CHAPTER 36

Diseases and Disease Control on Crocodile Farms in Zimbabwe

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THE presence of disease can seriously affect the commercial viability of crocodile farming ventures. In addition, the morality of subjecting crocodilians to conditions under which they may easily succumb to infections or other diseases, is being questioned increasingly throughout the world. It has become important to study the causes of mortality and debilitating diseases among captive crocodilians, and efforts in this direction have now been initiated in a number of countries.

Within Zimbabwe, crocodile farming relies heavily on the raising of Nile crocodiles *Crocodylus niloticus*, which are mostly collected from the wild as eggs (see Childs Chapter 6 and Hutton and Van Jaarsveldt Chapter 31). Mortality between the egg stage and the final raised culling stock is considerable, and is a significant factor in the economics of crocodile farming *per se*. As a consequence, efforts are continually being made to investigate and improve the situation. This chapter discusses some of the findings from these investigations, but does not attempt to review crocodilian diseases worldwide. Even in Zimbabwe there are problems to which we do not yet have satisfactory answers.

MORTALITY

Information on the extent of mortality on the five Zimbabwe crocodile farms, between 1980 and 1983, was extracted from the monthly stock returns made to the Department of National Parks and Wild Life Management (Table 1). The data are subdivided into three age classes: neonates (from hatching to six weeks of age); hatchlings (from six weeks to one year of age); and, rearing stock (one to three years of age). The significance of these losses is perhaps best indicated by their extent relative to the number of animals utilized gainfully during that same period (Table 1). One farm, situated at a higher altitude than the others and thus at a site which experiences relatively colder winters, accounted for about half the total deaths, but hatched only one quarter of the crocodiles.

POST-MORTEM AND LABORATORY EXAMINATIONS

Since 1977, post-mortem examinations have been conducted on some of the crocodiles dying from disease (Table 2). In the period 1980 to 1983, 263 crocodiles were examined, less than 2% of those that died. A high proportion of these were "runts" — hatchlings with retarded growth.

Many of the crocodiles examined suffered from a number of different conditions, often apparently unrelated, and it was therefore difficult to make a primary diagnosis in all cases. A list of conditions diagnosed, together with the number of crocodiles affected, either as a primary cause of disease or incidentally, is in Table 3. The figures do not necessarily relate to the frequency with which these conditions occur on the farms.

Due to the difficulty in assessing gross pathological changes in small crocodiles, considerable emphasis was placed on histopathological examination. Whenever possible, bacteriological cultures and haematological and biochemical tests were carried out. Selected tissues were also examined by Dr Elliott Jacobson of the University of Florida at Gainesville, and Dr Chris Gardiner of the Armed Forces Institute of Pathology at Washington, where electron microscopic studies were done.

DISEASES

Viral Hepatitis and Enteritis

As present it is not clear if these represent infection by two different viruses or are merely the involvement of different organ systems by a single virus type. Viral hepatitis was first seen in a hatchling in 1980, but more recently the syndromes have been recognized with increasing frequency and are now known to occur on three farms. In common with most diseases, infection is most frequently seen in the "runt" hatchlings. Symptoms are non-specific, the crocodiles usually being found moribund or dead. Gross pathological findings are also non-specific. There may be swelling and discolouration

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Table 1. The extent of mortality on crocodile farms in Zimbabwe between 1980 and 1983.

Total eggs collected (wild and captive laid)	= 42,891
Total eggs hatched	= 36,052
Percentage hatch (hatching rate)	= 84.1
Neonatal deaths (first six weeks; December to January)	= 1479
Percentage neonatal deaths	= 4.1
Hatchling deaths (6 weeks to 1 year of age; February to November)	= 9777
Percentage hatchling deaths	= 27.1
Total mortality to one year of age	= 11,256
Percentage mortality to one year of age	= 31.2
Total raising mortality (1-3 years of age)	= 4648
Percentage rearing mortality (52,690 animals reared in this time period)	= 8.8
Grand total of deaths	= 15,904
Total cropped during same period	= 7933

Table 2. Post-mortem examinations carried out on *Crocodylus niloticus* being raised on crocodile farms in Zimbabwe (1977-84). Data include examinations of preserved organs supplied by crocodile farmers.

Neonates (to six weeks of age)	= 38
Hatchlings (six weeks to one year)	= 263
Rearing stock	
Yearlings	= 66
Over two years	= 35
Adults	= 3
Age not recorded	= 15
Total	420

Table 3. The results of the post-mortem examinations of *Crocodylus niloticus* from crocodile farms in Zimbabwe.

Condition and/or Pathological finding	Numbers affected Primary	Incidental
Viral hepatitis	15	
Viral enteritis	15	
Pox virus dermatitis	7	
Bacterial septicaemia	27	
Bacterial hepatitis	33	58
Bacterial pneumonitis	6	
Ophthalmia	16	9
Necrotic stomatitis	3	
Bacterial enteritis	2	4
Retained yolk sac/peritonitis	11	6
Fungal granulomatous hepatitis	1	
Fungal granulomatous pneumonitis	2	
Fungal stomatitis		1
Fungal dermatitis		2
Coccidiosis	35	32
Dujardinascariasis	5	15
Visceral nematodiasis	2	5
Cutaneous nematodiasis	1	
Gout/nephrosis	18	13
Rickets	5	
Vitamin A deficiency	2	
Congenital deformity	1	
Physical extremes/trauma	29	
Hepatosis/pneumonitis syndrome (heat stress)	29	
Nil or not determined	155	

of the liver, which has a mottled pink appearance; the intestinal wall is sometimes swollen and congested with the intestinal contents being fluid and occasionally blood stained; and, the rectal contents are soft, pale and contain excess mucous.

With the hepatic form of the disease, histopathological examination of the liver reveals areas of necrosis and inflammatory cell response, mainly lymphocytic, particularly around the portal tracts. Nuclei of some hepatocytes are enlarged with marginated chromatin and show prominent inclusions which may fill the nucleus (Fig. 1).

In the intestine there is loss of villous structure, necrotic debris on the luminal surface and in the crypts, generalized inflammatory cell reaction in the mucosa, and occasionally hypertrophic epithelial cell nuclei with intranuclear inclusions (Fig. 2). A more detailed description of this syndrome, based on *C. niloticus* tissues from Zimbabwe, has been given by Jacobson *et al.* (1984). Adenovirus-like particles were demonstrated in the small intestine.

The role of this infection in the episodes of massive mortality occurring on some farms, particularly during winter, is not clear. However, it seems likely that damage to the intestine caused by viruses predisposes the crocodiles to bacterial enteritis and subsequent septicaemia. The infection is also often associated with intestinal coccidiosis.

Pox Virus Infection

Pox virus infection was first described in three *Caiman crocodilus* by Jacobson *et al.* (1979). In 1982 several hundred *C. niloticus* hatchlings in Zimbabwe, between 7 and 11 months of age, developed the disease, and over 400 of them died.

The infection appears as circumscribed brown areas on the pale ventral skin, which can be either raised or in the form of shallow ulcers (Fig. 3); brown plaques are usually evident in the mouth. As the disease progresses the skin covering the head and neck shrinks, resulting in deformity of the maxilla and mandible and prominence of the aural (auditory) orifice. There is also some hyperkeratinization. Affected crocodiles have difficulty in eating and may starve.

Histologically, intracytoplasmic viral inclusions can be demonstrated, often with secondary bacterial invasion and exudative dermatitis of the epidermis (Fig. 4). Electron microscopic studies have revealed typical pox virus particles (E. R. Jacobson, unpublished data).

In 1984 the disease reappeared on the same farm but with considerably less morbidity and mortality.

Pox virus inclusions have also been demonstrated in the epidermis of eyelids of some crocodiles with ophthalmia (see later), but it is unknown if the virus is a primary cause of the syndrome.

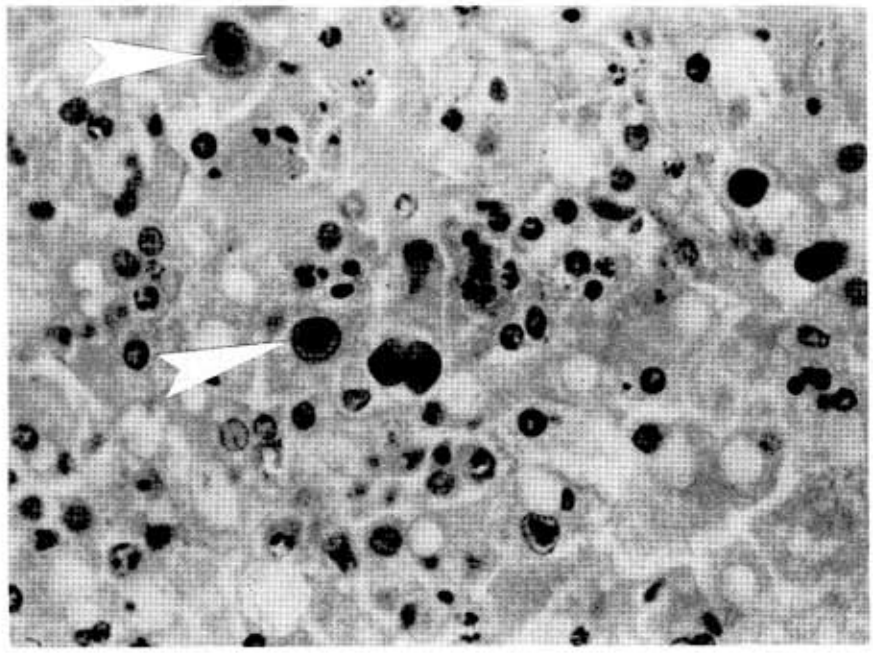

Fig. 1. Viral hepatitis in *Crocodylus niloticus*. A section of liver showing degeneration of hepatocytes, inflammatory response and intranuclear inclusions (arrows) (× 400).

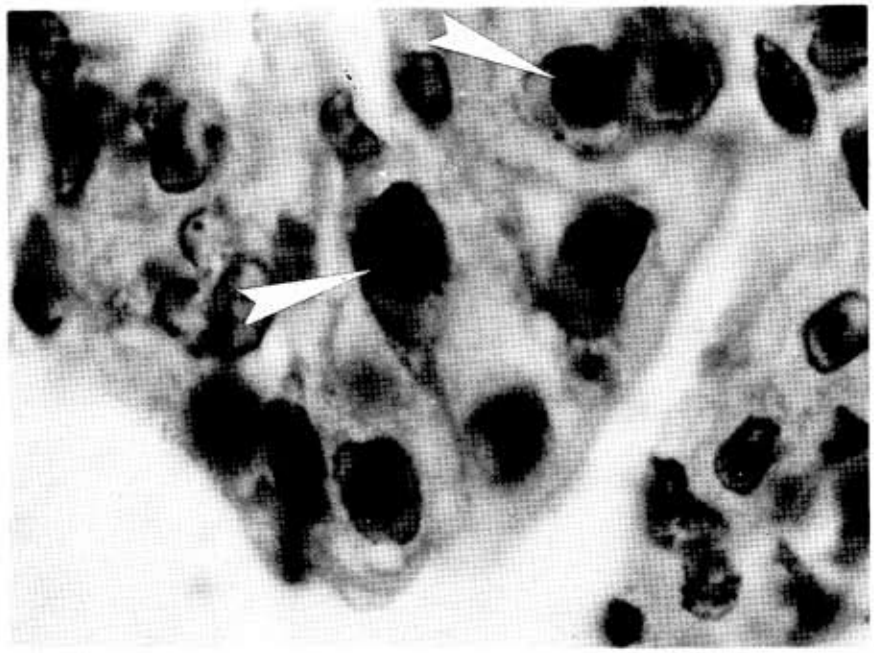

Fig. 2. Viral enteritis in *Crocodylus niloticus*. A section of duodenum with intranuclear inclusions in intestinal epithelial cells (arrows) (× 1000).

Bacterial Infections

Bacterial infections are frequently diagnosed in hatchling crocodiles in Zimbabwe, and can be either localized in various organ systems or the cause of generalized disease.

Acute bacterial septicaeamia causes death, usually within one to two days, and lethargy is the only notable symptom.

On post-mortem, all organs are congested and there may be a fibrino-purulent peritonitis, pleuritis or pericarditis. The liver is sometimes swollen and

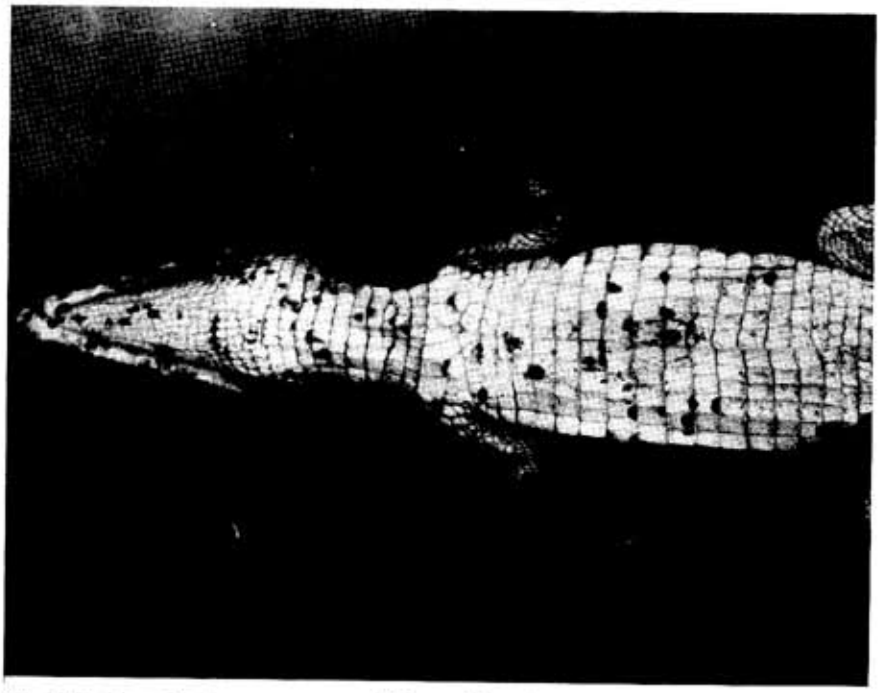

Fig. 3. Pox virus infection on a one-year-old *Crocodylus niloticus*, with circumscribed lesions on the skin of the ventral abdomen.

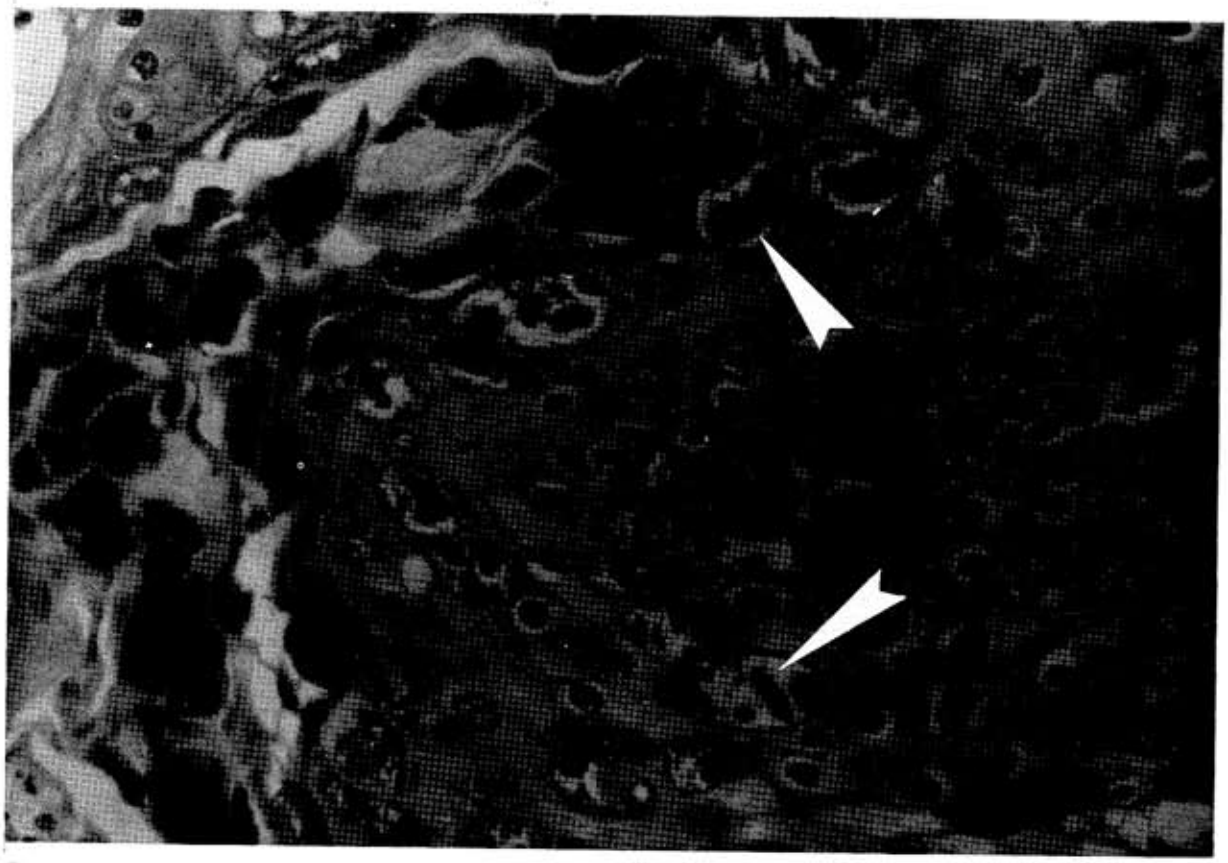

Fig. 4. Pox virus infection in *Crocodylus niloticus*. A section of epidermis with intracytoplasmic inclusions (arrows) (×400).

Table 4. The bacteria isolated during post-mortems of *Crocodylus niloticus* from crocodile farms in Zimbabwe. Of 236 cultures, 108 isolations were made. In most cases where bacterial septicaemia was diagnosed, no bacteria culture was possible.

Disease	Bacteria identified					
	Salmonella arizona	*Salmonella* (other)	*Aeromonas* spp.	*Escherichia coli*	*Pseudomonas aeruginosa*	*Streptococcus* group C.
Viral hepatitis		1				
Viral enteritis	2		1			
Pox virus			3			
Bact. septicaemia	2	2				
Hepatitis	9	6	4	2		
Pneumonitis	1		2			
Ophthalmia			2			1
Necrotic stomatitis			1			
Yolk sac peritonitis			1			
Coccidiosis	5	4	8			
Gout/nephrosis	4	3			1	
Nil	14	17	12			
	37	33	34	2	1	1

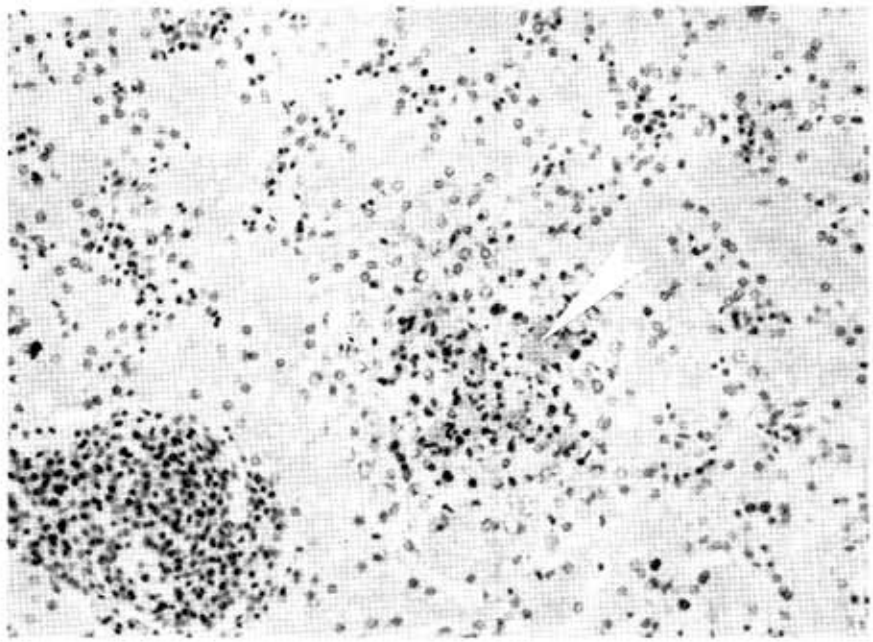

Fig. 5. Bacterial septicaemia in *Crocodylus niloticus*. A section of liver showing focal area of coagulative necrosis with a bacterial colony (arrow) (× 160).

the lungs are uncollapsed and contain serous or purulent material. Petechiae may be evident on serosal surfaces.

Histologically there is often a purulent pneumonitis and necrotic areas in the liver and kidney caused by colonies of bacteria and inflammatory reaction (Fig. 5).

Usually *Aeromonas* spp. (*A. hydrophila*) are cultured, but in a recent outbreak amongst a pen of prime crocodiles, pure cultures of *Salmonella derby* were obtained.

In neonatal hatchlings, particularly those with poorly resorbed yolks, ascending infection of the umbilicus may result in inflammation of the yolk sac and subsequently, a general invasion.

Pneumonitis and especially hepatitis are forms of localized bacterial infections most often diagnosed in unthrifty crocodiles. In the liver this usually takes the form of granulomas or microabscesses. Bacterial enteritis is often a sequel to viral or coccidial infection of the intestine.

Ophthalmia can occur in an epizootic form, affecting entire pens of crocodiles. Both hatchlings and yearlings are prone to this condition. In the early stages there is a serous discharge from the eyes which may stick the eyelids together. As the disease progresses caseous exudate collects in the conjuctival sac; the nictitating membrane and cornea may become severely inflamed and the whole orbit becomes swollen. Frequently a more generalized dermatitis with thickened, cracked skin on the head

and dorsum is associated with ophthalmia. Affected individuals have difficulty in feeding and are reluctant to take to the water. Ultimately they may die from dehydration, malnutrition or some other infectious process. In any event, the disease runs a prolonged course and is debilitating. Apart from the possible involvement of pox virus mentioned previously, *Streptococci* and *Aeromonas* spp. have been isolated from affected eyes. Small intracellular organisms resembling rickettsiae were also demonstrated by electron microscopy.

A summary of all isolations of bacteria is given in Table 4. The significance of them is often unclear and 40% of them occurred in the absence of any obvious pathology.

Fungal infections

A few cases of fungal granulomatous hepatitis or pneumonitis have been diagnosed histopathologically but these appear to be secondary infections and are not considered important. A common condition is fungal dermatitis in which the dorsal skin appears dry and has a fine white coating; in the mouth, a more proliferative reaction is present. On histopathological examination fungal hyphae and spores can be demonstrated in the superficial epidermis (Fig. 6). The species of fungus has not been identified and the infection usually disappears spontaneously.

Coccidiosis

Coccidiosis is one of the major diseases of captive crocodiles in Zimbabwe, and it is seen as both a primary cause of mortality and as an incidental finding. It cannot be differentiated symptomatically from most other diseases which run a subacute or chronic course. However, the more severely affected individuals are usually runts.

On post-mortem, pathological findings may include small haemorrhages on the serosal surfaces, a swollen congested small intestine containing fluid and scant rectal contents which are soft and mucoid. Where there is involvement of the biliary system, the gall bladder wall may be thickened and the contents inspissated; pale foci are sometimes present in the liver.

Histopathologically there may be severe damage to the intestinal mucosa with atrophy of the villous processes, and inflammatory cell response in the lamina propria. Numerous parasites are usually evident in the epithelial cells (Fig. 7); all stages of the coccidian life cycle have been demonstrated, including sporulated oocysts which have also been found in the liver parenchyma, lung and spleen. Secondary bacterial necrotic enteritis and granulomatous hepatitis are common sequels, which bring the infection to a fatal conclusion.

Shedding of oocysts in the faeces is a rare occurrence and diagnosis can only be made on the histopathological findings. The unusual features of this parasite have created problems in its taxonomic classification (C. H. Gardiner, pers. comm.) though it is possibly a member of the recently described family Calyptosporidae (Overstreet *et al.* 1984). However, members of the genus *Calyptospora* require an intermediate host, while epizootiological features of crocodilian coccidiosis in Zimbabwe indicate that direct transmission occurs naturally, and this has been demonstrated experimentally (unpublished data).

Helminthiasis

A number of helminth parasites have been identified in farmed crocodiles, but at present, only two appear to be of economic importance.

Dujardinascaris spp. are frequently present in the stomachs of hatchling and rearing stock. Usually there are less than ten worms, but on occasion a mass of worms may be present, to the exclusion of any ingesta.

The eggs are easily identified in faecal samples by the flotation technique and counts of 150,000 eggs per gram are not uncommon, but appear to bear little relation to the degree of infestation in the individual.

In a review of the genus *Dujardinascaris* (Sprent 1977) it was noted that larval nematodes, possibly *Dujardinascaris* sp., were found in species of African fish and other aquatic vertebrates, and that these represented an intermediate host for the genus. In Zimbabwe, the infestation has only become a problem since hatchling *C. niloticus* started to be fed on lake sardines, *Limnothrissa miodon*. An encysted larval nematode was identified in the liver of one of 18 sardines examined histologically (unpublished data). Larval nematodes, possibly immature *Dujardinascaris*, have been demonstrated in the gastric submucosa of hatchlings.

Worm trails on the belly skin of crocodilians have long been known in the skin trade (King and Brazaitis 1971), and have been observed in wild *C. niloticus* in Zimbabwe (J. Hutton, unpublished data). They have been found in animals of cropping age on one of the Zimbabwe farms. The nematode responsible belongs to the genus *Paratrichosoma* which was described by Ashford and Muller (1978). Zig-zag trails on the surface of the skin are made by migrating nematodes, just beneath the stratum corneum, where the eggs are deposited. Affected skins are down-graded in the market place, and thus the parasite potentially represents a considerable economic problem.

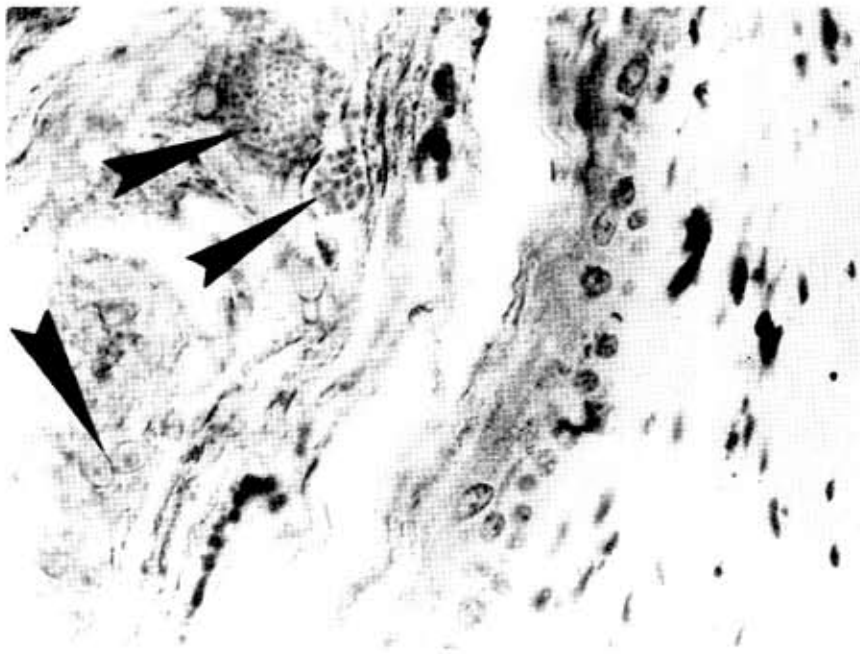

Fig. 6. Fungal dermatitis in *Crocodylus niloticus*. A section of epidermis with hyperkeratinization and fungal elements (arrows) (× 400).

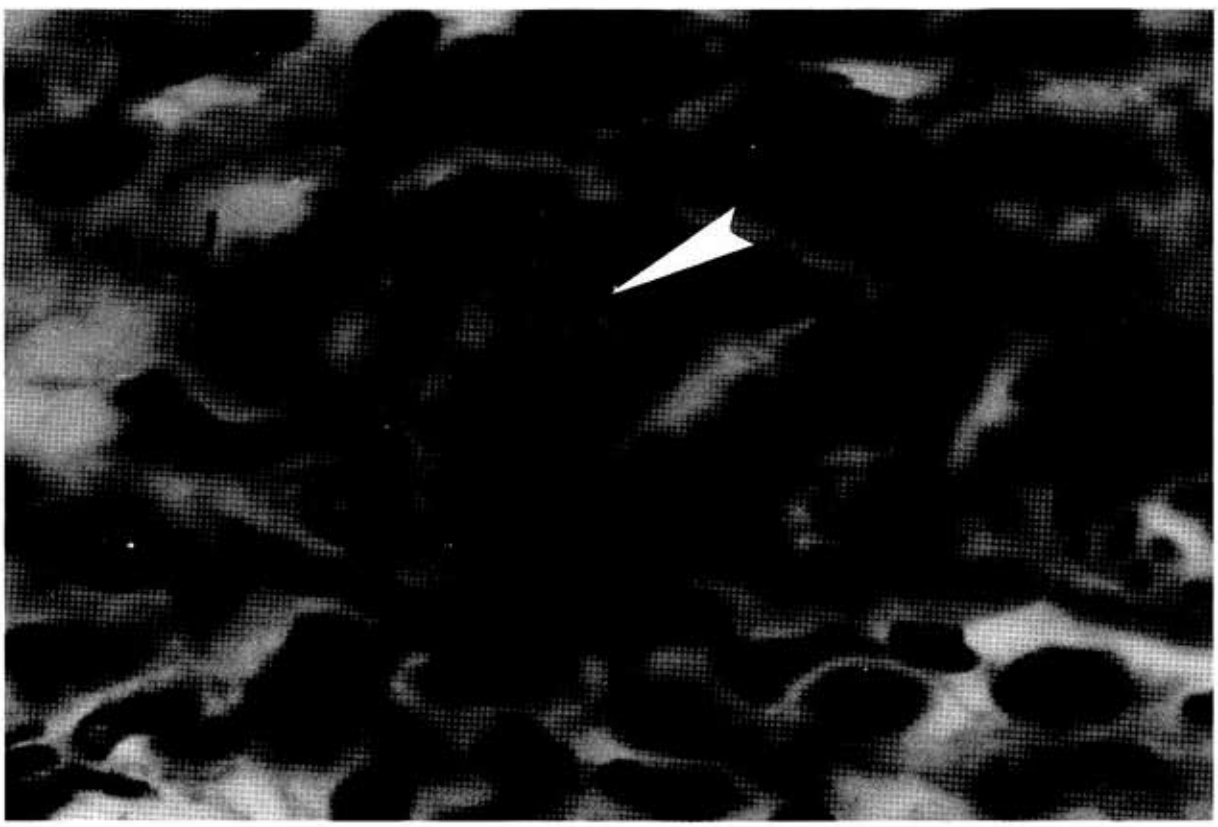

Fig. 7. Coccidiosis in *Crocodylus niloticus*. Duodermal mucosa with mature schizont containing merozoites (arrows) (× 1000).

Other helminth parasites located include *Micropleura vivipera*, a filarial worm found free in the abdominal cavity, unidentified trematodes found in the rectum, ureters and lungs and, unidentified encysted, nematode larvae found occasionally in all viscera.

Kidney Disease

Classical crocodilian gout is well known to those who farm American alligators (*Alligator mississippiensis*) (McNease and Joanen 1981), and is caused by feeding protein in excess of an animal's ability to

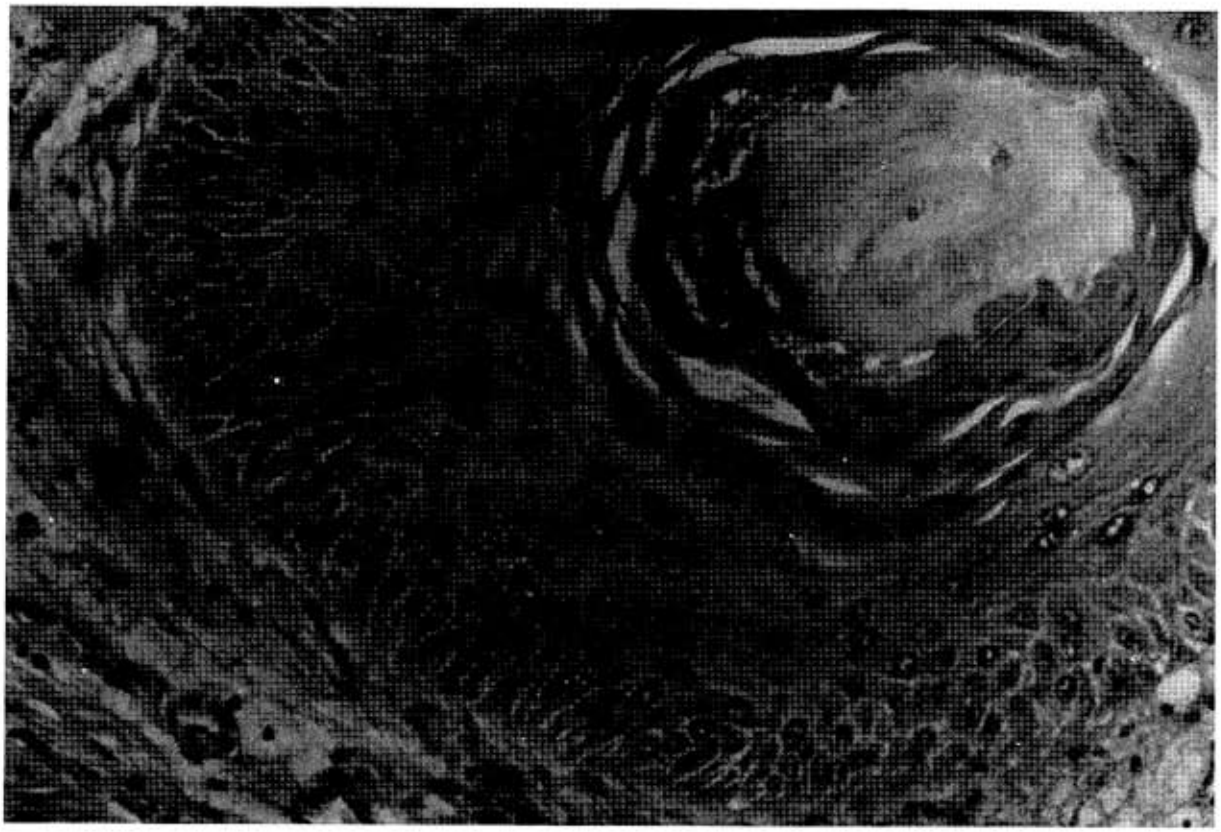

Fig. 8. The result of Vitamin A deficiency in *Crocodylus niloticus*. A uriniferous tubule with squamous metaplasia and accumulated excretory products in the lumen (× 160).

utilize the amino acid products of digestion, in synthesizing its own protein requirements. This results in an excess of the less soluble excretory products, ammonium salts and urates. [The physiological processes involved in *A. mississippiensis* protein digestion and assimilation have been described in detail by Coulson and Hernandez (1983).]

The predisposing causes of gout may be overfeeding, metabolic depression (which occurs at temperatures below 20-25°C, when protein synthesis becomes less efficient), and/or water deprivation. Regardless, excess urates are deposited in the kidney tubules and/or the joints and/or viscera, resulting in locomotory difficulties and organ malfunction.

The first symptom noted is paralysis of the hind legs which progresses to total paralysis. In the early stages withholding feed will result in spontaneous recovery. Crocodiles which die have an accumulation of white material in the kidneys, which may be considerably enlarged, and urates may also be seen in the liver and joints. Secondary bacterial invasion of the kidney can also occur, and once the organ is affected to this extent, the damage is irreversible.

This form of gout is not commonly observed on crocodile farms in Zimbabwe. Paralysis, or alternatively convulsions, and bloating occur in crocodiles which are overfed during winter. A somewhat different syndrome was observed on one farm when water and air temperatures rose above 40°C, and caused considerable mortality among three-year-old crocodiles. Post-mortem examination revealed lung oedema, with some inflammatory reaction, and liver and kidney cellular degeneration without any obvious deposition of urates.

Table 5. Biochemical and haematological values of normal and runt hatchling *Crocodylus niloticus* from crocodile farms in Zimbabwe: N.D. = not determined.

	Normal (mean and range)	Runt (mean)
Haemoglobin (g%)	7.4 (6.4-8.7)	3.9
Packed cell volume (%)	22 (13-27)	13
Total plasma protein (g%)	5.3 (4.6-8.5)	4.2
Albumin (g%)	1.9 (1.5-2.3)	1.5
Globulin (g%)	3.1 (2.2-3.9)	3.0
Glucose (mg%)	81.6 (61.1-117.3)	73.2
Calcium (mg%)	10.5 (8.6-13.8)	10.2
Phosphorus (mg%)	3.0 (1.3-4.4)	2.7
Uric acid (mg%)	4.1 (1.4-7.5)	N.D.
SGOT (iu)	16.6 (6.7-22.7)	42.7
SGPT (iu)	13.1 (9.0-20.4)	34.8

Other forms of kidney pathology include tubular hyperplasia and squamous metaplasia of the main kidney ducts (Fig. 8). This latter change is attributed to a vitamin A deficiency; the loss of mucous secreting cells affects the passage of urates along the ducts, resulting in typical kidney gout.

Other Deficiency Disease

In addition to lesions in the kidneys, vitamin A deficiency may also lead to conjunctivitis and keratitis, while anasarca is observed in grossly deficient individuals. The effect of marginal vitamin A deficiency on growth rate and susceptibility to infectious disease remains to be quantified, but could be important.

The feeding of red meat alone is likely to result in deficiencies of vitamin A, vitamin D and calcium and must be corrected with a food supplement or additive. Lack of calcium and vitamin D causes rickets, which is reflected in uneven growth of the jaws, soft pliable bones (particularly the mandible and maxilla) and fractures of the spine with permanent posterior paralysis.

Few data are available on requirements and/or deficiencies of trace elements. In a limited trial on one Zimbabwe farm, vitamin E and selenium were administered to hatchlings parenterally (by intramuscular injection), and it appeared to have beneficial effects in terms of weight gains during a two month observation period (unpublished data).

Runt Syndrome

It is unknown whether the syndrome in which hatchlings fail to grow is a specific disease entity or the result of a combination of hereditary, congenital, environmental, nutritional and/or infectious conditions. Runts can be identified from six to eight weeks post-hatching and up to 30% of hatchlings may be affected.

These individuals fail to grow as fast as other crocodiles (in length and weight), they take on an emaciated appearance and are listless. Some refuse to eat but others show relatively normal appetites, but still have retarded growth. There are some haematological and biochemical characteristics of runts (Table 5), but they are not considered diagnostic.

Post-mortem findings are non-specific. Apart from poor body condition, there is usually a mild degree of ascites as well as intestinal and liver atrophy; the liver is usually a grey colour. The mesenteric fat storage organ (abdominal fat body) is usually absent.

Histopathological examination reveals increased melanin uptake in the liver and vacuolar degeneration of the hepatocytes. There is usually atrophy of the intestinal villous processes.

Because many of the hatchlings which die from infectious disease are runts, it seems possible that an immune deficiency is also involved.

Miscellaneous Conditions

Congenital deformities are seen and include coiled and curled tails, external yolk sacs and abnormalities of the head, limbs and spine. Some of these may be directly related to extreme incubation temperatures (Ferguson 1985).

Traumatic injuries from piling, fighting and hysterical reaction to management procedures, as well as drowning, can also cause mortality. Additionally, sudden changes in temperature or exposure to extremes of temperature can cause many deaths.

TREATMENT OF DISEASE

Treatment of individuals and of whole pens of crocodiles within Zimbabwe has been undertaken on an empirical basis and the results have not been subjected to any detailed scientific analysis or scrutiny.

Viral Hepatitis and Enteritis

Oxytetracycline hydrochloride powder [Terramycin soluble powder (Pfizer) containing 55 mg oxytetracycline hydrochloride per g] mixed in food at 500 mg kg^{-1} for three consecutive feeds, or 25 mg kg^{-1} body weight dosed by stomach tube for three consecutive days. Antibiotic treatment is used to minimize the effects of secondary bacterial invasion. Disinfectants can also be applied to pond water, for example chlorine at 2 to 4 parts per million or potassium permanganate at 10 parts per million.

Pox Virus Dermatitis

Oxytetracycline hydrochloride aerosol spray [Terramycin anti-infective spray with marker (Pfizer) and 1.5% oxytetracycline hydrochloride] applied topically daily; disinfection of pond water as for viral hepatitis and enteritis.

General Bacterial Infections

As for viral hepatitis and enteritis. Bacterial culture and antibiograms should be performed whenever possible to monitor antibiotic resistance, although this has not yet been a problem on Zimbabwe farms.

Ophthalmia

Chloramphenicol-gentian violet aerosol spray [Pedichlor aerosol (CAPS) containing 5% chloramphenicol with gentian violet] preferably applied daily; or, daily application of a chloramphenicol-sulphacetamide-vitamin A ointment [Chloramide ointment (CAPS), containing 5% chloramphenicol, 15% sulphacetamide and 1500 iu vitamin A per gram]; or chloramphenicol injections [Chloro-25 injectable (CAPS) containing chloramphenicol 250 mg ml^{-1}], 10 mg subcutaneously in the eyelid.

Fungal Dermatitis

Potassium permanganate in pond water at 10 parts per million.

Coccidiosis

Sulphachloropyrazine [ESB3 water soluble powder (Ciba-Geigy) containing 30% sulphachloropyrazine] in the food at a rate of 1.5 g kg^{-1} for three consecutive feeds, or dosed as a 3% solution at the rate of 5 ml kg^{-1} body weight by stomach tube daily for three days. Disinfection of pond water as for viral hepatitis and enteritis can also be undertaken.

Dujardinascariasis

Fenbendazole [Panacur (Hoechst) containing 10% fenbendazole] mixed in feed at 200 mg kg^{-1} for two consecutive feeds.

Vitamin Deficiency and Food Supplementation

The premix developed for alligators in the U.S. [McNease and Joanen (1981); see Chapter 32 for ingredients], added to food at a rate of one percent by weight.

Runt Syndrome

An homogenised mix of 250 g of fresh whole fish, 250 ml of water and 1 ml of concentrated multivitamin drops [ABIDEC drops (Parke-Davis), each ml of which contains: vitamin A 5000 iu; vitamin D 4000 iu; vitamin B1 1 mg; vitamin B2 0.4 mg; vitamin B6 0.5 mg; nicotinamide 5 mg; and, ascorbic acid 50 mg], dosed by stomach tube at 20 ml kg^{-1} body weight twice a week. Vitamin E-Selenium injections [Injacom E-Selenium (Roche) containing 15% vitamin E acetate and 0.05% selenium], 0.5 ml kg^{-1} intramuscularly at monthly intervals.

FACTORS ASSOCIATED WITH DISEASE

Not only is it unacceptable to lose 30 to 40 percent of farmed crocodiles from disease or managerial deficiencies, it is also impractical to apply therapeutic measures to a large number of sick individuals. It is simply essential to understand the factors that facilitate the appearance of disease and try and minimize their effects.

Clutch-associated Mortality

In order to determine whether or not high mortality was associated with clutch of origin, crocodile farmers were urged to identify hatchlings from each clutch by toe-clipping individuals with a unique sequence, and then recording subsequent mortality against clutch. The results for one year on two different farms are presented in Tables 6 and 7.

On one farm (Sengwa; Table 6) most of the mortality occurred between December and April, before the onset of winter; the majority of deaths were in a relatively small number of clutches. On the other farm (Rokari; Table 7) there was a more even distribution of mortality among clutches and it occurred throughout the year. However, both farms hatched a number of clutches with very poor survival. The eggs and hatchlings from these clutches had no obvious characteristics that allowed them to be identified before or at the time of hatching.

It is unknown if these results reflect genetic predispositions to high mortality or whether they are a consequence of incubation environments in the field or within the farms, or the result of trauma during collection and transport. However, clutches collected earlier in the egg-laying season did tend to have lower mortalities (unpublished data).

Hatching and the Neonatal Period

The stacking of boxes containing incubating eggs in close proximity to each other leads to hatchlings calling between them, which possibly stimulates premature hatching. In some clutches the majority of individuals hatch with large amounts of unabsorbed residual yolk in the abdomen. This provides a good growth medium for bacteria, and the umbilicus provides the usual pathway of entry for it. Maintaining neonatal crocodiles at high and constant temperatures (Joanen and McNease 1976), in an environment where the umbilicus is unlikely to become contaminated (one that is clean and dry) has significantly improved survivorship in Zimbabwe.

Table 6. The relationship between percentage mortality during raising to one year and clutch of origin for *Crocodylus niloticus* at Sengwa Crocodile Farm, Zimbabwe, 1983. Total mortality was 22.1%; 17.4% between December and April, and 4.7% between May and November (autumn and winter).

Mortality (%)	No. of clutches	Eggs hatched	Hatchling Rate (%)	Deaths	% of total deaths
0- 10	40	1529	92.8	61	8.3
11- 20	20	740	93.0	99	13.5
21- 30	5	186	85.3	49	6.7
31- 40	6	171	92.4	53	7.2
41- 50	7	213	86.9	97	13.2
51- 60	3	120	90.2	65	8.9
61- 70	1	32	64.0	22	3.0
71- 80	4	140	95.2	106	14.5
81- 90	1	35	94.6	29	4.0
91-100	5	156	74.3	152	20.7
	92	3322	90.6	733	

Table 7. The relationship between percentage mortality during raising to one year and clutch of origin for *Crocodylus niloticus* at Rokari Crocodile Farm, Zimbabwe, 1984. Total mortality was 29.6%; 11.4% between December and April, and 18.2% between May and November (autumn and winter).

Mortality (%)	No. of clutches	Eggs hatched	Hatchling Rate (%)	Deaths	% of total deaths
0- 10	11	297	84.6	16	3.1
11- 20	14	529	95.8	87	16.6
21- 30	5	176	97.2	44	8.4
31- 40	9	309	98.4	111	21.2
41- 50	8	265	95.7	117	22.3
51- 60	1	44	100.0	24	4.6
61- 70	1	43	97.7	27	5.2
71- 80	–	–	–	–	–
81- 90	1	47	95.9	38	7.2
91-100	3	61	88.4	60	11.4
	53	1771	94.2	524	

If water is provided in neonatal nurseries the addition of tetracycline may also prevent bacterial infection.

Trauma due to piling or premature rupture of the umbilicus, with haemorrhage, has also caused mortality among neonatal crocodiles. Stress from unnecessary handling should be avoided and neonates which are unlikely to survive (grossly premature individuals or ones with obvious deformities) should be removed at hatching.

Hatchling Period

An overview of mortality on all the Zimbabwean farms in relation to time of year, in bi-monthly periods (1980-83) is on Figure 9. Hatchling mortality is greatest during winter and spring (May to September), although this is not the case with rearing stock (1-3 years of age).

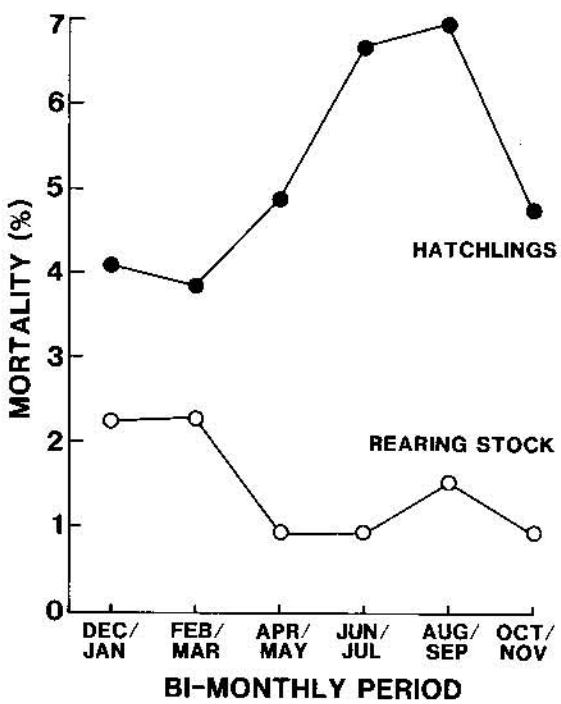

Fig. 9. Mortality of *Crocodylus niloticus* hatchlings (6 weeks to 1 year of age; closed circles) and rearing stock (1-3 years of age; open circles) on crocodile farms in Zimbabwe, as a function of time of year. Data are from the period 1980 to 1983.

The effects of temperature on metabolism and growth in crocodilians has been well documented for *A. mississippiensis* (Joanen and McNease 1976; Coulson and Hernandez 1983) and some other species (see Lang Chapter 30). Maintaining crocodilians under optimal temperature regimes can enhance growth and assist in the control of infectious diseases. It may not always be feasible to raise crocodilians in controlled environment chambers, of the type used with *A. mississippiensis* (Joanen and McNease 1976), but practical measures can be taken to maintain temperatures between 25 and 30°C, and perhaps to provide spot sources of higher temperatures (see Lang Chapter 30), even in outside pens.

At higher temperatures ponds rapidly become unhygienic and an ideal medium for the spread of pathogens. Thus frequent cleaning and water changes are essential. Limited experience indicates that chlorine in the water has no obvious deleterious effects at 2-4 parts per million, and can be used if disease problems occur.

Drawing water and food from sources in which wild crocodiles are resident is an obvious way of introducing diseases. Unfortunately it is unavoidable on most crocodile farms in Zimbabwe, as they are situated in reasonably remote, wildlife areas.

Hatchling pens should be designed with features that minimize the occurrence of piling; there should be adequate feeding space and a method of easy cleaning that avoids stressing the animals unduly. Whilst hatchlings tolerate routine management procedures such as cleaning of pens by familiar handlers, unusual operations like measuring, grading and sorting into new pens can be very stressful.

The dietary requirements of *C. niloticus* are essentially unresearched, so they are not discussed in any depth here. In the farm situation, neonates initially need to be encouraged to take any food which is not alive and moving (particle size is also important). Feeds containing red meat are more

acceptable to them, but fish (lake sardines) appear to give better growth in hatchlings and should provide a more balanced diet. Prolonged use of a single source of food, especially red meat, is likely to lead to deficiencies. Vitamin A is relatively unstable, especially at high temperatures, and a deficiency may occur even when it is supplemented. Insufficient food intake, particularly during winter, and improper mixing of vitamins, may also lead to deficiencies.

Fresh food is the most acceptable diet, but for practical reasons food has to be stored and this should be frozen rather than chilled. It has not yet been demonstrated that food is a source of infectious disease, although some evidence suggests *Dujardinascaris* infestation may result from feeding freshly netted fish from Lake Kariba. Overfeeding can cause gout, and there are dangers in feeding when a crocodile's metabolic rate has been affected by low temperatures. Uneaten food left on pen floors or in the water is obviously unhygienic and may enhance disease.

Sick crocodiles should be removed to isolated "hospital" pens (even if treatment is not contemplated) or killed and disposed of. Even recovered individuals may remain a source of infection to others and the question of re-introducing them into rearing pens must be considered carefully. Runt crocodiles, even in the absence of infectious disease, require special care in terms of both housing and feeding.

CONCLUSIONS

Only in Zimbabwe has disease been reported as having a serious effect on the viability of crocodilian farming operations. This is in no way an indictment of the crocodile farmers of Zimbabwe, who for the most part have had immense logistical and other problems to deal with. It may also reflect a lack of data on the extent of disease related problems in other countries, many of which have only recently started farming crocodilians (for example in Australia; see Onions Chapter 34). However, regardless of these possibilities, 5% mortality would be acceptable — 30-40% is not.

The reasons for high mortality within crocodile farms in Zimbabwe may be numerous and interrelated, but are likely to include the following:

1. Problems associated with egg collection and incubation;
2. Insufficient attention to the neonatal period;
3. Impractical pen design and inadequate temperature control;
4. Incorrect feed and feeding practices;
5. Poor hygiene;
6. Delays in responding to obvious disease problems; and,
7. Farming crocodiles in close proximity to wild populations.

It has not yet been possible to provide recommendations backed by adequate research on all these factors, and where problems have been solved to date, it has usually been by trial and error or by the enterprise of individual operators. However, without these advances, disease may well have rendered the commercial production of crocodiles in Zimbabwe totally impossible.

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