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Diseases and Infections of Snakes: A Review[1]

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INTRODUCTION

Snakes are scaly, ectothermic, lungbreathing vertebrates of the order Serpentes in the class Reptilia.[2] They are generally separated on the basis of their anatomy, venom production, and adaptation to burrowing, terrestial, aquatic or arboreal life. Use of snakes for experimental purposes is not uncommon in major research centers, in spite of the fact that laboratory colonies of snakes do not exist—if reproduction in captivity is a criterion. Experimental collections are comprised of animals taken from the wild and maintained in terraria; therefore, knowledge of the diseases they contract and the microbial agents they carry is essential to their management in captivity.

Terrestial snakes are best suited for laboratory use because of their small to medium size, mild disposition and simple dietary requirements. The nonvenomous types most commonly used are garter snakes (genus *Thamnophis*), water snakes (*Natrix*), and kingsnakes (*Lampropeltis*). They can be used for investigations where it is desirable to have metabolically retarded, quiescent experimental animals under test for long periods. Ectothermic adaptability provides an additional variable which may be useful.

Reviews of the microbial infections of snakes are rare. In 1936 Glidden summarized certain diseases of snakes known at that time; Klauber (1956) listed numerous diseases and infections of rattlesnakes; and diseases of reptiles have been briefly reviewed by Schlumberger (1958) and by Reichenbach-Klinke and Elkan in 1962 (published in 1965) as parts of general treatises on diseases of cold-blooded vertebrates. Otherwise, the details of investigations of diseases of snakes are scattered among many scientific publications. The purpose here is to bring together current detailed information on diseases of snakes including data not previously reviewed.

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[2]The generic, species and common names of snakes used throughout this paper are those recommended by Conant *et al.* (1956).

GENERAL CLINICAL SIGNS

The appearance of lethargy is natural for reptiles especially when the environmental temperature is cool. Therefore, clinical signs of disease are not readily discernible until lesions are visible or until emaciation, convulsions, exudates or unusual excretions, or weakness become obvious. Refusal to eat may be deceptive since newly captured snakes may take no interest in food for weeks after arrival into the laboratory. Kingsnakes or water snakes, however, will refuse food or fail to respond normally to sensory stimulation when seriously affected by bacterial or protozoan caused diseases of the oral and alimentary tracts. Respiratory infection may induce gaping. Septicemic infections may cause a rapid change from normal behavior to extreme lethargy, weakness, incoordination, and death. Enteric infections may cause abnormal excretions, such as bloody stools. Grass and garter snakes are more active than kingsnakes so that physical changes in them are more noticeable. Determinations of body temperature of snakes are of no value since ectotherms always accommodate to environmental temperatures. Other signs of disease, for example, the cellular or chemical changes in composition of the blood of snakes, have been rarely studied.

VIRAL INFECTIONS

Encephalitis

Studies of virus infections of snakes have been centered on the possibility that reptiles were reservoir hosts for arthropod borne encephalitis viruses. Thomas *et al.* (1958, 1960) used garter snakes (*T. sirtalis parietalis* and *T. elegans vagrans*)to prove that western encephalitis virus was perpetuated in these snake species during winter hibernation. Working with eastern encephalitis virus (EEV), Karstad (1961) broadened the study of reptilian reservoirs to include 11 species of snakes of the genera *Elaphe, Coluber, Natrix, Faraneia, Heterodon, Opheodrys, Haldea, Diadophis, Drymarchon, Ancistrodon,* plus 10 species of turtles and lizards. Initially, Karstad collected blood samples from 99 reptiles and tested them for EEV neutralizing antibodies. Five of the 57 snakes in the group were found to possess significant titers, that is neutralization indices of 2.0-3.0, thereby suggesting natural infection with EEV. But the virus was not isolated from any of the high titered animals. Subsequently 33 serologically positive and negative snakes of the 10 genera mentioned above were subcutaneously inoculated with virus doses ranging from 10-1000 chicken embryo LD_{50}s to determine the persistence of the state of viremia and development of antibody titers. The average duration of viremia was 12 days although one case of 42 days was recorded. Virus concentration in the blood ranged as high as $10^{10.5}$ chick LD_{50} per ml blood. High neutralization antibody titers were also developed. No sign of illness attributable to virus infection was observed in any snake although several died due to refusal to eat in captivity.

Karstad concluded that individuals of all major reptile groups

were susceptible to moderate doses of EEV by subcutaneous inoculation, that high serologic titers in wild reptiles indicated natural infection, and that reptiles may circulate EEV in greater quantity than previously reported in wild animals.

Gebhardt *et al.* (1964) extended work of Thomas *et al.* and found that the blood of 37 of 84 wild racer, garter and gopher snakes of the genera *Thamnophis, Coluber* and *Pituophis* contained western encephalitis virus. These authors further observed in snakes a cyclic viremia which appeared and disappeared according to changes in environmental temperature. Young snakes born of infected females were found to be infected. A high percentage of mosquitos feeding on infected snakes became infected with WEE virus. No pathological effect of the virus on the snakes was reported.

BACTERIAL DISEASES

Ulcerative stomatitis. — The most common and troublesome disease in captive snakes is ulcerative stomatitis, commonly called "mouth rot". It is caused by any one of several strains of a bacterium, *Aeromonas hydrophila* (which is essentially identical with *A. liquefaciens* or *A. punctata*). Burtscher (1929, 1931) described 44 cases of "deep inflammation" of the jaw bone and oral cavity in 13 species of snakes (*Natrix natrix, Coronella austrica, Tarbophis fallax, Coluber leopardinus, Zamenis dahli, Coelopeltis monspessulana, Vipera berus, V. berus prester, V. ammodytes, Eryx johni, Epicrates cenchris, Spilotes pullatus,* and *Naja tripudians.* He isolated a bacterium common to all the cases, presumably *A. hydrophila,* and found the disease could be reproduced readily in *N. natrix* with it. The disease also has been produced experimentally in California kingsnakes (*Lampropeltis getulus californiae*) (Page, 1961). *Aeromonas hydrophila* also has been isolated from the inflamed ulcerous mouths of the Ecuadorian kingsnake (*L. doliata mircopholis*) tree boa (*Boa endyris cooki*); Mexican boa (*Constrictor, constrictor mexicana*); coast patch-nose snake (*Salvadora hexalepis virgultea*); copperhead (*Ancistrodon mokeson cupreas*); Philippine python (*Python reticulatus*); and a watersnake (*N. rhombifera rhombifera*), (Page, 1962).

Aeromonas hydrophila is a gram-negative, polarly monotrichate, gelatin liquefying, fermentative rod whose normal habitat is the soil or natural waters. Therefore it is not surprising that a variety of organisms of this type have been isolated from cold-blooded vertebrates, both diseased and normal. A primary characteristic of the organism is its strong proteolytic activity at temperatures of 20° C and below which is one of the reasons why it has been associated with ulcerative inflammations of the skin of frogs, toads and fishes.

Aeromonads in the mixture of organisms usually found in ulcerative lesions in the mucous membranes or skin of reptiles can be readily isolated on solid bacteriological media containing ordinary bovine, sheep, rabbit or human

blood in a tryptose agar base. Tests described by Ewing, Hugh, and Johnson (1962) assist in distinguishing aeromonads from common enteric bacteria. Two biochemical types (gas forming, Voges-Proskauer positive; nongas forming, Voges-Proskauer negative) of aeromonads exist, and both cause disease in reptiles.

The first sign of ulcerative stomatitis in snakes is the appearance of a frothy, fibrinous exudate around the lips and in the mouth. The animals then refuse to attack live prey or eat. Within a week, flecks of white or yellowish-white caseous masses are seen in the buccal cavity. The mouth tissues become friable and bleed easily. Ulcers form in the mouth and fill with fibrinous exudate which in time becomes caseous. When the core of an ulcer is ejected, a fresh bleeding wound remains. The acute stage with fibrinous exudates lasts from the seventh to the tenth day after infection and becomes chronic thereafter. Partial obstruction of breathing passages by exudate causes the snake to gape to improve respiration The chronic condition may remain unchanged for weeks and the snake becomes steadily weaker. Some cases are less severe with fewer ulcers, and the animal occasionally eats, prolonging the course of the disease. Should the snake be unable to resist systemic invasion of bacteria from the mouth during the acute stage, septicemia and death follows. Spontaneous recovery is rare.

A presumptive diagnosis can be made from the typical signs and lesions and is confirmed by isolating and identifying the causative bacterium.

Diseased snakes should be isolated from unaffected ones. Several prophylactic treatments with effective antibiotics or chemotherapeutic agents should be given to all cage mates of affected snakes. Contaminated cages should be cleaned and sterilized before reuse. Since *A. hydrophila* is commonly found on the skin and in the mouth of diseased and normal frogs, toads, alligators and some snakes, the habitual disinfection of the hands of persons caring for mixed collections of ectotherms is necessary. Quaternary ammonium disinfectants are suitable for this purpose.

Successful topical treatment of ulcerative stomatitis in snakes with an aqueous solution of 25% sodium sulfamethazine has been reported (Kauffeld, 1953; Page, 1961). The exudates in the mouth ulcers are gently removed with drug-soaked cotton swabs, and the mouth is sprayed with several milliliters of the drug solution. Similar treatment is continued daily until clinical improvement occurs. Most cases improve in 2-5 days. The spray treatment is ineffective when applied once a week for three weeks. Treatment with "silver vitellin", a 25% silver-conjugated protein (Hunt, 1957), and various tetracyclines (Rothman Rothman, 1960) has had equivocal success.

Septicemia.—According to necropsy records at various zoos, snakes have died from septicemia. This term is used when necropsy reveals discolored, hyperemic organs and tissues, and microscopic evidence of bacteremia. In some instances, the identity of the terminal invader and the events leading to its overgrowth were obscure. Of those identified, *A. hy-*

drophila and *Pseudomonas aeruginosa* were the principal invaders. The routes of infection were by extension of infection from inflamed and ulcerated areas of the mouth, or intestines, or through bite wounds from blood sucking mites carrying pathogenic bacteria. Cases involving snakes of the genera *Tarbophis, Coelopeltis, Naja, Natrix, Boa,* and *Lampropeltis* are recorded (Burtscher, 1931; Camin, 1948; Page, 1961.)

Because of the rapidity with which death follows invasion of the blood by organisms such as *A. hydrophila,* few clinical signs precede the terminal stages. Camin (1948) reported that the first sign, sluggishness, was seen in snakes 2-3 days after they were bitten by mites carrying *A. hydrophila.* Convulsions occurred one hour prior to death. Gross examination revealed hemorrhages in the oral mucosa, hemorrhage or congestion in the visceral organs, and sanguinous exudates in the abdominal cavity. Pure cultures of *A. hydrophila* were recovered from heart blood.

In one case observed by the author, death of a boa (*Constrictor c. constrictor*) was attributed to septicemia following the development of severe intestinal inflammation anterior to a mass of indigestible material obstructing the lumen. The bacterial population in the lumen of the damaged intestine and in the surrounding visceral organs was predominantly *Ps. aeruginosa.* Pure cultures of this organism were recovered from the heart blood and the liver.

Salmonellosis. — Numerous reports by Hinshaw and McNeil in the 1940's established that at least 8 species of snakes were hosts to pathogenic bacteria of the genus *Salmonella.* In most cases, the serpentine host was an unaffected carrier but in some cases signs and lesions were evident. In one study (Hinshaw and McNeil, 1945); 11 of 41 garter and gopher snakes were *Salmonella* carriers. LeMinor *et al.* (1958) reported isolating *S. java* and *S. arizona* from native venomous snakes of France. Steiniger (1961) observed a common viper (*V. berus*) kept in a terrarium that excreted *S. typhimurium* for several weeks.

Hinshaw and McNeil (1946 a,b) observed lesions of acute disease in 2 of 15 rattlesnakes (*Crotalus viridis* and *C. cerastes*) infected with bacteria of the arizona group serotype 10. Although few or no signs of disease were observed in the snakes prior to their death, bacteria were isolated from multiple necrotic foci in the liver. Two specimens exhibited no gross lesions but the bacterium was isolated from the blood, lungs, kidneys and intestine. In the report of Rewell *et al.* (1948) of an ulcerative enteritis in a python (*P. sebae*) caused by *Salmonella takoradi,* part of the inflamed intestinal mucosa was denuded leaving a raw surface.

No specific treatment for diseased snakes carrying *Salmonella* or other enteric bacteria has been reported. Control measures include the elimination of any contaminated food sources. Secondly, the excreta of all members of a collection should be tested for the presence of pathogenic enteric bacteria with isolation or elimination of any carriers.

Mycobacterial Infections.—Tuberculosis has been described by Aronson (1938) in a diseased ringsnake (*N. natrix*), gopher snake (*P. catenifer*), cat snake (*Tarbophis fallax*) and in common garter snakes (*T. sirtalis*). The disease has also been seen in an unnamed Indigo snake by Glidden (1936) and in an unnamed cobra by Nigrelli (as cited by Vogel, 1958). The disease in reptiles is not common and little is known of its natural distribution and incidence. Francis (1958) reported that from 1931-1933 there was an average of 3400 reptiles and amphibians in the London Zoo, but only 11 instances of tuberculosis were observed.

Mycobacteria from serpentine hosts are pathogenically and biochemically distinct from endothermal animal strains. *Mycobacteria thamnopheos* from garter snakes is a slightly curved, acid-fast bacillus which frequently has a parallel arrangement but occasionally shows beaded and barred forms (Aronson, 1929). It is pathogenic for garter snakes, frogs, certain lizards and goldfish, but is nonpathogenic for rabbits, guinea pigs, chickens and pigeons. Vogel (1958) devised a key for differentiating *Mycobacteria* from ectothermal animals based on biochemical reactions.

Clinical signs of tuberculosis in snakes have not been described. Gross lesions such as caseous nodules in the subcutaneous tissue along the spine, about the heart and aorta, and in the liver and spleen of naturally infected snakes have been seen at necropsy. Aronson (1929) observed slightly elevated, grayish-white, irregularly shaped nodules distributed throughout the organs of garter snakes. Splenomegaly was present. Lung cavities contained large numbers of acid-fast bacilli. Aronson stated that the tubercles consisted of central necrotic areas surrounded by large epitheloid cells. The tubercles were separated from each other by narrow bands of fibrous connective tissue. Numerous infiltrating cells surrounded the tubercles but no calcification or giant cell formation was observed.

In an extensive study of the types of pathologic conditions found in reptilian kidneys, Zwart (1964) observed a case of renal infection of a python (*P. spilotes*) with "cold-blooded tubercle bacilli". Extensive proliferation of epitheloid cells and central necrosis in the tubercles was observed. No attempt was made to isolate or identify the disease agent.

No treatment is known. Isolation or destruction of infected hosts and their surviving cagemates and sterilization of their cages before re-use are the only preventive measures.

Leptospirosis.—The isolation of *Leptospira ballum* from an eastern hog-nosed snake (*Heterodon platyrhinus*) was reported by Ferris *et al.* in 1961. The snake was captured in an area from which numerous *Leptospira*-carrying rodents were collected, although no evidence existed that it preyed upon mice. Serum of the snake had an agglutination titer of 1:10,000.

In 1962, Abdulla and Karstad reported incontrovertible evidence that (1) serologically negative garter snakes (*T. sirtalis*) were susceptible to experimental infection with *L. pomona*, (2) that the

organisms localized in the liver and kidney where they persisted for more than 6 months, and (3) that transmission of the infection by natural means between inoculated and uninoculated cagemates occurred. Inoculated snakes harbored the organisms for the 6 month period of the experiment and developed high agglutinin titers. Furthermore, they carried the infection successfully through a 70 day period of induced hibernation. Uninoculated cagemates of the inoculated snakes became infected by natural means and harbored the organisms for at least 6 months. This evidence suggested that pond-frequenting garter snakes had a high potential as an overwintering host for *L. pomona*.

In regard to leptospirosis causing tissue damage in snakes, the authors reported that there was little evidence to suggest that leptospiral infection interfered with the general health of the inoculated snakes. One snake died 56 days after inoculation and evidence of an interstitial nephritis was observed. The livers and kidneys of other inoculated snakes, however, did not appear damaged at termination of the experiment.

Serums from numerous other species of snakes have been tested for leptospiral agglutinins by various investigators. White (1963) found the serums of 16 to 110 water snakes (*Natrix spp.*) and moccasins (*Ancistrodon spp.*) agglutinated one or more leptospiral serotype antigens, predominantly *L. ballum*. Also, 22 of 44 serums from 7 different species of land snakes contained agglutinins which reacted with either *L. pomona, L. ballum, L. grippotyphosa* or *L. icterohaemorrhagiae* serotypes. Attempts made by White to isolate *Leptospira* from these snakes failed, possibly due in part to the presence of large numbers of bacterial and fungal contaminants.

Failure to recover leptospires from serologically positive snakes was also reported by Andrews *et al.* (1965). These workers detected agglutinins for *L. ballum* (and other serotypes) in the serums of 3 of 10 species of snakes studied. The incidence of serologic positives within the 3 species ranged between 11 and 38%. The significance of this serology is not known because of the failure to recover leptospires. No evidence of pathological changes in the snakes caused by leptospiral infection was reported.

MYCOTIC INFECTIONS

Incidental to a study of virus infections in snakes, Karstad (1961) reported that minor infections of the skins of experimental snakes with bacteria and fungi occurred and that the fungus *Geotrichum candidum* was isolated from caseous subcutaneous nodules in a banded watersnake. In a personal communication to the author, Karstad indicated that he had isolated *Geotrichum* from pustules on the skin of a captive garter snake. Karstad commented that this condition is commonly associated with a continuously damp environment for captive snakes.

Other than these reports, the author is not aware of any other published evidence of mycotic infections of snakes.

PROTOZOAN DISEASES AND INFECTIONS

Rhizopoda

Endamoeba invadens. — Ulcerative enteritis, hepatitis and gastritis caused by *E. invadens* is a common cause of fatality in captive snakes. The natural and experimental disease caused by this agent in reptiles was thoroughly studied by Ratcliffe and Geiman (1938). Listings of serpentine species affected have been compiled by Fantham and Porter (1953-1954) and epizootics among zoo animals have been discussed by Hill (1953-1954) and Graham-Jones (1963). Ratcliffe and Geiman's work was the most comprehensive, so their results will be discussed in detail.

The clinical signs observed by Ratcliffe and Geiman in 32 naturally infected snakes of 7 genera (*Natrix, Thamnophis, Epicrates, Constrictor, Lampropeltis, Cyclagras,* and *Pseudoboa*) were variable because of the different stages of infection encountered. Data obtained from experimentally infected snakes (3 species of *Natrix*) served to explain these variations. For at least 2 weeks after feeding cysts of *E. invadens* (originally recovered from water snakes) to the experimental snakes, the hosts did not eat, and only occasionaly could they be tempted to eat after this period. Their weight declined and their excreta sometimes contained blood-stained mucus which by microscopic examination revealed cysts and trophozoites of *E. invadens.* Survival times varied between 13 and 77 days depending somewhat upon the number of cysts that were fed. Signs of disease were variable in the snakes fed small numbers of cysts.

Ratcliffe and Geiman observed that tissue changes caused by *E. invadens* were found in the stomach, small intestine, large intestine and liver, although the organisms were observed in blood vessels, lung, spleen and pancreas with a minimal damage. The major effects of the disease in both natural and experimental cases were found in the large intestine and liver although the stomach and small intestine were often involved. The disease apparently began in the large intestine forming lesions with subsequent spread of amoebas via lymph and blood to the liver and other organs. Ulcers of the stomach mucosa developed as a secondary response in a majority of affected snakes, although in some cases, lesions were found only in the stomach. In the colon, small intestine and stomach, tissues became necrosed and inflamed. Intestinal lesions, which in the early stages measured 1-5 mm across, gradually became confluent and covered much of the mucosal surface and penetrated the mucosa and wall of the colon. Thrombi and inflammatory changes occurred in the blood vessels following entrance of organisms into the blood stream from the lymph channels. Similar lesions of less severity than those in the colon formed in the stomach and small intestine. Amoebas invaded the liver through the portal vein, the branches of which became blocked by thrombi. Necrosis observed in the liver were probably caused by obstruction of blood vessels.

Ratcliffe and Geiman concluded that the lesions of the digestive

tract were initiated by lytic action of *E. invadens* but that the characteristic changes were due to combined action of amoebas and bacteria and to vascular thrombosis. They also believed that *E. invadens* caused "focal necrosis of the hepatic parenchyma but obstruction of branches of the portal vein by thrombi and emboli lead to massive necrosis of the liver and obscured the effects of the amoebas."

More recently, Zwart (1964) observed 16 cases of *E. invadens* infection in snakes and lizards. In 7 of the cases, amoebas had caused necrosis and inflammation of renal tissue.

An account of an epizootic caused by *E. invadens* among snakes and lizards in the Gardens of the London Zoological Society was reported by Hill and Neal (1953-1954). *Endamoeba invadens* was isolated from natural cases and used to reproduce the disease in grass snakes (*N. natrix helvetica*) and slow-worms (*Anguis fragilis*). Hill included a host list for *E. invadens* comprising 33 species of snakes (including new hosts belonging to the genera *Ancistrodon, Dimades, Eunectes, Naia, Boaedon* and *Python*).

In an extensive, systematic account of endoparasite infections of North American snakes, Fantham and Porter (1953-1954) speculated on the effects of crowding, temperature variations, humidity, and diet on amoebiasis in reptiles. The only management practice that seriously altered the spread of amoebas in terraria was lowering snake population densities. Treatment of infected snakes with emetine bismuth iodide was generally ineffective, possibly due to poor absorption and consequent failure of the drug to reach the parasites. Diagnosis was greatly aided by use of saline enemata to determine the presence of amoebas in fecal excretions.

Subcutaneous amoebic cysts have been frequently seen in newly acquired snakes and the possibility that they result from bites of amoeba-infected lice must be considered. In these cases Graham-Jones (1963) suggests that new snakes should be treated with a de-lousing oil.

Mastigorphora

Haemoflagellates.—Four species of trypanosomes (*T. butananense, T. erythrolampi, T. mattogrossense,* and *T. merremi*) have been observed by several investigators to be present in the blood of various South American snakes. Also, a new species of trypanosome, *T. thamnophis,* was described by Fantham and Porter (1953-1954) as inhabiting garter snakes (*T. sirtalis*). This organism's presence in the blood of garter snakes could not be associated with clinical signs or lesions in infected animals.

Other flagellates observed by Fantham and Porter in the cloaca and intestines of snakes free of gross lesions were *Herpetomonas homalosoma* var. from a garter snake (*T. sirtalis*), *Eutrichomastix serpentis* from the "North American snake" (*Storeria dekayi*), *Chilomastix* sp. from garter snakes (*T. sirtalis* and *T. ordinoides*), and *Trichomonas bitis* from diamond-backed water snake (*N. rhombifer*) and the green snake (*Opheodrys vernalis,* formerly *Liopeltis vernalis*). A *Trichomonas* sp. was also seen by these authors in the Say's King-

snake (*L. getulus holbrooki*). This same species of snake was observed to harbor a possibly new species of flagellate *Giardia sp.* which had features distinct from *Giardia* organisms seen in amphibians and rodents. These entozoa were observed to persist in their hosts from two months to two years, with no apparent harm to any of the hosts.

Sporozoa

Coccidiosis.—Fantham and Porter (1953-1954) reported infection by *Eimeria bitis* of gall bladders of garter snakes (*T. sirtalis* and *T. sauritis*). They found that the mucosa of the bladder lining was shredded and tissue damage extended into the submucosa and bile-ducts of the bladder. The bile was viscid and varied in color from pale green to yellow.

Isospora naiae was reported by these authors to be the predominant pathogen in a case of intestinal and gall bladder infection in a timber rattlesnake (*C. horridus*). They found shreds of mucous membrane containing schizonts and gametocytes in the intestinal mucous and evidence of destruction of the epithelium of the villi. Oocytes were found in the bile, but their association with the disease in the gall bladder was not clear.

Zwart (1964) recently discovered a new coccidium (*Klossiella boae*) in *Boa constrictor* but the organism did not cause distinctive lesions.

Haemogregarian Infections. — Haemogregaria of the genus *Haemogregarina* are the most frequently observed protozoa in the blood of snakes. Wenyon (1926) stated that *H. serpentium* was recovered from the lungs of cobras, vipers, and pythons. In these cases, the haemogregaria coexisted with the linguatulid, *Armillifer moniliformis,* which may have been the original host. Haemogregaria have been seen in the blood of the Santa Cruz garter snake (*T. elegans atratus*) and in the western rattlesnake (*C. viridis*) by Laveran (1902), and in the pine snake (*Pituophis melanoleucus*) by Laveran and Pettit (1909).

An unnamed species of *Haemogregarina* was observed by Fantham and Porter (1953-1954) in the blood of a water snake (*N. sipedon*), pine snake (*P. melanoleucus*), Say's Kingsnake (*L. getulus holbrooki*) and cottonmouth (*Ancistrodon piscivorus*) and timber rattlesnake (*C. horridus*). The haemogregaria observed in the various snake species differed in encapsulation, nuclear morphology, staining and in numbers found in and outside of erythrocytes, but no clinical signs or lesions could be attributed to the infection.

HELMINTH INFECTIONS

Since snakes feed principally on live mammals, amphibians, and insects and occasionally on animals of their own kind, they are exposed to numerous parasites harbored by their victims. Therefore, the fact that a variety of helminths have been found in snakes is not unusual nor does that fact imply any necessary significance in disease. Flukes, flatworms, tapeworms and round worms have been

observed in the intestines and other organs of snakes. Trematodes of the genus *Neorenifer,* for example, have been found in the mouths, lungs, intestines, and of snakes, but descriptions of the gross or microscopic lesions are lacking. Rather than repeat long lists of helminths found in various ophidian hosts, the reader is referred to Nevue-Lemaire (1936) for a taxonomic listing, and to Goodman (1951), Fantham and Porter (1953-1954), and Klauber (1956) for descriptions of various helminth-reptile encounters.

ARTHROPOD DISEASES AND INFECTIONS

Linguatulosis—Several forms of linguatulids seriously affect many species of snakes. Tongue worms of the genus *Armillifer* have been recovered principally from Asiatic and African snakes (genera *Python, Constrictor, Vipera*) but a few American snakes (genera *Crotalis, Ancistrodon*) have been affected (Hill, 1934). Members of the *Python* genus are the most common hosts.

Armillifers are degenerate legless endoparasitic arachnids which usually pass through an egg to larval stage in one host and a nymphal and adult stage in another host. A mammal or fish is often an intermediate host. The larval stage of *A. armillatus* is found in numerous mammals, mainly rodents, e.g. ground squirrel, pouched rat, in which the parasitic life cycle is never completed and nymph forms perish with their host. The encysted nymphs are walled off in a fibrous capsule. Reptiles are exposed when they prey upon a larvae-infected host. In the ophidian host, the larvae of *A. moniliformis* pierce the stomach wall within 24 hours after ingestion, and reach connective tissue in the lungs after 4-23 days. Within another month they mature in the lungs where they lay eggs that are expelled in sputum (Swellengrebell and Sterman, 1961). When the larvae are ingested by pythons, they migrate through the intestinal wall to the lungs which they reach usually in three days. They mature in ten weeks (Broden and Rodhain, 1910). Mature armillifers destroy lung tissue and impair lung function. The disease is chronic and its severity depends on the intensity of the infection.

Cysts may be seen in lung tissue and sputum of snakes. Diagnosis can be made by microscopic examination of the sputum for the presence of armillifer eggs, nymphs or adults.

There is no known treatment. Control consists of preventing the spread of eggs through the colony by removal of affected members. Because the eggs can be transmitted to the drinking water by the caretaker, washing of the hands between care of each colony is recommended.

Another linguatulid which afflicts ophidians is *Porocephalus crotali,* commonly found in rattlesnakes. Its life cycle resembles that of armillifers, and its intermediate hosts are muskrats, opossums and skunks, and at least one species of mouse. Penn (1942) studied the life cycle of *P. crotalis*

in the muskrat and snakes and was able to experimentally produce infestations of snakes of the genera *Natrix, Thamnophis, Ancistrodon* and *Crotalus*. Self and McMurry (1948) found rattlesnakes (*C. atrox*) in Oklahoma heavily infected and also recovered larvae from a local white-footed mouse (*Peromyscus leucopus aridulus*).

Acarinosis.—The common snake mite, *Ophionyssus natricis,* has a worldwide distribution attributed in part to the exchange of infected snakes among herpetologists. Most snakes appear to be susceptible to the ravages of this mite, and many are found infected in nature. Schroeder (1934) described the life cycle of *O. natricis* and listed American snakes of the genera *Coluber, Thamnophis, Lampropeltis, Elaphe, Masticophis,* and *Crotalus* as most frequently parasitized. He stated that a majority of snakes examined over a three-year period were infected. Camin (1948) reported infections in *Natrix* and *Heterodon.*

Camin (1948) implicated *O. natricis* in the transmission of hemorrhagic septicemia (caused by *Aeromonas hydrophila*) in snakes. This gasmid mite is a fast moving, bloodsucking arachnid which travels constantly from one feeding spot to another, never fixing itself permanently. Its bloodsucking proclivities are sufficient to cause the death of a snake in three days when the infection is intense (Schroeder, 1934). In chronic infections, death may be preceded by signs of weakness and emaciation. The presence of *O. natricis* on the skin is diagnostic. The adults are readily visible, the immature forms barely visible; however, small, round, white fecal deposits from the mites can be seen easily.

O. natricis infection can be controlled with pyrethrum, malathion, or silica gel powder. Use of pyrethrum or malathion must be carefully controlled since some snakes are highly susceptible to continuous exposure to these chemicals. A light dusting with 4% malathion powder will relieve the infection if the snakes are in contact with the insecticide for 4 hours (personal observation). The insecticide is fatal to snakes if it is not removed from the skin or the cage within 48 hours. Silica gel*, which acts as a desiccant, has no apparent toxicity for snakes and effectively kills mites in 24-48 hours. Schroeder (1934) recommended dipping snakes in an aqueous solution of rotenone. This solution, however, can be toxic to snakes. Because mites drown and detach themselves when the infected part of a snake is immersed in water, dipping may be satisfactory until effective and safe insecticides are available. Control also includes ridding the snake's environment of mites to prevent re-infection.

PHYSICAL, TOXIC AND ALLERGIC EFFECTS

Captive snakes are subject to trauma from numerous sources. In some cases, they may attack each other when caged together at feeding time. Snakes held in screened cages may rub their noses raw in efforts to escape. Repeated injury in this area leaves an abraded wound subject to invasion by proteolytic bacteria, such as *Aero-*

*Sold commercially as "Dri-Die", Fairfield Chemical Div., New York, N.Y.

monas hydrophilia, Pseudomonas aeruginosa and *Proteus vulgaris* which produce ulcers. Prevention of injuries can be achieved by careful handling, proper housing, and separation of snakes. Superficial wounds may be treated with disinfectants. If anesthesia is required, placing the animal in a cold room, 5-10° C, for one-half hour will slow its reactions sufficiently to allow treatment (Graham-Jones, 1963).

Most snakes survive wide variations in air temperature in nature but appear to be more sensitive to such variations in the laboratory. Collections of garter snakes (*Thamnophis*) may be kept in hibernation at 7-15° C in low humidity and in a relatively quiescent state at 15-20° C. The range for normal activity is 20-30° C, although they can survive higher temperatures if given adequate ventilation and protection from high humidity (Benedict, 1932). Exposure of snakes in poorly ventilated glass cages to sunlight may prove rapidly fatal due to heat intensification.

Fatal Emesia.—Newly captured water snakes (*N. rhombifera rhombifera* have been observed by the author to succumb to a condition characterized by sudden excessive vomiting. Although the snakes readily accepted live rodents as food during the first few weeks in captivity, they suddenly began vomiting. Disgorgement of stomach and intestinal contents as well as prolapse of part of the alimentary tract occurred. This fatal emesia could not be attributed to any obvious management factor.

Snakes (and tortoises) appear to be resistant to anaphylactic shock. Placidi and Placidi (1960) attempted to sensitize water snakes (*N. viperina, N. natrix*) and tortoises (*Testudo groeca*) by injecting them intradermally with serums from horses and lower vertebrates. Challenge inoculation was given two and three weeks after the initial dose. No phenomenon of sensitization or shock comparable with that obtained in guinea pigs or rabbits was observed. Placidi and Placidi concluded that the general phenomenon of anaphylactic shock did not exist in the animals studied.

NUTRITIONAL AND METABOLIC DISTURBANCES

Since captive snakes are usually maintained on a diet of healthy, live rodents or insects, they are presumed to obtain sufficient protein, fats, carbohydrates, minerals and vitamins. Thus, nutritional or metabolic disturbances in these animals have not been of much scientific concern.

Dessauer and Fox (1959) observed that snakes can experience a transient plasma hydration and change in the mineral composition of plasma during estrus. Concentrations of calcium, magnesium, protein, and all phosphorus fractions rose to high levels during the period in which yolk accumulated in follicles. The only low levels found during this stage were in two ribbon snakes (*Thamnophis sauritus*) which showed definite evidence of follicular atresia.

NEOPLASMS

Wadsworth (1956, 1960) summarized reports of various sarcomas, carcinomas, adenomas, and fibromas observed in 18 species of snakes over the previous 50 years, and Stolk (1957) reported studies on melanomas of the skin of vipers (*Vipera berus*). The true incidence of neoplasms in reptiles is unknown. Tissues submitted for examination usually come from zoo animals which are under close scrutiny and may live longer in captivity than they might in the wild thereby giving slow growing neoplasms a greater chance to develop.

Superficial tumors of the skin, subcutaneous tissue or mucous membranes are often visible as lumps or nodules. Wadsworth (1956) described a cloacal enlargement in a Southern Pacific rattlesnake (*Crotalus viridis helleri*). Grossly, it was an elongated, spindleshaped, pericloacal mass. Microscopically the growth appeared to be hemangiomatous, containing cystic and necrotic areas with some polymorphonuclear leukocytes.

Unless neoplasms are malignant and metastasize, the snake may survive for years. Internal tumors, however, may cause death by pressure on vital organs. Hill (1952) described an asteosarcoma in a rufousbeaked snake (*Ramphiophis rostratus*) which formed an irregular, hard, nodulated mass 16 inches behind the snout. Death resulted from intestinal obstruction caused by the growth.

According to Lucké and Schlumberger (1949) all of the main types of tumors found in endothermal animals have been observed in ectothermal vertebrates; however, Wadsworth (1956) stated that some varieties of neoplasms are more common in reptiles than are the corresponding tumor types in birds and mammals. There were, nevertheless, no significant structural differences between ophidian neoplasms and the corresponding neoplasms in mammals, birds, and other reptiles.

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