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CAUSES OF MORTALITY AND DISEASES IN TORTOISES: A REVIEW

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Abstract: Most of the 40 species of tortoises are experiencing population declines. Of the various causes of mortality in wild populations of tortoises, the interactions of disease and population dynamics are least understood. Although habitat degradation is considered the most significant threat to wild populations of tortoises, disease is being observed more frequently in certain populations. An upper respiratory tract disease has been seen in populations of desert tortoise, *Gopherus agassizi,* in the Mojave Desert, USA, and certain populations of the gopher tortoise, *Gopherus polyphemus,* in Florida, USA. Much more information is available on diseases of captive tortoises than on those of wild tortoises. Of infectious diseases, viral, bacterial, mycotic, and parasitic diseases have all been reported. Noninfectious diseases identified in tortoises include various nutritional diseases, hypothyroidism, and neoplasia. Virtually nothing is known about the effects of pollutants/toxicants in individual or populations of tortoises.

Key words: Tortoise, mortality, disease.

INTRODUCTION

Of the 40 extant species of tortoises, most, if not all, are experiencing population declines. Although collection of tortoises for the pet trade and use by local human populations as food items in many areas of the world have contributed, habitat degradation and destruction of the environment probably accounts for the most significant worldwide declines. Unfortunately, except for a handful of species such as the desert tortoise (*Gopherus agassizi*), Bolson tortoise (*Gopherus flavomarginatus*), and Hermann's tortoise (*Testudo hermanni*), there is generally a lack of detailed ecological research on most species.[77] An understanding of life histories becomes extremely important when developing conservation programs. Of all the life history information relevant to structure of tortoise populations, the effects of infectious agents and disease on wild populations have been least studied.

In attempts to conserve several species of tortoises, such as the Galapagos tortoise (*Geochelone elephantopus*) and Bolson tortoise, breeding and rearing facilities have been created. Many other species are being captive bred and reared by the private sector for sale in the pet trade. Although a number of breeding programs have been in existence for over 20 yr, little information is available on causes of illness and mortality in captive-reared tortoises. Because health assessment is an issue that is only recently being discussed with regard to translocation and reintroduction of captive animals into the wild, obviously health assessment will be difficult to perform without good baseline normative biomedical data. Because health and disease are directly related, one cannot be understood without appreciating the other.

In this paper, causes of mortality and diseases, including both infectious and noninfectious diseases, in captive and wild tortoises will be reviewed. Most of the information available was gathered from captive tortoises, of which many were pets. Relatively little information is available on causes of disease in wild tortoises.

CAUSES OF MORTALITY

Few thorough retrospective or prospective studies have been conducted on causes of mortality in either free-ranging or captive tortoises. In the wild, by the time a dead tortoise is found, generally all that remains are the hard parts. Virtually nothing is known about causes of mortality in neo-

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nates, a life stage that is only beginning to be studied in detail.[1]

Studies conducted on the Mexican Bolson tortoise demonstrated that mortality was apparently very high in the nest and hatchling stages; it was estimated that only one out of eight nests in the field was unpredated, and about 67% of hatchlings died in the first year.[1] Between 1980 and 1985, only two out of 27 hatchling and juvenile Bolson tortoises matured and were located within the study colony. Five animals were found dead, of which three appeared dehydrated. Of hatchlings and 1–4 yr olds, 67% and 69%, respectively, in this study died within 12 mo of release.

Of 86 Bolson tortoises hatched since 1983 from eggs incubated at the Laboratorio de Desierto, Man and Biopshere Mapimi Reserve, Durango, Mexico, overall survival was 76% (65/86).[2] Age-specific survival rates were 1 yr olds, 86% (48/56); 2 yr olds, 55% (11/20); and 3 yr olds, 60% (6/10). Mortality was analyzed in 51 hatchlings, and specific causes of death were divided into the following six categories: 1) sun, 2) cold, 3) decalcification, 4) desiccation, 5) drowning, and 6) undetermined. The most commonly assigned mortality category was "undetermined" (7/21). No necropsy or pathologic findings were given.

Constantly high or fluctuating mortality appears characteristic of immature chelonians in general.[1] Mortality between the embryonic stage and 1 yr of age in the gopher tortoise, *Gopherus polyphemus,* was estimated at 94.2%.[4] In another study with gopher tortoises in southwestern Georgia, USA, the annual birth rate (number of young born alive) was only a small portion of the fecundity, due primarily to nest predation. In this study, an average of only one successful clutch per 9.5 nests was found.[83]

A considerable body of information is available on population declines of the desert tortoise in the western Mojave Desert, USA.[11–13] The following is a list of those problems indicative of downward trends in these populations: 1) declines in densities, 2) declines in numbers registered during a 60-day survey, 3) declines in proportions of juveniles and small immature tortoises, 4) reduction in recruitment of young individuals into the adult population, 5) abnormally high mortality rates for the breeding population, 6) abnormally high mortality rates for juveniles, 7) human-induced sources of mortality, and 8) deterioration of habitat. In the Desert Tortoise Natural Area (DTNA), Kern County, California, densities of all age classes have declined significantly since 1979. In 1979, there were 149 tortoises/km^2, but in 1985 there were 75 tortoises/km^2, representing a loss of 50%. The vast majority of the losses have occurred in the young tortoises, from hatchling size to those approaching sexual maturity. Causes of death for 54 tortoises at the DTNA interpretive center plot between mid-1981 and mid-1985 included 1) gunshot or vehicle (18%), 2) vandalism (1.9%), 3) overturned by sheep (1.9%), 4) predation by ravens (29.6%), and 5) unknown (48.1%). In 1988, desert tortoises with signs of an upper respiratory tract disease were found in the DTNA, and subsequently this disease was seen in other locations in the Mojave Desert.[69]

Since 1975, the desert tortoise population on the Beaver Dam Slope, Arizona and Utah, USA, has experienced a high mortality rate. During the decade preceding 1987, the Arizona Strip District Office of the Bureau of Land Management (BLM) reported 217 tortoise carcass remains within the Virgin River–Pakoon Basin Habitat Management Area.[74] An analysis of skeletal remains (shell bone) of 24 carcasses examined on 5 and 6 August 1987 and 10 of 80 carcasses examined on 9 and 10 August 1987, indicated that the most frequently encountered lesion was osteopenia, which included thinning of trabeculae of the peripherals and lateral areas of the hypoplastron, producing a pronounced spongy appearance. In all, 14 carcasses from Utah and 13 carcasses from Arizona displayed obvious signs of shell bone thinning. Malnutrition

was considered responsible for the osteologic lesions observed in desert tortoises from the Beaver Dam Slope. Nutrient availability had declined on the Beaver Dam Slope since the studies in 1948[117] and was considered a likely cause of recent desert tortoise mortality on the Beaver Dam Slope.

Declines of desert tortoises on the Chuckwalla Bench Area of Critical Concern, Riverside County, California, USA, have occurred over the last 10 yr and are associated with the presence of cutaneous dyskeratosis in all age classes of tortoises.[70] The disease was present in 1979 when tortoises on a permanent study site were first photographed. The lesion commenced at seams between adjacent scutes and spread toward the middle of each scute in an irregular pattern. The histologic appearance of the lesion was compatible with a dyskeratosis and was suggestive of either a nutritional deficiency disease or toxicosis. However, the exact cause of the disease is yet to be determined.

As with wild tortoises, there are few detailed reports on causes of mortality in collections of captive tortoises. Most of the reports in the literature are documentations of specific disease problems in recently imported or pet tortoises. Causes of mortality were reported for 84 desert tortoises and two Texas tortoises (*Gopherus berlandieri*).[102] Of the organs evaluated, more lesions were seen in the liver (72.6%) than in the lungs (53.8%), intestine (50.7%), kidney (40.6%), and heart (34.3%). Bacterial hepatitis accounted for 22 of 49 liver diseases noted; nine cases of interstitial nephritis were seen.

Probably the best account of causes of mortality in captive tortoises is that based upon the postmortem examination of 144 tortoises representing 17 species from 1 January 1965 to 31 December 1975 at the Zoological Society of London.[76] Although several of these tortoises were known to be recently imported, the majority were housed in the London Zoo (77.8%). Of the tortoises examined, 2.2% were pets submitted for necropsy by veterinary surgeons in practice. In this survey, various forms of enteritis were common. Nutritional diseases, particularly osteodystrophies, were commonly found. With the exception of a few nematodes, parasites were relatively unimportant; the vast majority were considered nonpathogenic. Only six cases (4.2%) of respiratory disease were seen and were less important than seen in other necropsy studies involving chelonians.[62]

INFECTIOUS DISEASES

Viruses

Herpetoviridae: The first report of a herpesvirus-like agent associated with a lesion in a tortoise is that of a case involving a desert tortoise.[55] A 6-yr-old cachectic desert tortoise that had been in captivity since hatching was found to have 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 (*Geochelone carbonaria*) imported with the Argentine tortoises and housed together remained clinically healthy.[67] At necropsy, necrosis of the oral mucosa with accumulations of necrotic cellular debris around the glottis, the roof of the oral cavity, and internal nares was seen. Light microscopic examination revealed desquamated degenerating epithelial cells that contained eosinophilic intranuclear inclusions. Electron microscopic examination revealed that the inclusions consisted of viral particles containing 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. Virus isolation attempts in green sea turtle embryo fibroblasts were negative.

Of 13 spur-thighed tortoises (*Testudo graeca*) from two private colonies, herpesvirus-like particles were detected by electron microscopy in two animals with stomatitis.[26] Initially, treatment with a number of systemic and local antibiotics had no effect on the course of the disease, although swabs taken from the oral lesions resulted in the isolation of a variety of microorganisms. 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 describing viral epidemics in pet trade Mediterranean tortoises (*Testudo* spp.) and detailing 300 case histories derived from the Tortoise Trust, England, the author concluded that a virus was the responsible agent[58] based upon findings in Germany of viral inclusion bodies in the livers of diseased tortoises. The viral organisms were identified in the report as RNA forms and were associated with immune system deficiencies. The reservoir species was considered to be the pet trade collected Turkish *Testudo ibera* group. However, detailed pathologic evaluations were not reported, and a causal relationship between the identified virus and the epidemics awaits virus isolation and fulfillment of Koch's postulates.

In 16 Hermann's tortoises and eight spur-thighed tortoises with necrotizing glossitis/stomatitis, intranuclear inclusions were found in epithelial cells in the tongue, trachea, bronchi, alveoli, endothelial cells of capillaries of the glomeruli, and within neurons and glial cells in the medulla oblongata and diencephalon.[94] Electron microscopic examination of the liver and trachea demonstrated 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. The authors considered imported tortoises to be latent carriers of this virus. Stress and parasitism may have contributed to the clinical manifestation of the virus in the imported tortoises. Electron microscopic examination has revealed herpesvirus-like particles in the intestinal contents of a Hermann's tortoise, several of which had caseous material in the upper digestive tract, hepatomegaly, and enteritis.[84]

Iridoviridae: A Hermann's tortoise was found at necropsy to have multiple gray spots disseminated throughout the liver; the spleen was congested, and small white foci were seen on the cut surface.[57] Histopathologic examination revealed multiple areas of hepatic necrosis, and hepatocytes adjacent to necrotic areas contained strongly basophilic intracytoplasmic inclusions; inclusions were also seen in mucosal epithelial cells of the intestine. Electron microscopic examination revealed intracytoplasmic inclusions that consisted of accumulations of hexagonal virus particles located around electron-dense material. A few free virions were also observed in nuclei. The virions were composed of an electron-dense nucleocapsid enclosed by a hexagonal envelope containing two or more distinct layers. Mean virus diameter was 140–160 nm. Based upon size and morphologic properties, the agent was considered to be an iridovirus.

Poxviridae: Although a desert tortoise was considered to have poxvirus infection because intracytoplasmic inclusions were seen in epidermal cells, no ultrastructural findings were presented to confirm this interpretation.[41]

Togaviridae: Field investigations into the ecology of Venezuelan encephalitis in south Texas during the summer and fall of 1971 led to the isolation of two strains of western equine encephalitis (WEE) from the blood of Texas tortoises.[112] In a subsequent study, subcutaneous inoculation of 34 Texas tortoises with either of two strains of WEE virus resulted in viremia that lasted up to 105 days.[17] Environmental temperature had marked effects on the length of the previremia period, the maximum viremia level

attained, and the duration of viremia. Temperatures of 30°C shortened the previremia period and duration of viremia and elevated the maximum viremia level. Lower temperatures had the opposite effect. No disease was reported in any of the tortoises inoculated, which suggested that Texas tortoises could serve as an overwintering reservoir for WEE virus.

Bacteria

Relatively few bacteria have been implicated as primary pathogens in tortoises. In most situations, bacteria have been reported as causative agents of disease simply based upon isolation from lesions, either on the body surfaces or within visceral structures. An understanding of the normal bacteria flora of the affected body system and/or structure becomes extremely important when interpreting culture results. In a study of respiratory disease in desert tortoises, bacteria were isolated from the respiratory tracts of both clinically healthy tortoises and tortoises showing signs of a respiratory disease.[34] The results of this study failed to implicate a specific bacterial organism as a cause of respiratory disease in the studied tortoises. In a follow-up study, when bacterial isolates from the respiratory tract of captive healthy desert tortoises were compared with bacteria from free-ranging tortoises, again no major differences were observed.[108] A bacterium belonging to the genus *Pasteurella* was isolated from the respiratory tract of both groups of tortoises, and eventually species status was proposed for these isolates under the name *Pasteurella testudinis* sp. n.[107] 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 spur-thighed and Hermann's tortoises, and a variety of organisms have been isolated from both ill (11 different organisms) and healthy (17 different organisms) tortoises.[87] As with desert tortoises, no major differences were noted. Some investigators have suspected that a virus is the cause of this disease.[66] Although not isolated at the time from tortoises with rhinitis, *Mycoplasma* was mentioned as a suspect organism.[87]

In 1988, desert tortoises with upper respiratory tract disease (URTD) were seen in the Desert Tortoise Natural Area, Kern County, California.[69] 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.[79] Additionally, carcasses of 627 tortoises were recovered from the sampled areas. Since first being seen in desert tortoises in the DTNA, URTD has been seen in desert tortoises in multiple locations throughout the Mojave Desert of southern California. Desert tortoises with URTD have also been seen in the 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 microscopic lesions were confined to the upper respiratory tract (URT) of ill tortoises.[69] Electron microscopic studies revealed small (350–900 nm) pleomorphic organisms resembling *Mycoplasma* in close association with the surface epithelium of the URT of ill tortoises. *Pasteurella testudinis* was cultured from the nasal cavity of all ill tortoises and of one of four healthy tortoises. A *Mycoplasma* was cultured from the nasal passageways of four ill tortoises and was ultrastructurally similar to the pleomorphic organism present on the mucosa in tissue sections. This is only the second report of isolation of a mycoplasma from a reptile. *Mycoplasma testudinis* was isolated from the cloaca of a spur-thighed tortoise.[59] Because many captive desert tortoises ill with respiratory tract disease exist in private collections throughout southern California, an extremely pathogenic organism may have

been introduced into wild populations at multiple sites by released pet tortoises. Other predisposing factors such as habitat degradation and drought may also be contributing to the severity and spread of this disease.

Of bacterial infections of the gastrointestinal system, salmonellosis has been reported by several authors.[18,89,120] *Salmonella* has been recovered from wild *T. hermanni* in Bulgaria.[93] *Arizona,* which is now considered a species of *Salmonella,* was found to be excreted by 10.5% of 691 tortoises from southeast and southwest Bulgaria.[28] Forty-six of the tortoises that were positive for *Arizona* were also positive for *Salmonella.* In an examination of reptiles that originated from zoological gardens, 29 *Salmonella* strains and 15 *Arizona* strains were isolated.[122] In Germany, the feces of 125 clinically healthy tortoises were cultured, and 28 different *Salmonella* spp. were cultured from 55 tortoises (44%).[115] In a survey of acclimatized tortoises in England, only 2.8% were infected.[76] In this study, *Salmonella* infection was found to be the cause of death in only two tortoises. *Salmonella* infection was also reported as the cause of death of two Galapagos tortoises at the San Diego Zoo.[89] One tortoise had a massive infection of *S. newport,* which involved the liver, lungs, intestine, and spleen. The other died from a *S. sandiego* infection, which was recovered from abdominal fluid, intestine, and urinary bladder.

Few primary bacterial infections of the integument have been described in tortoises. Sloughing of the horny shell plates, with local bacterial infection, occurs in tortoises that are kept under unhygienic conditions.[40] Without reporting details, Rosskopf mentioned that there are many infectious organisms that may be associated with shell disease, most of which result from poor husbandry practices.[101] A red-footed tortoise with *Streptococcus* dermatitis and a Burmese brown tortoise (*Manouria emys*) with a large caseous bacterial abscess involving a forelimb have been seen (pers. obs.). In another case, a Burmese brown tortoise with a focal ulcerative shell lesion on the plastron died and was found on necropsy to have a fistulous tract extending from the shell lesion to a bacterial abscess in the coelomic cavity (pers. obs.).

I evaluated skin biopsies of four captive desert tortoises in a private collection in Arizona. These tortoises had developed hyperkeratotic skin lesions at multiple soft tissue sites. Light microscopic examination revealed a filamentous organism resembling *Dermatophilus.* However, microbial isolation attempts were negative and the identity of this organism remains unknown.

Diseases of the middle ear have been seen in tortoises by several investigators.[50,76] Spur-thighed tortoises have been seen with the auditory canal blocked with a thick inspissated material. This problem has been seen in hibernating tortoises. As the canal fills, the tympanic scale may bulge. A variety of gram-negative microorganisms have been cultured from these lesions.

Fungi

Various fungal diseases have been reported in tortoises in zoological collections worldwide. In a review of over 200 necropsies and the published literature of the chelonians necropsied, 3% of the deaths were due to mycotic pulmonary disease; terrestrial species were more commonly affected than aquatic forms.[62] Mycotic infections are often associated with predisposing factors, including high humidity, malnutrition, overcrowding, and debris buildup in the animal's environment. Low environmental temperature has been considered a predisposing factor in many cases. No reports could be found on mycotic disease in wild tortoises.

There are few documented reports of fungal infection of the integument of tortoises. *Fusarium* was isolated from under the scales of a radiated tortoise (*Geochelone radiata*).[36] Colonization of the keratin layer of the shell by fungi often follows previous trauma and in many situations may not rep-

resent primary invasion.[8] A generalized case of chromomycosis involving the lower jaw and multiple visceral organs in another radiated tortoise also has been described.[37] A large cervical subcutaneous mass found in an adult leopard tortoise (*Geochelone pardalis*) was composed of multiple granulomas containing branching septate hyphae (pers. obs.). The infection more than likely developed following a traumatic penetrating wound through the overlying skin.

There are several reports of mycotic disease in Galapagos and Aldabra tortoises (*Geochelone gigantea*). Fatal pneumonitis caused by *Aspergillus amstelodami* was reported in a Galapagos tortoise that lived at the Chicago Zoological Park for over 30 years.[47] In this study, both *Geotrichum candidum* and *A. amstelodami* were isolated from another Galapagos tortoise with pulmonary disease. Another fungus, *Beauvaria,* was isolated from a Galapagos tortoise with pulmonary abscesses. A severe generalized case of penicilliosis was also described in a Galapagos tortoise.[52] Multiple lesions were found in the lungs, stomach, liver, and pancreas, from which *Penicillium* was cultured.

Paecilomyces fumoso-roseus was cultured from pulmonary abscesses in an Aldabra tortoise that had been in captivity at the Chicago Zoological Park for about 5 mo prior to death.[47] From the Copenhagen Zoo, a fatal case of aspergillosis of the lungs was seen in an Aldabra tortoise that had been ill for over 1 year.[5] *Basidiobolus ranarum* was found in a granulomatous lesion located in the mouth of an Aldabra tortoise.[16] An 80-yr-old, 202-kg male Aldabra tortoise with a history of anorexia and progressive lethargy died, and necropsy revealed numerous randomly scattered yellow nodules throughout the omentum, oral and gastric mucosa, and liver.[56] Microscopically, all lesions consisted of granulomatous inflammation with branching, septate fungal hyphae; *Paecilomyces lilacinus* was cultured from the liver.

In a yellow-foot tortoise (*Geochelone denticulata*) dying of pneumonia at the Rotterdam Zoo, *Penicillium lilacinum* was isolated from the lung.[10] In a radiated tortoise with chromomycoses, dark-brown hyphae were found in the lower jaw, liver, spleen, lungs, pancreas, tongue, and thyroid glands.[37] The fungus was not isolated.

Candida tropicalis infection was reported in a spur-thighed tortoise.[121] This animal had a complete loss of appetite and exhibited signs of respiratory disease, including dyspnea and bubbling from the nose. Fecal analysis demonstrated yeast and short hyphal structures. *Candida tropicalis* was isolated from feces, and oral therapy with nystatin eliminated the yeast infection.

Parasites

Tortoises are hosts to numerous species of protozoan and metazoan parasites. Flagellates include *Retortomonas testudae* in a Chaco tortoise (*Testudo argentina*)[6] and *R. cheloni* and *Monocercomonoides filamentum* in Indian star tortoises (*Geochelone elegans*).[71,72] The ciliates *Nyctotherus kyphodes, N. teleascus,* and *Balantidium testudinis* and the amoeba *Entamoeba insolita* have been identified in the intestinal tract of Galapagos tortoises.[46] Other amoebae identified in tortoises are *E. testudinis* in Mediterranean tortoises and *Hartmanella* spp. in radiated tortoises and Aldabra tortoises.[38] Coccidia are only poorly known in tortoises and include *Eimeria paynei* in gopher tortoises,[31] *E. brodeni* in Mediterranean tortoises,[21] and *Toxoplasma gondii* in Horsfield's tortoises (*Testudo horsfieldi*).[88] *Sarcocystis*-like parasites have been seen in skeletal muscles of a Jaboty (yellow-foot) tortoise,[76] and cysts of unidentified sarcosporidia have been seen in muscles of Mediterranean tortoises.[91] Hemoparasites include *Haemogregarina* sp. in an angulated tortoise (*T. angulata*)[90] and *H. testudinis* in Burmese brown tortoises.[86] *Haemoproteus testudinis* has been identified in leopard tortoises,[85] and *H. caucasica* has been identified in Mediterranean tortoises.[82] The only hemoparasite in a tortoise for which the entire life cycle has been elucidated is *Hepa-*

tozoon mauritanica in Mediterranean tortoises.[92]

No acanthocephalans (spiny-headed worms) and only a few cestodes and trematodes have been identified in tortoises. The cestode *Ophiotaenia lopesi* was identified in the yellow-footed tortoise,[98] and the trematode *Telorchis aculeatus* has been found in the Mediterranean tortoise.[119] Numerous species of ascarid, atractid, and oxyurid nematodes have been identified in the intestinal tract of tortoises. In most cases, nematodes appear to be nonpathogenic, even in heavy infections.[76] Oxyurid nematodes are particularly common in tortoises and are an example of niche diversification in a parasitic species flock.[104] For the most part, oxyurids have developed a commensal relationship with their host. In a study of oxyurids in the gopher tortoise, Bolson tortoise, and desert tortoise, the following 15 species and subspecies were identifed: *Tachygometria macrolaimus tetrapapillata, T. dentata nearctica, Thaparia macrocephala, Thaparia microcephala, Alaeuris mazzottii, A. paramazzottii, A. gopheri gopheri, A. gopheri pudica, A. gopheri macrolabiata, A. caballeroi, A. kinsellai kinsellai, A. kinsellai sonorae, A. longicollis, Oxyuris* sp., and *Gopheruris aspicula.*[96]

Ectoparasites of tortoises include the mite *Eutrombicula alfreddugesi,* various species of ticks, and larvae of the dipteran fly *Cistudinomyia cistudinis.* Of 178 Texas tortoises, one was found to be heavily infested with the trombiculid mite *Eutrombicula alfreddugesi.*[48] This mite is also commonly seen on the gopher tortoise in the southeastern United States.[116] An unidentified trombiculid mite was seen on a desert tortoise in southwestern Utah.[22,24]

Ticks identified on tortoises include *Amblyomma tuberculatum* from gopher tortoises,[20,25,61] *A. clypsolatum* from Indian star tortoises,[105] *A. testudinarium* from Burmese brown tortoises,[60] *Hyalomma aegyptium* from Hermann's tortoises,[106] and *H. detritum* from Horsfield's tortoises.[75]

The most frequently found ectoparasites of desert tortoises are the argasid ticks, *Ornithodoros parkeri* and *O. turicata.*[54,103,117] *Ornithodoros parkeri* also is found on the gopher tortoise in Florida.[20] However, there is confusion in the literature regarding precise identification at the specific level, and in a recent report only *O. parkeri* appears to parasitize free-living desert tortoises in California.[51]

In the following section only those parasitic infections known to cause or be associated with illness in tortoises will be described.

Protozoa: Entamoeba invadens, the causative agent of enterohepatitis of captive lizards and snakes, was for a long time believed to be of minor importance as a pathogen of chelonians. However, an epizootic of amebiasis was reported in red-footed tortoises[68] and several cases have involved leopard tortoises (pers. obs.). Of 500 red-footed tortoises imported to southern Florida, approximately 200 died during a 2-mo period. Clinical signs were nonspecific and included anorexia, listlessness, and watery diarrhea. Necropsy consistently revealed a thickened duodenum, with necrotic mucosa and multifocal to diffuse areas of hepatic necrosis. Histologic evaluation of tissues demonstrated numerous amoebae in intestinal and hepatic lesions. Predisposing conditions probably were involved. Tortoises were imported during the winter months and were more than likely exposed to temperatures below that for optimal growth of the amoebae. Additionally, tortoises were feeding minimally during this period. Without food, particularly carbohydrates, in the tortoise gastrointestinal tract, encystment of amoebae would not be favored. Thus, once temperatures reached that for optimal growth for the parasite, the tortoises would be predisposed to tissue invasion by trophozoites. This appears to have happened during March and April when the tortoises died.

Hexamita parva was diagnosed in nine chelonians, including Horsfield's tortoise, Tafrail tortoise (*Testudo marginata*), Indian

star tortoise, and red-footed tortoise.[123] Hexamites were seen primarily in the kidneys, within collecting ducts and in tubules showing acute and subacute lesions and inflammatory reactions, and in the liver, within bile ducts. The disease was slowly progressive, finally leading to death mainly due to nephritis.

Nematodes: Although the majority of helminths encountered in 144 tortoise necropsies were considered nonpathogenic, in several cases the helminth infection resulted in pathologic changes.[76] In one case, oxyurid nematodes of the genus *Tachygometria* were considered pathogenic in a Hermann's tortoise. Migrating larval forms of the common roundworm *Angusticaecum* spp. may have been responsible for pulmonary lesions in at least one spur-thighed tortoise.

The major ascarid of tortoises is *Angusticaecum holopterum,* which occurs in many species of *Testudo* and the African hinge-back, *Kinixys belliana.*[109] They are large nematodes and occur in the stomach, small intestine, and occasionally the large intestine. They have been reported to cause blockage in the colon.[76] Infection is direct, occurring by ingestion of embryonated eggs with the food. The larvae move to the lungs and eventually migrate to the esophagus and stomach.

Between September 1982 and January 1984, verminous colitis was diagnosed during postmortem examination of eight red-footed tortoises and three leopard tortoises at the National Zoological Park, Washington, D.C.[100] The nematode was determined to be a viviparous pinwormlike nematode of the genus *Proatractis* (family Atractidae). Clinical signs were nonspecific and included anorexia, lethargy, and depression. Pathologic findings were roughening and thickening of the mucosa of the cecum and colon, and in severe cases a myriad of tiny (0.5–1.0 cm) nematodes were evident on the mucosal surface. Unlike most pinworms, proatractids are viviparous and the larvae are capable of completing the life cycle within the same host. If the tortoise is stressed or debilitated, massive infections and increased invasiveness apparently result. In this outbreak, introduction of wild-caught tortoises into the collection and overcrowding were probably contributory.

Diptera: Larval stages of the dipteran fly *Cistudinomyia cistudinis* frequently parasitize box turtles (*Terrapene carolina*) and gopher tortoises in the southeastern United States. This parasite was first described as *Sarcophaga cistudinis*[3] and was subsequently renamed *Cistudinomyia.*[113] Adult flies lay larvae directly on the host;[78] however, larvae are unable to penetrate intact skin, requiring breaks in the integument to gain access to subcutaneous sites. The larvae can cause significant tissue damage and death. In Florida, five Aldabra tortoises in outdoor enclosures were found parasitized by larvae of this dipteran, with lesions located primarily on the perineum and caudal aspects of the hind legs.[111] Following removal of larvae and cleaning of the cavity with dilute povidone–iodine solution, the lesions healed in approximately 5 weeks.

NONINFECTIOUS DISEASES

Metabolic bone disease

Metabolic bone disease encompasses a variety of pathologic conditions that develop as a result of 1) prolonged deficiencies of calcium; 2) deficiencies in phosphorus; 3) improper ratios of calcium to phosphorus in the diet; and 4) vitamin D deficiency. The exact vitamin D analogue in tortoises, necessary for uptake of calcium and ossification of the shell, is unknown. Because the majority of the tortoise skeleton is incorporated into the shell, metabolic bone disease often is first exhibited in this structure.

Nutrition of tortoises is an art rather than a science. Although several good papers exist on digestive physiology of tortoises[14,15] and information is available on food preferences of some species in the wild,[19,23,53] little is known about nutritional requirements of tortoises. Because of this, most

diets of tortoises have been empirically formulated.

In the recent past, diets of tortoises have consisted of those commercially available fruits and vegetables that are inexpensive and easy to procure. Foods such as iceberg lettuce, tomatoes, melons, and cucumbers made up the bulk of the diet of many captive tortoises. When natural forages are compared with commercially available produce, marked differences in protein, mineral, and energy content are found.[33] These findings were supported in a recent review.[73] Low dietary crude fiber levels have been implicated in the pathogenesis of bacterial and cloacal infections of tortoises.[9] Calcium deficiency disease is common in herbivorous tortoises and may be prevented by offering tortoises the following foods: natural grasses, alfalfa hay, alfalfa sprouts, clover, guinea pig diets, rabbit diets, dandelion, and high-protein supplements such as low-fat dry dog food.[101]

The common occurrence of metabolic bone disease resulting from diets low in calcium and/or vitamin D and high in phosphorus is well documented in chelonians.[41,65,101] In a review of necropsy cases involving tortoises, 12 of 144 were reported to have nutritional osteodystrophies.[76] Nutritional osteodystrophies resembling rickets were found in the young and osteomalacia in the older tortoises. Other cases of nutritional osteodystrophy have also been reported in tortoises.[64]

In carcasses of desert tortoises from the Beaver Dam Slope, Arizona and Utah, osteoporosis (osteopenia) was the most frequently encountered lesion.[74] In all, 14 carcasses from Utah and 13 carcasses from Arizona displayed obvious signs of shell bone thinning. However, no histomorphometric studies were performed to define the nature of the bone thinning.

In an attempt to better understand the shell disease in tortoises on the Beaver Dam Slope, histomorphometric characteristics of the carapace of tortoises on the Beaver Dam Slope were compared with those of an apparently healthy, well-nourished population at City Creek, Utah.[118] Fifteen biopsies were collected from the allegedly malnourished adult tortoises of the Beaver Dam Slope and were compared with 17 biopsies from outwardly healthy adult tortoises of the City Creek population. The following measurements were performed on each shell biopsy: 1) shell thickness (mm), 2) shell porosity (%), 3) osteoid surface (%), and 4) osteoid seam width (mm). Results of this study indicated that shell thickness and porosity were nearly identical in the two populations. However, the desert tortoises of the Beaver Dam Slope population exhibited a two-fold increase in osteoid surface relative to desert tortoises of the City Creek population. Because measures such as osteoid surface are known to increase in osteomalacia, these findings suggested the occurrence of a mild osteomalacia in desert tortoises of the Beaver Dam Slope population. However, because there was no accompanying increase in osteoid seam thickness, the observed osteomalacia was interpreted to be mild.

Vitamin A deficiency

Vitamin A deficiency is a commonly encountered deficiency disease of chelonians.[30] Characteristic histologic changes included squamous metaplasia of multiple epithelial structures. The Harderian and lachrymal glands of the orbit are particularly prone to these changes. Chelonians with hypovitaminosis A often will manifest palpebral edema. Published reports suggest that hypovitaminosis A is more common in aquatic chelonians than in tortoises. Of 144 tortoises necropsied, only two cases of suspected vitamin A deficiency were seen.[76] Vitamin A deficiency was considered as a possible predisposing factor in respiratory infections of captive desert tortoises.[35] However, in a recent study on URTD of desert tortoises in the western Mojave Desert, concentrations of serum and liver vitamin A were not significantly different between clinically healthy desert tortoises and

desert tortoises with URTD.[69] Although squamous metaplasia of the mucosal epithelium of the URT was seen, it was considered secondary to chronic inflammation.

Hypothyroidism

There are several reports of hypothyroidism in Galapagos tortoises. However, except for one report, these studies are limited to clinical and histologic findings.[44,114] In a recent report, a debilitated and anorexic adult male Galapagos tortoise had T3 and T4 values of 4.50 ng/dl and 0.29 μg/dl, respectively, which were significantly lower than those of three clinically healthy Galapagos tortoises.[95] The hypothyroidism was corrected by administration of synthetic levothyroxine and subsequent improvement of the diet.

Toxicosis

Virtually nothing is known about the effects of pollutants or toxicants in populations of tortoises. In desert tortoises in the western Mojave Desert, liver mercury concentrations of tortoises with respiratory tract disease (0.326 ppm) were significantly higher than those of healthy tortoises (0.0287 ppm) from the eastern Mojave Desert.[69] Although these values were below those considered toxic for mammals,[81] depressed immune function has been seen in rodents exposed to sublethal amounts of mercury.[80,81] Additional studies are needed to identify the source and significance of mercury in this population.

Neoplasia

There are only a few reports in the literature describing neoplastic diseases of tortoises. A carcinoma of the stomach of an elephantine tortoise (presumed to be *G. elephantopus*) was described.[97] However, no microscopic description was given. A fibroadenoma of the lung was reported in a Horsfield's tortoise.[32] A 320-g male Hermann's tortoise developed a lymphoblastic lymphosarcoma that involved all major organs.[63] A parathyroid adenoma was identified in a red-footed tortoise.[43] An adenomatous proliferation of the intrahepatic bile ducts was reported in a male African pancake tortoise (*Malacochersus tornieri*).[29]

Anomalies

Shell anomalies, which may be environmentally and/or genetically based, have been reported for the desert tortoise on the Beaver Dam Slope, Washington County, Utah and in the Desert Tortoise Natural Area.[49] The tortoises examined in the DTNA population had 11.2% scute anomalies as compared with 20.4% for tortoises on the Beaver Dam Slope. There also was a difference between the two populations with regard to the types of anomalies and distribution on the shell. The Beaver Dam Slope population had significantly fewer scute reductions and significantly more supernumerary marginals than did the DTNA population. The DTNA had a greater number of tortoises with reduced marginals, supernumerary vertebrals, and fused toes.

Trauma

Traumatic injuries are commonly seen in wild tortoises. Crushing injuries to the shell occur from off-road and on-road vehicles. Vehicular injuries and injuries from dog bites (and other predators) are commonly seen in gopher tortoises at the Veterinary Medical Teaching Hospital, University of Florida. Cattle may step on tortoises, resulting in significant and life-threatening injury to the shell. Penetrating wounds through the carapace into the lung field often result in pneumonia. Several female Aldabra and Galapagos tortoises have suffered forelimb fractures resulting from breeding attempts by large males (pers. obs.). Cactus spines embedded in subcutaneous tissues of desert tortoises resulted in a localized inflammatory response (pers. obs.). Cases of drowning and near-drowning of desert tortoises have been seen in Tucson, Arizona, from falls into swimming pools (Jarchow, pers. comm.).

MISCELLANEOUS DISEASES AND LESIONS

A variety of noninfectious diseases have been reported to affect the integument of tortoises. Cornified epidermal cysts were reported in a spur-thighed tortoise.[110] In another report, small nodular subcutaneous cysts were seen at a variety of sites on the skin of spur-thighed tortoises, but chiefly in the region of the head and neck.[50]

Captive tortoises are commonly seen with flaking and sloughing scutes.[50] Although bacteria and fungi are often cultured from underneath these affected scutes, in most situations these organisms probably are secondary invaders. The cause of this flaky condition is not understood, but it may be associated with dehydration or a nutritional deficiency.

An apparently nutritionally related disease is commonly seen in captive-reared tortoises. With this disease, as tortoises grow, the scutes become malformed, developing a pyramid appearance. Hobbyists who raise these animals believe the problem to be related to excessive protein in their diet. However, all reports are purely anecdotal, and the exact cause of this abnormal growth is unknown.

I have examined several Aldabra and Galapagos tortoises in which the colons were impacted with sand. A long-term captive-reared leopard tortoise was seen with duodenal ulcerations presumably resulting from sand abrading the mucosal surface. Tortoises kept in outdoor enclosures with a sand substrate will invariably ingest sand when eating. It may take several years before enough sand is ingested to cause a medical problem.

A variety of noninfectious diseases of the urinary system have been seen. Although visceral gout was not reported in one study,[76] it has been reported as occurring in tortoises.[7] Although one author described nephrolithiasis as common in tortoises,[99] another author observed only a few cases.[76] A syndrome described as metastatic calcification of the renal tubular epithelium and glomeruli with calcium cast formation has been reported.[27] Although the author believed this syndrome to be related to nutritional factors, most likely vitamin D deficiency, another investigator found that nephrocalcinosis was often unassociated with nutritional osteodystrophies or suspected hypovitaminosis D.[76] Cystic calculi were seen in six tortoises, and a ruptured urinary bladder and peritonitis was seen in a single animal. Cystic calculi have been seen in captive desert tortoises,[39] and cystic calculi have been found in several wild desert tortoises (pers. obs.).

Egg peritonitis and oophoritis has been reported to occur in tortoises.[76] Captive tortoises with impacted eggs are not uncommon, and although some may respond to injections of oxytocin, salpingotomy and cesarian delivery is often necessary.[45]

There are few diseases reported to affect the reproductive tract of male tortoises. Testicular interstitial cell hyperplasia was reported in an adult male desert tortoise.[42] There are no reports of neoplasia of the reproductive tract of tortoises.

Without going into details, Graham-Jones mentioned that in tortoises, diseases of the eye can occur spontaneously and should always be regarded as indicative of generalized malnutrition and inanition.[50] Panophthalmitis is usually bilateral; eyes are obscured with caseous exudate. Conjunctivitis may occur with panophthalmitis or as a separate condition. Conjunctivitis has been seen in several tortoises (pers. obs.), the causes of which have yet to be determined.

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