hermann's tortoises (*T. hermanni*) and eight spur-thighed tortoises with necrotizing glossitis/stomatitis, intranuclear inclusions were found in epithelial cells in the tongue, trachea, bronchi, and alveoli, in endothelial cells of capillaries of the glomeruli, and within neurons and glial cells in the medulla oblongata and diencephalon.[32] Electron microscopic examination of the liver and trachea revealed hexagonal nucleocapsids in the nuclei of hepatocytes and epithelial cells of the trachea. Enveloped virions in the cytoplasm were 110–120 nm and were morphologically consistent with herpesvirus. Imported tortoises were considered latent carriers of this virus. Stress and parasitism may have contributed to the clinical manifestation of the virus in the imported tortoises.

Mycoplasmosis of tortoises

Relatively few bacteria have been implicated as primary pathogens in tortoises. In most situations, bacteria have been reported as causative agents of disease based upon isolation from lesions, either on the body surfaces or within visceral structures. An understanding of the normal bacterial flora of the affected body system and/or structure is extremely important when interpreting culture results. In a study of a respiratory disease in desert tortoises, aerobic bacteria were isolated from the respiratory tract of both clinically healthy tortoises and tortoises showing signs of respiratory disease.[12] The results of this study failed to implicate a specific bacterial organism as a cause of the respiratory disease. In a follow-up study, when bacterial isolates of the respiratory tract of captive healthy desert tortoises were compared with isolates from free-ranging tortoises, no major differences were observed.[38] A bacterium belonging to the genus *Pasteurella* was isolated from the respiratory tract of both groups of tortoises, and species status eventually was proposed for these isolates under the name *P. testudinis* sp. nov.[37] Because this organism has been isolated from the respiratory tracts of ill and healthy desert tortoises, its significance in respiratory disease of desert tortoises is unknown.

Rhinitis (upper respiratory tract disease) has also been seen in long-term captive Mediterranean tortoises (*T. graeca* and *T. hermanni*), and a variety of organisms have been isolated from both ill (11 different organisms) and healthy (17 different organisms) tortoises.[30] As with desert tortoises, no major differences were noted. The authors proposed that if rhinitis in tortoises is of bacterial origin, those bacteria involved will be members of the normal flora acting as opportunistic pathogens.

In 1988, desert tortoises with upper respiratory tract disease (URTD) were seen in the Desert Tortoise Natural Area (DTNA), Kern County, California, USA.[23] In 1989, a detailed survey of the DTNA and nearby areas in the Rand Mountains and Freemont Valley indicated that 43% of 468 live desert tortoises encountered on the sections surveyed showed signs of this disease.[29] Additionally, carcasses of 627 tortoises were recovered from the sampled areas. Since this first outbreak in the DTNA, desert tortoises with URTD have been seen in multiple locations throughout the Mojave Desert of southern California. Desert tortoises with URTD have also been seen in Las Vegas Valley, Nevada, the Beaver Dam Slope, Utah/Arizona, and the Sonoran Desert, Arizona.

Pathologic studies of 17 ill desert tortoises from the DTNA and one ill desert tortoise from Utah indicated that major microscop-

signs and lesions, as did a juvenile scaleless Texas ratsnake (*E. obsoleta lindheimeri*) (pers. obs.). Cryptosporidiosis has also been identified in lizards[40] and tortoises.[16]

Cryptosporidium infection is insidious because the disease often goes undetected until clinical signs are dramatic. Snakes manifesting clinical signs of illness may persist from months to over 1 yr before eventually dying. As in other animals, there is no effective chemotherapy for treating reptiles ill with cryptosporidiosis.

Recent studies in a variety of wild and captive reptiles have indicated that more than one species of *Cryptosporidium* probably infects reptiles, and prolonged shedding of oocysts was found.[40] The exact route of transmission is unknown, and predisposing factors are yet to be determined. For instance, adenovirus has been seen in gastric mucosal epithelial cells of snakes with cryptosporidiosis (pers. obs.). A virus may be present first, immunocompromising the host and allowing *Cryptosporidium* to cause disease.

THE PROBLEM AND THE NEED

Reptiles are a very transportable group of vertebrates. Large numbers of reptiles are shipped around the world for use in biomedical research, as part of the pet trade, and for exhibit and captive propagation programs in zoological parks. With these animals comes a great array of potential pathogens. Although the effects of stress of shipment on the immune system of these animals during and shortly after transport have not been evaluated, a maladaptation syndrome has been reported[7] and accounts for significant mortality in recently collected reptiles. Stress-related immunosuppression may allow latent infections to become active. Mortality in reptiles is greatest within the first 6 mo of shipment (pers. obs.).

Although a number of infectious agents have been identified as significant pathogens in reptiles, far fewer reptile pathogens have been identified than exist. Because of a general lack of interest and poor funding opportunities, few reptile diseases have been studied in depth. For instance, although a URTD has been recognized in multiple species of tortoises in Europe and the USA, only recently has funding become available to conduct in-depth studies in the desert tortoise, a species whose populations north and south of the Colorado River in the southwestern USA are federally listed as threatened. Although funds from state and federal agencies have been limited for studying this disease, settlement of a law suit in Las Vegas Valley, Nevada, resulted in a large sum of money available for study of this disease in the desert tortoise. By combining the tools of biotechnology with pathologic investigations, the causative agent has been identified and an ELISA developed to determine exposure to this agent. Without major funding, the causative agent of this disease would still be unknown and a serologic test would not be available. Unconventional sources of funding are needed to carry out similar investigations on other diseases of reptiles.

The intermixing of reptiles in the pet trade and in private and zoological collections has more than likely contributed to those epizootics that have been reported in captive reptiles. The retroviruslike infection in Burmese pythons may have originated in boa constrictors, and paramyxoviral infections in viperid snakes may have originated in a nonviperid species. For those threatened/endangered reptiles being bred in captivity, and especially for those whose progeny may be returned to the wild, the breeding group should be maintained isolated from other reptiles. However, for many zoos and private collections, this may not be practical. The key for reducing the risk of occurrence of an epizootic in any collection is a sound preventive medicine program. Although the value of preventive medicine should be obvious to everyone, the most obvious is often neglected. To use an old cliche, "Bad habits are difficult to break." For most people, it takes a major epizootic to realize that improvement is needed. Unfortunately, the

value of a sound preventive medicine program is only appreciated after the occurrence of a major die-off.

A sound preventive medicine program begins with quarantine of all new animals. For captive reptiles, the health background of shipped animals often is poorly understood. In many situations, reptiles guaranteed to be healthy by the shipper actually have significant problems. Seldom is there adequate transfer of health files along with the transport, and in many situations, particularly for animals in private collections, health files do not exist.

Quarantine is necessary for both wild and captive reptiles. All new animals entering a collection should be quarantined for 90 days. Ideally, all new reptiles should enter the quarantine room at approximately the same time and should leave at approximately the same time. This schedule is seldom, if ever, followed. The quarantine room should be at a distance from the main collection. Minimally, there should be no air exchange between the quarantine room and the main collection.

The larger and more valuable a collection, the more important it becomes to quarantine all new acquisitions. New animals should be weighed upon arrival and monitored throughout the quarantine period. Fecal examinations should be performed on all new animals and appropriate parasiticides administered as needed. All animals are parasitized in the wild, and determination of whether or not a parasiticide should be administered to a parasitized animal is based partly on experience and partly on guesswork. Certain endoparasites, such as *Entamoeba invadens,* should always be considered potential pathogens, and infected animals should be medicated. Others, such as oxyurid nematodes of tortoises and iguanas, are generally considered commensals, and few reports have linked mortality with presence of these parasites. For the vast majority of endoparasites of reptiles, their role in disease is poorly documented.

Ectoparasites should be considered significant whenever encountered. Although tick infestations are for the most part relatively easy to control, mite infestations are more problematic. The snake mite *Ophionyssus natricis* continues to be a significant pest in reptile collections. Control of this parasite is difficult because of its apparent resistance to various mitacides. Once established in a large collection of reptiles, this parasite may be impossible to eradicate. It is far easier to eliminate this parasite from individuals in quarantine than from affected animals and their environment in the main collection. For every mite on the snake, there may be a dozen within the cage.

When possible, new animals entering the quarantine room should be given a physical examination.[20] This examination may not be practical for small reptiles or those species easily stressed by manipulation. At times, the animal should acclimate for a period of time before a physical examination is conducted. In zoological collections, protocols should be developed between the reptile department and animal health department for evaluating new acquisitions. Minimally, weights should be obtained on all new acquisitions and the animal given a cursory visual inspection.

Reptiles should be monitored throughout the quarantine period, and both anatomical and behavioral abnormalities should be noted. Often behavioral problems are noted prior to the development of clinical signs of illness. Although some diseases, such as inclusion body disease of boid snakes, result in rather dramatic signs in the terminal stages of the disease, the incubation period is unknown, and clinical signs in the early stages of this disease may not be appreciated. Diseases such as cryptosporidiosis are also extremely insidious and go unnoticed until pathologic changes are extremely severe. With many diseases, the animal is found dead in its cage in the morning, having appeared clinically normal the day before. Keen observation is extremely important in identifying disease problems while the animal is still treatable.

As in other vertebrates, complete blood counts and plasma/serum biochemical determinations may provide valuable information on the health status of an animal. However, blood samples can be obtained rapidly and safely in some species but not in others. Because normal blood values are available for only a few of the 6,000 species of reptiles, each collection should establish its own data base. Protocols should be established and standardized for collecting blood and other biomedical samples. Blood collection techniques vary widely among investigators and among species of reptiles. Once collected, blood is often mishandled, resulting in spurious data.[28] Additionally, plasma/serum banks should be established for all major collections because these samples will become valuable in retrospective seroepidemiologic studies. For instance, exposure to ophidian paramyxovirus can be determined by a hemagglutination inhibition serologic assay. As more assays for exposure to various pathogens are developed for reptiles, the value of plasma/serum banks becomes more apparent.

Health assessment of animals prior to release from the quarantine room to the main collection is extremely important; all animals must be well scrutinized. Animals that are doing poorly, i.e., those that are not feeding or that have lost a moderate amount of weight, should not be released into the main collection. An ill animal should never make it out of quarantine.

All animals that die in quarantine should be necropsied. Necropsy is the key in determining causes of mortality. Dead animals should be refrigerated if a necropsy cannot be performed immediately. Freezing results in artifactual change in tissues and should be avoided. Tissues from all major organ systems, not just those tissues with gross lesions, should be collected and fixed in neutral buffered 10% formalin. Zoological collections should utilize commercial, state, or university diagnostic laboratories for histopathologic evaluation of collected tissues. A complete history and description of gross findings should go along with the tissues to the laboratory. Unfortunately, the vast majority of pathologic findings and other biomedical information on necropsied reptiles seldom make it into the literature. Zoological parks and pathology laboratories have become repositories of large amounts of important information that has been sequestered away in file cabinets. All major zoological parks should have either on site or access to an ultrafreezer for long-term storage of frozen tissues and plasma/serum.

Relocation, repatriation, and translocation programs have become quite popular. Although perceived as a potentially valuable conservation strategy, few reptile RRT programs have been successful or the outcomes have not been properly monitored to determine success or failure.[9] Although the Kemp's ridley (*Lepidochelys kempi*) and other sea turtles have been given a head start and released into the wild, the success of any of these programs is, at best, questionable.[31,39] Beyond such issues as effects of released animals on genetics and social structure of populations and the need for a better understanding of the biological requirements of the species to be released, disease is an issue that is becoming increasingly more important. Twenty years ago, we knew little about pathogens in reptiles. Today, we realize that these animals can harbor an array of pathogens. Unfortunately, our ability to screen reptiles for those few pathogens that we now know to be significant is crude at best. Protocols must be developed for each species intended for release so that the health of animals can be assessed properly and, where applicable, appropriate diagnostic tests can be performed prior to release. The release of infected animals into smaller insular populations could be devastating, as was the case for the black-footed ferret (*Mustela nigripes*) infected with canine distemper virus. State and federal agencies in charge of protecting endangered/threatened species must become more involved in these issues.

Reptiles being bred in captivity for release to the wild must be quarantined in a facility isolated from other quarantined reptiles. Also, the breeding facility must be isolated from the main collection to ensure that pathogens from a reservoir species will not infiltrate the breeding program. Breeding programs for reptiles should be established within the geographic range of the species, under as natural conditions as possible.

Health status is one of a number of items to be considered in selecting animals for a breeding program and in determining which animals are going to be released to the wild. Animals entering the breeding program should be given as thorough an examination as possible and practical. Animals with an infectious disease should never be allowed into the program. Even those with metabolic and congenital diseases should be excluded. There should be no exchange of equipment or containers between animals in the breeding program and those in the main collection. Caretakers involved with the breeding program should not have had immediate contact with the main collection. Fomites containing pathogens can be carried on clothing and may be transferred from site to site. A dressing room should be adjacent to the breeding program site so that caretakers can cleanse themselves before and after entering the premises.

All animals that die in the breeding program must be given a complete necropsy. Causes of mortality in neonate reptiles are poorly understood and should be studied more carefully.

Exchange of information among various breeding programs is essential and will necessitate standardization of the methods of collecting and storing information. Data should be computerized in a way that allows ease of access and distribution. Formal networks should be established to facilitate this exchange of information. Network exchange is far superior to rumor, which up to now has been one of the major pathways for the distribution of information in the reptile community.

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