THE DIGESTIVE TRACT OF THE WHITEBACK GRIFFON VULTURE AND ITS ROLE IN DISEASE TRANSMISSION AMONG WILD UNGULATES

Author: HOUSTON, D. C.

THE DIGESTIVE TRACT OF THE WHITEBACK GRIFFON VULTURE AND ITS ROLE IN DISEASE TRANSMISSION AMONG WILD UNGULATES

D. C. HOUSTON,* Serengeti Research Institute and Edward Grey Institute

J. E. COOPER,** Veterinary Research Laboratory, Kabete, Kenya.

Abstract: The digestive tract of the whiteback vulture (*Gyps africanus*) is described. Some disease organisms were fed to a captive bird to discover if they could survive passage through the tract, and the role of these scavenging birds in the spread of diseases among wild ungulates is discussed.

INTRODUCTION

The food of griffon vultures has been found to consist almost exclusively of the muscle and viscera from carcasses of large ungulates. They obtain only a very small proportion of their food from predator kills and most of the animals on which vultures feed have died from malnutrition, disease or accidents.[10,11] Vultures therefore must frequently feed from animals which have died from infectious disease. During food searching these birds regularly travel over large distances, sometimes flying 240 km or more between nest site and the feeding area, and marked birds have been recovered over 1,100 km from their ringing site.[13] It is possible therefore that they could be important disseminators of some disease organisms among grazing animals, and vultures and other scavenging animals have often been suspected of dispersing diseases, such as anthrax,[4,15,19,20] tuberculosis[24] and brucellosis,[21] and of introducing disease to domestic stock.

For a vulture to play such a role in disease transmission the organism must be able to do one of the following:

a) Cause clinical or sub-clinical infection in the vulture and be passed out in the bird's secretions or excretions, or spread by vectors.

b) Be transmitted mechanically on the bird's feet or feathers.

c) Be regurgitated with pellets from the vulture's crop.

d) Pass through the vulture's alimentary tract and be voided in the faeces.

The first mentioned (a) will not be discussed here. Little is known of diseases of vultures apart from isolated cases from zoological collections and a few other reports,[5,16] but this is unlikely to be an important method of spreading diseases of ungulates. The second method, by transmission of pathogens on feet and feathers can undoubtedly occur. But probably the most important potential method of disease dissemination comes from organisms which can survive in the digestive tract so that viable pathogens are spread by the bird's faeces or regurgitated pellets.

Despite the interest in the possibility of spread of disease by vultures, very little experimental work on the subject has been carried out.[2,24] This paper firstly reports some observations and experiments on the alimentary tract and digestion of the whiteback griffon vulture. It then discusses the role scavenging birds

---

* Present address: Zoology Department Glasgow University, Glasgow, Britain.
**Present address: Clinical Research Centre, Watford Road, Harrow, Middlesex, Britain.

may play in the spread of pathogenic organisms. The field observations were made incidentally to a study of the feeding ecology of vultures in the Serengeti National Park from 1968 to 1970.

METHODS

Several vultures were shot during a study of the body condition of these birds and the opportunity was taken to examine their digestive tracts. Rectal swabs were taken from these birds, stored in transport agar and conveyed to the Veterinary Research Laboratory at Kabete for aerobic and anaerobic bacterial culture. Intestinal parasites were removed and identified. Several birds, which were digesting food when shot, had the pH values of portions of their gut determined within 3 min of death by narrow range pH indicator papers. The tracts of several birds were also measured. One tract was used for an histological examination, sections being cut and stained with haemotoxylin and eosin. Three hand reared captive birds were used to record food intake, and the minimum time required for the passage of food through the digestive tract was estimated by feeding meat injected with a heavy suspension of carmine particles and recording the time interval before red carmine became visible in the bird's faeces. One of these captive birds was taken to Kabete where transmission studies could be made. For two weeks prior to the experiments this captive bird's faeces were cultured regularly to determine the bird's 'normal' intestinal flora. Bacterial broth cultures (containing up to 2×10^6 bacteria) were then fed in pieces of meat and faecal samples were then taken from the bird at intervals of approximately 6 hours during the first 24 hours and at 24 hourly intervals thereafter and these were cultured and examined for viable bacteria.

RESULTS

a) Digestive tract structure

The exclusively meat diet of the griffon vulture requires a relatively unspecialized alimentary tract for digestion. Food is torn from a carcass by the sharp edges of the bill and rapidly swallowed with the aid of the horny, backward pointing serrations on the edge of the tongue. The oesophagus leads into a simple crop, which is a thin walled, distensible extension of the oesophagus. Food passes from the crop into a muscular stomach, which is highly distensible. There are two parts to the stomach, an anterior tubular proventriculus and a sac-like gizzard. In the proventriculus chemical digestion commences and the food is passed as a yellowish chyme past the pyloric sphincter into the duodenum and small intestine. The small intestine leads into the large intestine, where there are two caeca, which are small as in most carnivorous birds. The large intestine is short and terminates in the cloaca. In table 1 the lengths of the various sections of the alimentary tract are shown.

No literature has been traced on the histological appearance of the digestive tract of the whitebacked vulture and the following notes are taken from microscopical examination of a series of tissues of one freshly killed whiteback vulture.

The *buccal cavity* is covered with a thick layer of stratified squamous epithelium. The submucosa is very vascular and contains mucous glands but there is no muscularis mucosae. There are distinct keratinised outgrowths (papillae) on the tongue.

The *oesophagus* is also composed of an inner mucosa of stratified squamous epithelium which shows a thin layer of keratinisation and some desquamated cells. There is considerable corrugation of the mucosa, the oesophagus being distensible in order to accommodate large amounts of food. The submucosa is thin and contains numerous large submucous glands. The muscularis mucosa is relatively thick and consists of both longitudinal and circular layers, as does the external muscle layer.

The *crop* is structurally the same as the oesophagus and has been described elsewhere[6] but there are less mucous

glands. This is in contrast to the chicken where such glands are absent.[3]

The *proventriculus* is lined with columnar epithelial cells and is highly glandular. There are many folds in the proventricular wall and tubular glands empty into gastric pits between these folds. Other glands empty directly into the lumen of the proventriculus. There are scattered foci of mononuclear (lymphocyte type) leucocytes in the deeper layers of the mucosa. The submucosa is very vascular but relatively acellular. The proventricular muscles consist of a thick layer of circular muscle fibres exterior to which is a thin (10% of the thickness of the circular) layer of longitudinal fibres. The serosa on the outside of the proventriculus, gizzard and intestine is composed of a thin layer of simple squamous epithelium.

The *gizzard* (or ventricle) is poorly developed in the vulture as compared with herbivorous birds. It is lined with columnar epithelium and many tubular glands are present. In other birds a layer of hard horny material (koilon) may be present on the mucosal surface, but in the whiteback vulture this appears to be absent and only a thin layer of cellular debris separates the mucosa from the lumen of the gizzard. The submucosa is thin but contains a circular layer of fibrous tissue (the stratum compactum). The outlying circular muscle layer is thick and the longitudinal thin, as in the proventriculus.

The small intestine commences with the duodenum which is a loop of intestine just distal to the gizzard. The structure of this and the rest of the small intestine is similar and a jejunum and ileum cannot be differentiated. The mucosa is raised into villi which are lined with columnar epithelium containing many goblet cells. Extensive tubular glands empty into the crypts of Lieberkuhn (as in the mammal). The villi are densely infiltrated by many mononuclear (lymphocyte type) leucocytes, with distinct lymphocytic nodules. There are thin strands of fibrous tissue within the villi and at the bases—a thin, yet distinct, stratum compactum. The muscularis mucosae is well developed. There are distinct circular and longitudinal muscle layers, outside which is the serosa.

The *caeca and large intestine* appear identical histologically. They closely resemble the small intestine but the villi are more flattened and less numerous. There is no distinction between colon and rectum.

The wall of the *cloaca* shows a transition from a columnar epithelial lining (with lymphatic and glandular tissue present) to the stratified squamous epithelium of the peri-cloacal skin.

TABLE 1. Length and pH of the digestive tract of whiteback vulture.

Part of Tract	Number examined for length	Mean length cm	pH of tracts in which food was being digested
Oesophagus	1	35	—
Crop	—	—	7, 7.5
Stomach	1	16	1, 1.2, 1.5
Duodenum	3	61	6, 6.5, 6, 6
Small intestine	3	190	6, 7.5, 6-7
Caecum	2	0.85	—
Rectum	3	7.7	6.5

There are no villi in the anterior portion of the cloaca but its wall has raised symmetrical corrugations which allow for considerable distension.

b) pH values in the tract

The pH of the various portions of tract examined are given in Table 1. In the crop, duodenum, small intestine and rectum the pH was around neutral, but in the stomach it was extremely acidic, the lowest recorded being pH 1.0. No distinction was made between proventriculus and gizzard.

c) Rate of digestion

Whiteback vultures fed with meat infected with a heavy suspension of carmine particles took from 5½ to 6 hours before the first faeces containing carmine appeared. Up to 1,200 g of food was stored in the crop and it took about 28 hours before all this food had passed into the stomach. Faeces would therefore probably continue to be produced until at least 34 hours after a large meal. A slightly larger griffon vulture, the Ruppell's griffon, Gyps ruppellii, was found to take from 6 to 7½ hours before the first faeces appeared after feeding.

d) Bacteriology

Rectal swabs were taken from 12 vultures which were shot, and they yielded Escherichia coli in all cases. The only other bacteria isolated were Citrobacter freundii in two cases and Proteus vulgaris in another.

e) Parasites

Eighteen birds were examined in detail for internal parasites. Several nematodes, Porrocaecum sp. were present in 16 of these birds, and a small cestode, species unknown, was found in one bird.

f) Experimental feeding

For 2 weeks prior to these feeding trials the birds' faeces were regularly cultured. A pure culture of non-haemolytic E. coli was recovered on each occasion, and this organism, too, was invariably cultured during the course of the experiments. The bacteria used were as follows:

Bacteria ingested	Bacteria recovered in faeces
Aeromonas formicans Gram-negative bacillus	Not recovered
Streptococcus pyogenes Gram-positive coccus	Recovered up to 72 hours after ingestion
Bacillus anthracis Avirulent strain of the gram-positive bacillus which causes anthrax	Not recovered
B. anthracis spores From commercial anthrax vaccine 'Blanthrax': Burroughs Wellcome	Recovered after 5½ hours, but not after 17 hours

DISCUSSION

The anatomical and pH studies indicate how well adapted the digestive tract of the whiteback vulture is to the bird's role as a scavenger. Such features as the sharp bill, barbed tongue and thick stratified squamous epithelium of the buccal cavity and oesophagus enable the rapid swallowing of large food items, while the large crop permits a considerable quantity of food to be swallowed. When fully distended the crop will hold about 1,200 g of food, about 20% of the body weight, which is sufficient food to maintain the bird for 3-4 days. Mechanical breakdown of food does not occur at any stage in the digestive tract. Food is broken down entirely by chemical action and this occurs under very low pH conditions in the stomach. In the cormorant, Phalacrocorax carbo, the pure gastric juice has been found to have a pH of the order

of 1-2[25] and this may be general for carnivorous birds.[8] However, the pH of digesting stomach contents in the heron, *Ardea cinerea*, kestrel, *Falco tinnunculus*, and barn owl, *Tyto alba*, are from 2.5-5.0,[17] and not as low as the 1-2 found in the griffon vulture. These highly acidic conditions of the stomach probably contribute to the breakdown of food and permit rapid digestion and, as Mennega[17] has emphasized, the high body temperature of birds also aids digestion.

The normal bacterial flora of the vulture's intestinal tract appeared to be largely gram-negative bacilli of the family Enterobacteriaceae, especially *E. coli*. This would be expected in carnivorous birds, since the majority of their intestinal flora is probably derived from their prey. These bacteriological findings agree with those for other East African members of the Falconiformes (Cooper, unpublished data) where *E. coli* is the usual bacterial flora but other organisms, including *Citrobacter* and *Proteus* spp. are encountered from time to time.

Experimentally fed cultures of a *Streptococcus* survived passage through the tract, a not unsurprising result in view of the fact that a number of this genus of bacteria can live in the intestine (e.g., *S. faecalis* and *S. viridans*) and have been cultured previously from the faeces of falconiform birds (Cooper, unpublished data). It is possible that the *Streptococcus* multiplied in the intestinal tract since red carmine normally ceased to be passed in the faeces after 24 hours and yet the *Streptococcus* was retrieved after 72 hours. The presence of *E. coli* as a member of the normal intestinal flora of the whiteback vulture does not necessarily indicate that this bacteria can also survive passage through the stomach since this bacterium might be established in the intestine of vultures when very young, when their stomach pH might not be as low as that of adult birds. Judging by our experience with *A. formicans*, possibly *E. coli* ingested by an adult bird would not reach the intestine in a viable condition.

The vegetative form of *B. anthracis* was found to be killed by passage through the digestive tract, although as might be expected the highly resistant spores survived. Urbain and Nouvel[24] reported that anthrax survived passage through the digestive tract of the hooded vulture *Necrosyrtes monarchus* and Blancou and Rajaonarison[2] reported survival through the tract of a buzzard, *Buteo brachypterus*, although it is not known if vegetative stages or spores of the bacteria were used.

It was found that *A. formicans* did not survive passage through the tract. This bacterium is very similar in its range of tolerance to Salmonella and was used as a model since *Salmonella* was considered a possible health threat to those working with the bird.

The role of vultures in spreading disease among wild ungulates

The role of disease in the regulation of wild ungulate populations in Africa has received very little attention. Unfortunately information on diseases of ungulates is often collected by veterinarians in a way that is of little use to ecologists, and ecologists frequently fail to take the opportunity to include information on disease in their studies. The impact of any disease on a wild animal population depends on the speed with which it can be disseminated. Scavenging animals could be a major factor in determining the speed with which a disease can spread. Among the scavenging animals of the African savanna all the mammalian scavengers, such as lion and hyaena, have restricted feeding ranges and it is only the avian scavengers which can travel large distances.[10,11] Among the scavenging birds there are seven species of vulture, some of which are resident in one area and it is only the griffon vultures which occur in very large numbers and which regularly travel large distances during their feeding.[12] These are therefore potentially the most effective disseminators of disease organisms.

Disease producing organisms ingested from an infected carcass may be spread by the digestive tract of vultures either through regurgitated food items or through the faeces. Regurgitation of pellets from the crop of vultures occurs only

occasionally in the wild, since indigestible food items, such as pieces of grass or hair, are only rarely swallowed in sufficient quantities to form a pellet—smaller amounts apparently passing through the digestive tract. Since the pH of the crop is almost neutral a number of organisms may remain viable there. Bot fly larvae from ingested zebra intestines have been found attached to the crop wall of vultures and have survived there long enough to elicit an inflammatory reaction.[6] Other organisms might similarly survive for short periods and then be voided later; however, this must be a very infrequent occurrence.

Almost all food, once swallowed, passes through the alimentary tract. The duration of the passage of food is relatively short, taking about 6 hours, so that although any digested organisms are subjected to adverse conditions, this exposure is for a limited period only. To survive passage through the tract any ingested organisms must be able to tolerate exposure to extremely low pH in the stomach, but there are also many other factors which will influence the survival of microorganisms, including their ability to withstand digestive enzymes, the rapid changes of pH both on entering the stomach and on entry to the duodenum, the density of organisms in the food and the extent to which they are protected from these conditions by the presence of ingesta. There is very little information on the effect of these various factors and so we can only make very tentative conclusions on the role of vultures in disease transmission. We know that vultures can continue to pass food through the tract until at least 35 hours after feeding, and one bacterium (*S. pyogenes*) which we fed to a bird was present in faeces up to 72 hours later. Pennycuick followed a griffon vulture in a glider for a distance of 75 km at an average speed of 47 km per hour.[18] Birds could therefore travel considerable distances before completing digestion of their food and any organism that could survive passage through their tract could be disseminated very effectively.

From the limited data available on the resistance of viruses, it would seem unlikely that many could survive a pH of 1-2, however brief the period of passage. Foot and mouth disease virus, for example, is extremely sensitive to slight change in pH.[1] Some of the most virulent diseases recorded in wild ungulate populations are caused by viruses. Rinderpest has caused devastating mortality among some ungulate species in the past, and as recently as 1961 caused large scale mortality of wildebeest calves.[23] An allerton type virus has caused extensive mortality of buffalo in the Serengeti.[22] Other viruses may be present in wild animal populations, although they may not necessarily cause clinical signs, and a recent survey has found antibodies to malignant catarrhal fever to be consistently present in wildebeest and some hartebeest, and antibodies to other viral diseases of importance to livestock, such as foot and mouth disease, bovine viral diarrhoea, bluetongue, rift valley fever, Nairobi sheep disease and African horse sickness.[9] It seems probable that these organisms would all be killed if eaten by griffon vultures.

Among bacterial infections, the organisms which could survive passage would probably be confined to those which have strongly resistant spores. Anthrax has been recorded in many antelope species.[19,20] The vegetative form of *B. anthracis* has been found to be killed in the tract, although the spores survive. However, *B. anthracis* does not form spores until exposed to the air for several hours and usually only under certain environmental conditions. Antelope species do not always bleed from the mouth after death from anthrax infections (Houston unpublished data) and many carcasses located by vultures will contain only vegetative forms of the bacillus. A group of griffon vultures usually feed extremely rapidly, and can completely remove the soft tissues from a Thomson's gazelle (weighing 20 kg) within about 8 min and a yearling wildebeest (100 kg) in about 30 min. In this way vultures must consume and destroy large numbers of bacteria before they have

formed resistant spores. Only a small proportion of their food comes from carcasses that have previously been fed upon by other scavengers and where there would be an opportunity for spore production.

Vultures must act in a similar way to reduce greatly the sources of infection from carcasses containing many other pathogens. *Brucella abortus* has been found to be widespread among some ungulate population.[9,21] This bacterium shows a degree of resistance to pH changes but would probably not survive passage through the stomach. Salmonellosis was probably the main cause of a 50% mortality among wildebeest calves in the Kruger National Park[14] and it is likely that this bacterium would be killed in the stomach.

Intestinal parasites, such as nematodes and cestodes, can obviously survive passage through the stomach in their infective stages. Griffon vultures could harbour parasites with intermediate hosts among ungulate species and so spread parasitic infections in their faeces. However, parasite burdens were found to be extremely light, and the intermediate

hosts of the few species recorded are unknown.

Finally, it must be remembered that pathogenic organisms could be carried on the birds' feet and feathers. Foot and mouth disease virus can survive for several hours on the feet of migrating birds[7] and there is therefore little doubt that highly resistant organisms, such as anthrax spores, could persist in this way. Griffon vultures often bathe and preen after a meal to clean their feathers, and this could result in the contamination of a lake or water hole.

The digestive tract of griffon vultures is likely to destroy all except the most resistant of organisms, and vultures must thereby reduce greatly the source of infection from carcasses of animals which have died from disease. However, some organisms might be carried passively on the birds' feet or feathers, and a few highly resistant species could survive passage through the gut; and so although vultures might help to reduce the spread of most infections in a locality, they could also introduce pockets of infection into new areas. The effect of vultures on disease transmission is therefore complex.

LITERATURE CITED

1. ANDREWS, C. and H. G. PEREIRA. 1967. *Viruses of Vertebrates*. 2nd ed. Bailliere, Tindall and Cassell, London.
2. BLANCOU, J. and J. RAJAONARISON. 1972. Notre sur le rôle vecteur des rapaces dans la propagation de certaines maladies bactériennes. Rev. Elev. Méd. vét. Pays. trop. 25: 187-189.
3. BRADLEY, O. C. 1950. *The Structure of the Fowl*. Oliver and Boyd, London.
4. BULLOCK, D. S. 1956. Vultures as disseminators of anthrax. Auk 73: 283-284.
5. COOPER, J. E. 1972. *Veterinary Aspects of Captive Birds of Prey*. Hawk Trust, Newent, Gloucestershire, England.
6. COOPER, J. E. and D. C. HOUSTON. 1970. Lesions in the crop of vultures associated with bot fly larvae. Trans. R. Soc. trop. med. Hyg. 66: 515-516.
7. ECCLES, A. 1939. The role of birds in the epizootiology of foot and mouth virus. Bull. Off. int. Epiz. 18: 118-148.
8. FARNER, D. S. 1961. Digestion and the digestive system. In *Biology and Comparative Physiology of Birds*, Vol. 1. London and New York.
9. FAY, L. D. 1972. *Report to the Government of Kenya on Wildlife Disease Research*. No. TA 3049, FAO, Rome, Italy.
10. HOUSTON, D. C. 1973. *Ecology of Serengeti Vultures*. D.Phil. thesis. Oxford.
11. ————. 1973. The role of griffon vultures as scavengers. J. Zool. 172: 35-46.

12. ————. 1973. Food searching in griffon vultures. E. Afr. Wildl. J. 12: 63-77.

13. ————. 1973. Mortality in the cape vulture (*Gyps coprotheres*). Ostrich 45: 57-62.

14. JANSEN, B. C. 1968. *A Practical Guide to the Study of the Productivity of Large Herbivores*. Editors, Golley and Buechner. Blackwells, Oxford.

15. KHANEVELD, F. C. and M. MANSJAER. 1941. The spread of anthrax by carrion eaters. Med. int. Bl. Diergeneesk, 53: 313-340.

16. KEYMER, I. F. 1972. Diseases of birds of prey. Vet. Rec. 90: 579-594.

17. MENNEGA, A. M. W. 1938. Waterstofionenconcentratie en vertering in de maag van eenige vertebrated. Dissertation, Rijks-Universiteir, Utrecht.

18. PENNYCUICK, C. J. 1972. Soaring behaviour and performance of some East African birds, observed from a motor-glider. Ibis 114: 178-218.

19. PIENAAR, U. de V. 1960. Uitbraaj vab miltsiehte onder wild in die Nasionale Kruger Wildtuin. Koedoe 3: 238-251.

20. ————. 1961. A second outbreak of anthrax among game animals in the Kruger National Park. Koedoe 4: 4-14.

21. SACHS, R., C. TAAK and C. M. GROOCOCK. 1968. Serological investigations of brucellosis in game animals in Tanzania. Bull. epizoot. Dis. Afr. 16: 93-100.

22. SCHIEMANN, B., W. PLOWRIGHT and D. M. JESSETT. 1971. Allerton type herpes virus as a cause of lesions of the alimentary tract in a severe disease of Tanzanian buffaloes. Vet. Rec. 89: 17-22.

23. TALBOT, L. M. and M. H. TALBOT. 1963. Wildebeest in western Masailand. Wildlife monographs no. 12.

24. URBAIN, A. and J. NOVEL. 1946. Spread of tuberculosis and anthrax by carnivorous birds. Bull. Acad. vet. Fr. 19: 237-239.

25. Van DOBBEN, W. H. 1952. The food of the cormorant in the Netherlands. Ardea 40: 1-63.