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Received: 22 July 2020 | Revised: 6 May 2021 | Accepted: 22 May 2021

DOI: 10.1002/ajp.23290

RESEARCH ARTICLE

AMERICAN JOURNAL OF PRIMATOLOGY WILEY

Pathological lesions of the digestive tract in free-ranging mountain gorillas (*Gorilla beringei beringei*)

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Funding information

Mountain Gorilla Veterinary Project Inc.

Abstract

The finding of parasites and bacterial pathogens in mountain gorilla feces and oral lesions in gorilla skeletal remains has not been linked to pathological evidence of morbidity or mortality. In the current study, we conducted a retrospective study of digestive tracts including oral cavity, salivary glands, esophagus, stomach, intestines (gastrointestinal tract [GI]), liver, and pancreas of 60 free-ranging mountain gorillas from Uganda, Rwanda, and the Democratic Republic of Congo that died between 1985 and 2007. We reviewed clinical histories and gross pathology reports and examined histological sections. On histology, enteritis (58.6%), gastritis (37.3%), and colitis (29.3%) were the commonest lesions in the tracts. Enteritis and colitis were generally mild, and judged likely to have been subclinical. Gastritis was often chronic and proliferative or ulcerative, and associated with nematodiasis. A gastro-duodenal malignancy (carcinoid) was present in one animal. A number of incidental lesions were identified throughout the tract and cestodes and nematodes were frequently observed grossly and/or histologically. Pigmentation of teeth and tongue were a common finding, but periodontitis and dental attrition were less common than reported from past studies of skeletal remains. Despite observing numerous GI lesions and parasites in this study of deceased free-living mountain gorillas, we confirmed mortality attributable to gastroenteritis in just 8% (5/60) cases, which is less than that described in captive gorillas. Other deaths attributed to digestive tract lesions included cleft palate in an infant, periodontal disease causing systemic infection in an older adult and gastric cancer. Of all the parasitic infections observed, only hepatic capillariasis and gastric nematodiasis were significantly associated with lesions (hepatitis and gastritis, respectively). Understanding GI lesions in this endangered species is key in the management of morbidity associated with GI ailments.

KEYWORDS

gastrointestinal lesions, great apes, nonhuman primates, parasitism, pathology

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1 | INTRODUCTION

The mountain gorilla (*Gorilla beringei beringei*), whose range is restricted to the Virunga Massif shared by Rwanda, Uganda and Democratic Republic of Congo (DRC) (Virungas) and the Bwindi Impenetrable Forest of Uganda (Bwindi), are classified as endangered in the IUCN Red List of Threatened Species™. Before this, for about two decades, the species was classified as critically endangered (Hickey et al., 2018). Presently, there are only about 1063 mountain gorillas left in the world, including an estimated 459 in Bwindi (WWF, 2019) and an estimated 604 in the Virungas (Hickey et al., 2019).

Since 1986, the Mountain Gorilla Veterinary Project (MGVP), now Gorilla Doctors, in partnership with the University of California Davis, has contributed to the conservation of mountain gorillas and their ecosystem through a One Health approach (Cranfield, 2008). In addition to providing veterinary care in situ to ill and injured human-habituated gorillas, Gorilla Doctors' veterinarians conduct necropsies according to established postmortem protocols, and a board-certified veterinary pathologist (L. J. L.) conducts histopathology examinations (refer to Table 1 for the glossary on terminologies used for clarity).

While numerous studies on the fecal parasites and bacteria in this species have been conducted (Graczyk et al., 2002; Kalema-Zikusoka et al., 2005; Muhangi, 2003), including a study that looked at the health implications of coprophagy for parasitism (Graczyk & Cranfield, 2003), limited information is available on the pathological effects of the bacteria and parasites identified in the feces of free-ranging mountain gorillas. Gastrointestinal disease is an important cause of morbidity in zoo-housed lowland gorillas (Strong et al., 2017). Similarly, though lesions suggestive of periodontal disease and dental abscesses have been found in studies of mountain gorilla skeletal remains, the impact of dental disease on the health of individual mountain gorillas is unknown (Watts & Pusey, 1993). The purpose of this study was to retrospectively review clinical records, postmortem reports, and archived histological sections of the gastrointestinal tract (GI), oral cavity, salivary glands, liver, and pancreas from free-ranging mountain gorillas that died between 1985 and 2007 with the aim of better understanding the role of oral lesions, liver and pancreatic lesions, gastrointestinal disease and parasitism in morbidity and mortality in this species. Because of the large array of parasites and potentially pathogenic bacteria that have been identified in fecal examinations of free-living mountain gorillas, it is probable that these parasites and enteric bacteria have co-evolved with these free-ranging mountain gorillas. We, therefore, hypothesized in the null that lesions associated with these parasites and enteric bacteria would not be expected to cause morbidity and mortality. Secondly, previous studies of skeletal remains have demonstrated evidence of periodontal disease and we envisaged that postmortem examinations would uncover evidence of oral lesions. We, therefore, hypothesized in the null that the oral lesions are not associated with morbidity and mortality. The generated data would produce the much-needed information needed in the management of morbidity with GI involvement. This would contribute to the conservation efforts for this endangered great ape species, a population limited to an area where the study has been carried out.

2 | METHODS

2.1 | Gross reports review and histopathology

This study was retrospective and utilized mountain gorilla tissue samples and reports archived, with the permission of the Rwanda Development Board, at the University of California (UC), Davis, School of Veterinary Medicine, William R. Pritchard Veterinary Medical Teaching Hospital (VMTH) to determine the prevalence and clinical significance of digestive tract lesions in free-ranging mountain gorillas. All included gorillas had died and tissues were collected postmortem as part of the health surveillance program of the Mountain Gorilla Veterinary Project (Gorilla Doctors) conducted with the permission of the range country authorities. Tissues were brought to the VMTH with appropriate export and import permits required by the Convention on International Trade in Endangered Species of wild fauna and flora (CITES) and US Public Health Service. Because of the diagnostic nature of the samples, no Institutional Animal Care and Use Committee (IACUC) review was required. The study adhered to the American Society of Primatologists ethical principles for the treatment of nonhuman primates. Animals from both the distinct populations in Virungas and Bwindi, were included. Most data were from gorillas who lived in family groups habituated to the presence of people for research and tourism, and many were animals with a long history of human observation. We reviewed 89 pathology records for mountain gorillas that died between 1985 and 2007; however, complete gross postmortem reports were available for only 55 individuals, and histological sections from the digestive tract from only 60 individuals (some of which did not have corresponding gross postmortem reports) were available for gross and histopathological review, respectively, and inclusion in this study. The gorillas were classified based on age as neonates (1 month old or less), infants (>1 month to 3.5 years), juveniles (>3.5 to 6 years), subadults (>6 years to sexual maturity), and adults (>10 years for females and >13 years for males) according to established criteria (Watts & Pusey, 1993).

The histological sections for all the individuals were archived at UC Davis, except for a set of tissues from one Bwindi gorilla that was held and reviewed at Makerere University, College of Veterinary Medicine, Animal Resources and Biosecurity (COVAB). Gross postmortem findings had been entered onto standardized forms developed specifically for MGVP/Gorilla Doctors. For histology, tissue samples had been collected into 10% (generally, neutral buffered) formalin and shipped to UC Davis following acquisition of the appropriate permits as stated above. This was followed by routine processing, wax embedding, and sectioning at 5 μm. Sections were stained routinely with hematoxylin and eosin (H&E) and special stains (e.g., Brown & Brenn stain for bacteria and Prussian blue for

TABLE 1 Glossary of used terminologies

Terminology	Meaning
-itis	A suffix denoting inflammation
Autolysis	Breakdown of all or part of a cell or tissue by self-produced enzymes usually beginning after death
Capillariasis	Infestation with or disease caused by nematode worms of the genus *Capillaria*
Cestodes, cestodiasis	Tapeworms, condition of having tapeworms
Cholecystitis	Inflammation of the gall bladder
Colitis	Inflammation of the large intestine (colon)
Dochitis	Inflammation of a duct, for example, choledochitis = inflammation of a/the bile duct
Enteritis	Inflammation of the small intestines
Esophagitis	Inflammation of the esophagus
Fibrosis	A condition marked by an increase of interstitial fibrous tissue in a given tissue, organ
Gastritis	Inflammation of the stomach
Gingival regression	An instance of regressing by the gums
Gingivitis	Inflammation of the gums
Glossitis	Inflammation of the tongue
Granulocytes	Neutrophils and eosinophils indicating either acute inflammation (neutrophils) or a reaction to allergens or parasites
Granuloma	A mass or nodule of chronically inflamed tissue with granulations that is usually associated with an infective process
Gross	Visible without the aid of a microscope
Hepatitis	Inflammation of the liver
Histiocytes, histiocytosis	Mononuclear tissue inflammatory cells capable of ingesting and killing pathogens
Histological sections	Tissues sections prepared for examination under the microscope
Histopathology	The study of microscopic changes
Inanition	Signs and effects of starvation
Incidental	A process found during an examination that is not the main problem
Inflammation	A local response to cellular injury that is marked by capillary dilatation, leukocytic infiltration, redness, heat, and pain and that serves as a mechanism initiating the elimination of noxious agents and of damaged tissue
Lesion(s)	Abnormality, pathological change(s)
Lithiasis	Presence of concretions or stones
Morbidity	The condition of suffering from a disease
Mortality	Death
Mucosa	A membrane rich in mucous glands, specifically one that lines body cavities and passages
Nematodes, nematodiasis	Elongated cylindrical worms parasitic in animals (as used in this study), infestation with nematodes
Pancreatitis	Inflammation of the pancreas

(Continues)

TABLE 1 (Continued)

Terminology	Meaning
Periodontitis	Inflammation of the supporting structures of the teeth and especially the periodontal membrane
Sialoadenitis	Inflammation of a salivary gland
Subclinical	A process that does not result in overt symptoms but which may impact health
Typhlitis	Inflammation of the cecum

Source: https://www.merriam-webster.com › medical.

iron) were used when deemed necessary (Bancroft & Gamble, 2008). Sections of digestive tract tissues (oral cavity/tongue, salivary glands, esophagus, stomach, small and large intestine including cecum and appendix, liver, and pancreas) were examined by two of the authors (D. M. and L. J. L.). Identification of parasites observed in histologic sections was performed using a standard key and texts (Chitwood & Lichtenfels, 1973; Gardiner & Poynton, 1999; Gardiner et al., 2001) with verification by one of the authors (C. H. G.). Evaluation of the severity of the lesions and their contribution to illness and death was subjective, but was based on clinical history, gross postmortem findings, and histopathology evaluated through the lens of known principles of pathology and of one of the authors' (L. J. L.) extensive experience in primate pathology. Conceptualization of the study, execution and writing up was done by all the seven authors (D. M., L. J. L., C. H. G., L. O., M. R. C., K. V. K. G., and A. B. M.).

2.2 | Statistical analysis

The results were entered into an Excel™ spreadsheet (Microsoft Corporation) and the prevalence of different kinds of lesions were computed. To determine if the presence of parasites was correlated with observed lesions, we performed a logistic regression comparing the presence of gastritis, enteritis, hepatitis, or fibrosis with capillariasis and other parasite infections (nematodiasis and cestodiasis) to establish any relationship between lesions and parasitism using both SAS Version 9.13 (SAS Institute Inc.) and R Version 3.2.4 (R Core Team, 2016). Statistical significance was set at a 5% level of significance ($p < 0.05$).

3 | RESULTS

3.1 | Age class distribution

Of the 60 gorillas for which histological sections were available for study, four (6.7%) were neonates (1 month old or less), 25 (41.7%) were infants (>1 month to 3.5 years), three (5%) were juveniles (>3.5 to 6 years), three (5%) were subadults (>6 years to sexual maturity), and 25 (41.7%) were adults (>10 years for females and >13 years for males).

3.2 | Gross lesions

Based on gross postmortem reports from 55 gorillas, gorillas exhibited a number of macroscopic digestive tract lesions (Table 2). In the oral cavity, the most prevalent finding was pigmentation of teeth and tongue in 11 (20%) gorillas (Figure 1). Dental disease (mainly attrition) or periodontal disease (regression or infection) were less frequent (four each, 7.3%). The youngest individual exhibiting dental disease at the time of death was a juvenile male (about 5.5 years old) with bilateral gingival regression, necrosis, and feed impaction between premolar 2 and molar 1. Periodontitis was severe in only one geriatric female and was associated with mandibular osteomyelitis and cardiac valvular endocarditis. In four individuals, all adults, in which the teeth were extensively worn (dental attrition), three were emaciated, but also had chronic infections or cancer, which could account for weight loss. Cleft palate was noted in two neonates (3.6%). Dental malocclusion was reported in one (1.8%) adult female. Esophageal lesions were rarely described ($n = 4$, 7.2%).

In several gorillas, the gastric mucosa was reddened due to congestion or hemorrhage ($n = 11$, 20%), sometimes coated with excess mucus ($n = 10$, 18.2%) and occasionally ulcerated ($n = 4$, 7.3%), necrotic ($n = 1$, 1.8%), or nodular ($n = 2$, 3.6%). Nodular thickening and fibrosis of the pylorus and proximal duodenum were extensive in one animal with cancer. In the small intestines, cestodes were evident in 16 (29.1%) gorillas and nematodes in four (7.3%), and the intestinal mucosa in 13 (23%), and/or serosa in four (7.3%) were often reddened due to congestion or hemorrhage. In eight of the gorillas, both cestodes and hemorrhagic and/or hyperemic mucosa were evident.

Several gorillas were observed with cecal ($n = 5$, 10%) and/or colonic (8, 16%) mucosal reddening or hemorrhage, although frank bleeding into the lumen was less frequent. In one adult, there was extensive distension of the entire large bowel (cecum and colon), mural edema, and serosal and mucosal hemorrhage. Colonic mural nodules were reported in seven gorillas (14%). The individuals where cestodiasis was recorded were 18 gorillas (36%), involving all sections of the GI tract distal to the stomach. Nematodiasis was seen in eight gorillas (14.8%), involving the same region.

Gross lesions were seen in the appendix of only 6 of 47 gorillas (12.8%) for which the appendix was described, and consisted of reddening of the mucosa or serosa by congestion or hemorrhage with luminal blood seen in one animal and nematodes in two.

TABLE 2 Gross lesions associated with the gastrointestinal tract (GIT) in free-ranging mountain gorillas

	Type of lesion/finding(s)	No.	Prevalence (%)
GIT section			
Oral[d]	Darkening/pigmentation of teeth and tongue	11	20
	Gingival regression and/or feed impaction	4	7.3
	Teeth extensively worn	4	7.3
	Cleft palate	2	3.6
	Draining tracts inside of jaws (with mandibular osteomyelitis)	1	1.8
	Swollen mandible	1	1.8
	Dental malocclusion	1	1.8
Esophagus[d]	Areas of erythema	1	1.8
	Pale serosa	1	1.8
	Rupture (due to gunshot)	1	1.8
	Thickened muscularis	1	1.8
Stomach[d]	Hyperemia/erythema of mucosa	11	20
	Excessive mucus	10	18.2
	Mucosal ulceration	4	7.3
	Nodules	2	3.6
	Pylorus firm and fibrous	1	1.8
	Foci of mucosal necrosis	1	1.8
	Stomach wall edematous	1	1.8
Small intestines[d]	Cestodes	16	29.1
	Mucosal hemorrhages or hyperemia	13	23.6
	Nematodes	4	7.3
	Excessive mucus	4	7.3
	Serosal hemorrhages or congestion	4	7.3
	Mural nodules	1	1.8
	Mucosal erosions	1	1.8
Colon[a]	Hemorrhages/hyperemia of mucosa	8	16
	Nodules (mucosal and mural)	7	14
	Nematodes	6	12
	Digesta with blood and mucus	4	8
	Tear/rupture/perforation	2	4
	Excessive mucus	1	2
	Edema of wall	1	2
Cecum[a]	Mucosal congestion and hemorrhages	5	10
	Intestinal adhesions to abdominal wall	5	10
	Cestodes	2	4
	Lumen empty	2	4
	Nematodes	1	2
	Thickened and congested wall	1	2
	Edema of wall	1	2
	Erosions of mucosa	1	2
	Bloody contents	1	2
	Hard contents with excess mucus	1	2
Rectum[b]	Congestion and hemorrhages of mucosa	7	14.9
	Fecal lobes with a lot of mucus and blood	3	6.4
	Lumen empty	3	6.4
	Tear/rupture/perforation	3	6.4
	Soft or loose stool	3	6.4
Appendix[b]	Nematodes	2	4.3
	Thickened and congested wall	2	4.3
Liver[c]	Areas of fibrosis	3	5.5
	Surface nodules extending into parenchyma	1	1.8
	Abscesses in parenchyma	1	1.8
	Nematodes	1	1.8
	Gall bladder enlarged	3	5.5

Note: Some animals had more than one type of lesion:
[a]Gut section was observed for lesions in 50 cases.
[b]Gut section was observed for lesions in 47 cases.
[c]Observation on gut section was made in 51 cases.
[d]Observation on gut section was made in 55 cases.

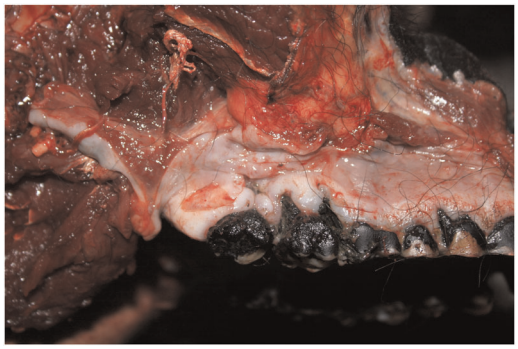

FIGURE 1 Black pigmentation of teeth and black dental plaque associated with gingival regression are present in the oral cavity of an adult female mountain gorilla that died due to extension of the infection into the jaw bone (osteomyelitis), bloodstream (septicemia), and heart (valvular endocarditis) (postmortem examination image courtesy of Dr. Chris Whittier and Mountain Gorilla Veterinary Project)

Macroscopically evident liver lesions were rarely described. The gall bladder was noted to be distended or thickened in three adults. No gross lesions were appreciated in major salivary glands or pancreas.

3.3 | Histological lesions

The histological lesions and their prevalence in sections of the GI of 60 individuals are presented in Table 3. Many of the sections of stomach and intestines were somewhat autolyzed, which reduced the pathologists' ability to detect protozoans and subtle lesions of the superficial epithelium. Inflammatory infiltrates, distortion of architecture, and metazoan parasites were still evident, however.

The most prevalent oral finding was superficial bacterial colonization of the tongue in 24 (40.7%) individuals, which was associated with dark pigmentation of the superficial epithelium in nine (15.3%) individuals. Additional lesions included sialoadenitis/sialodochitis in 22 (37.3%), glossitis in 15 (25.4%), and foreign body granulomas in the tongue, tonsil, or minor salivary glands in nine (15.3%) individuals. The inflammation, centered on the minor salivary glands and ducts, was primarily mild and lymphocytic. Granulomas in the oral cavity and pharynx were centered on plant material (presumed thorns or nettle "hairs").

Inflammation of the esophagus was noted in 11 gorillas (18.6%) and was generally mild, multifocal, and predominantly lymphocytic, although there was one case each (1.6%) of neutrophilic and eosinophilic esophagitis and erosive esophagitis and three (3.5%) gorillas with focal granulomatous esophagitis associated with plant foreign bodies. In one adult female, cytoplasmic swelling and intranuclear inclusion bodies were seen without necrosis or vesicle formation, and were suggestive of recrudescent herpesvirus infection. In all cases, the inflammation in the tongue and esophagus was judged unlikely to have been clinically significant.

TABLE 3 Microscopic lesions in the gastrointestinal tract (GIT) of free-ranging mountain gorillas

	Type of lesion	No.	Prevalence (%)
GIT section			
Oral cavity[a]	Superficial bacterial colonization	24	40.7
	Sialoadenitis/sialodochitis	22	37.3
	Glossitis	15	25.4
	Pigmentation	9	15.3
	Foreign body granulomas (tongue, tonsil, salivary gland)	9	15.3
Esophagus[a]	Esophagitis	11	18.6
	Esophagitis (with foreign body)	3	5.1
	Superficial bacterial colonization	2	3.4
	Pigmentation	1	1.7
	Epithelial erosion	1	1.7
	Intraepithelial edema with intranuclear inclusion bodies	1	1.7
Stomach[a]	Gastritis (including proliferative gastritis)	24	40.7
	Nematodes	10	16.9
	Hyperplasia of the mucosa (proliferative gastritis)	10	16.9
	Ulceration, erosion/erosive gastritis, and suppuration	7	11.9
	Protozoal (*Troglodytella* spp.) infection	3	5.1
	Crypt ectasia	3	5.1
	Congestion	3	5.1
	Mucosal hyperplasia (without gastritis)	1	1.7
	Carcinoid	1	1.7
	Mucocal necrosis (clostridial)	1	1.7
	Cestodes	1	1.7
Small intestines[a]	Enteritis	34	58.6
	Nematodes	15	25.9
	Hyperplasia of gut-associated lymphoid tissue	7	12.1
	Cestodes	5	8.6
	Germinal center depletion with histiocytosis	3	5.2

TABLE 3 (Continued)

	Type of lesion	No.	Prevalence (%)
	Crypt ectasia	3	5.2
	Crypt herniation	3	5.2
	Villous blunting	2	3.4
	Granulomatous enteritis with intralesional foreign body	1	1.7
	Mucosal and submucosal edema	1	1.7
	Carcinoid	1	1.7
	Mural abscessation	1	1.7
	Bacterial vasculitis	1	1.7
	Anaerobic bacterial enteritis (Gram negative)	1	1.7
	Protozoa	1	1.7
Colon[b]	Colitis	17	29.3
	Nematodes (luminal)	13	22.4
	Mural nodule with intralesional nematode	6	10.4
	Gut-associated lymphoid tissue hyperplasia	4	6.9
	Mucosal and submucosal edema	4	6.9
	Mural abscessation	4	6.9
	Crypt degeneration, necrosis, and abscessation	3	5.2
	Germinal center hyalinization	3	5.2
	Crypt ectasia	3	5.2
	Mural thrombi	2	3.4
	Crypt herniation	1	1.7
	Colonization by Gram-positive cocci	1	1.7
	Luminal bacteria	1	1.7
	Bacterial colonization of surface epithelium	1	1.7
	Ulcerations	1	1.7
Cecum[b]	Nematodiasis	6	10.3
	Typhlitis	2	3.4
		2	3.4

(Continues)

TABLE 3 (Continued)

	Type of lesion	No.	Prevalence (%)
	Hyperplasia of the gut-associated lymphoid tissue		
	Crypt degeneration, abscessation, and necrosis	1	1.7
	Crypt herniation	1	1.7
	Loss of crypts	1	1.7
	Colonization by Gram-positive cocci	1	1.7
Appendix[c]	Nematodiasis	13	56.5
	Appendicitis	6	26.1
Pancreas[d]	Acinar degeneration	18	78.3
	Acute pancreatitis	2	8.7
	Diffuse interstitial fibrosis	1	4.3
Liver[b]	Pigmentation (centrilobular lipofuscinosis)	22	36.7
	Fibrosis	21	35
	Hepatitis	16	26.7
	Lipidosis	11	18.3
	Hepatic capillariasis	11	18.3
	Hepatocellular atrophy	10	16.7
	Hepatopathy	9	15
	Pigmentation (hemosiderosis)	9	15
	Circulating neutrophilia, microabscesses, and leukocytosis	8	13.3
	Hepatic necrosis	3	5
	Liver carcinoid (metastatic from pyloric region of stomach)	1	1.7
	Bile stones	1	1.7
	Cholecystitis	3	5
	Granulomatous hepatitis	1	1.7

Note: A combination of them occur in the same case:

[a]Observed in 59 cases.

[b]Observed in 58 cases.

[c]Observed in 23 cases.

[d]Observed in 25 cases.

Twenty-four gorillas (40.7%) had gastritis that was often chronic and characterized by lamina proprial fibrosis, gland distortion or mucous metaplasia, ectasia, and varying degrees of mucosal proliferation. In 10 cases (16.9%), irregular papillary projections were formed by mucosal hyperplasia (Figure 2), which were large enough to be perceived grossly in two gorillas. Ulceration or erosion was present in seven animals (11.9%). Superficial erosions and pinpoint foci of hemorrhage were seen in infants and were compatible with so-called "stress ulcers." Deep ulceration accompanied proliferative gastritis in three adults, and severe focal ulceration was observed in an adult female with acute post-parturient heart failure, an adult female with endocarditis and sepsis, and an older adult female with carcinoid, the only gastrointestinal cancer identified. Chronic atrophic gastritis was present in this animal as well. In a 5-year-old male, the stomach was severely distended by gas and plant material and there was acute clostridial gastritis and aspiration pneumonia. Trichostrongyle nematodes were seen in 10 stomachs associated with mucosal proliferation (Figure 3). No spiral organisms suggestive of *Helicobacter* sp. were seen in any of the sections examined.

In the small intestine, histological lesions included enteritis in 34 (58.5%) gorillas. The enteritis varied in character and severity, but in general it consisted of mild separation and elevation of crypts by lymphocytes, plasma cells, and eosinophils. Villous blunting was infrequently seen ($N = 2$, 3.4%), as were crypt herniation or crypt "abscessation" ($N = 3$, 5.2%) individuals. Both animals with villous blunting had cestodiasis, as did one animal with moderate eosinophilic enteritis. However, 14 other gorillas with cestodiasis had no histological lesions in the small intestine. Hyperplasia of gut-associated lymphoid tissue (GALT) was present in seven (12.1%) individuals (6 infants and 1 juvenile) and three individuals had germinal center depletion with histiocytosis (all infants). Transmural inflammation was present in one juvenile and one adult and was associated with extensive submucosal edema. Protozoal ciliates, granulomatous enteritis with foreign body, mucosal/submucosal edema, gastro-duodenal carcinoid, mural abscessation, bacterial vasculitis, and anaerobic bacterial enteritis were all described, each in one (1.7%) individual. Nematodes were identified histologically in 15 (25.9%) individuals and cestodes in five (8.6%) of the individuals. No cases of giardiasis, cryptosporidiosis, or enterocytozoanosis were identified histologically.

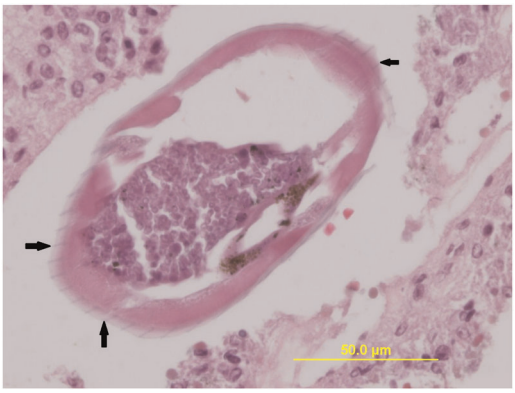

FIGURE 3 Histological image of a transverse section of a trichostrongylid nematode from the stomach of a mountain gorilla with chronic gastritis. Note the projections (arrows) on the surface of the worm which are longitudinal ridges characteristic of this group of nematodes (hematoxylin and eosin)

In the colon, the most prevalent lesion was inflammation (colitis) in 17 (29.3%). As in the small intestine, colitis was generally mild and consisted of varying numbers of lymphocytes, plasma cells, eosinophils with fewer neutrophils and macrophages. Accompanying lesions included crypt degeneration, necrosis, or crypt "abscessation" in three (5.2%) individuals, crypt ectasia in three (5.2%), and mucosal/submucosal edema in four (6.9%) gorillas. Colonic GALT was hyperplastic in four gorillas while GALT germinal center depletion/hyalinization was noted in three (5.2%). Six individuals (10.4%) were noted to have mural nodules (chronic abscesses) associated with nematodiasis (strongyles, *Oesophagostomum* sp.) (Figure 4) while mural abscessation, in which nematodes were not seen, was noted in an additional four individuals. Luminal nematodiasis was observed in

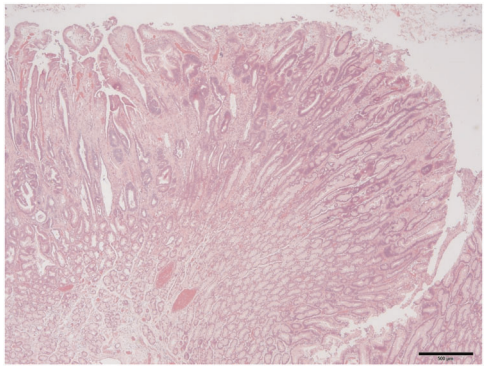

FIGURE 2 Histological image of the stomach in an adult mountain gorilla with chronic inflammation (gastritis) associated with focal mucosal proliferation and intestinal metaplasia. Scale bar = 500 µm (hematoxylin and eosin)

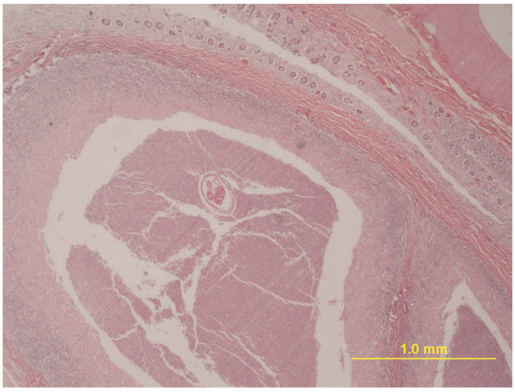

FIGURE 4 Section of the colon from an adult female mountain gorilla with a mural abscess containing sections from a strongylid nematode. Nematode morphology and type of lesion are characteristic of nodular worm infection which in apes is usually due to *Oesophagostomum* sp. (hematoxylin and eosin)

the colon in 13 gorillas (22.4%). Ciliated protozoans (presumed to be normal symbiotic fauna) were found in the colon, appendix, and cecum of the same individual in which they were seen in the distal small intestine (ileum).

Lesions in the cecum included nematodiasis in six (10.3%) individuals, typhlitis in two (3.4%), and hyperplasia of the GALT in two (3.4%). The nematodes were identified as pinworms. There was crypt degeneration, necrosis, and abscessation in one (1.7%), crypt herniation in one (1.7%) individual, loss of crypts in one (1.7%) individual, and colonization by Gram-positive cocci in one (1.7%) individual.

In the appendix, appendicitis, observed in six individuals, was generally mild and associated with infiltration by eosinophils; no cases of severe transmural or suppurative appendicitis were appreciated. Profiles of nematodes with morphology consistent with oxyurids were common in the appendix, particularly in younger animals.

Large intestinal lesions considered to be severe enough to have caused death were present in only one individual (see Figure 5). This was an adult male which had ulcerative transmural typhlocolitis with massive edema, colonization of the wall by stout Gram-positive rods suggestive of *Clostridium* spp., and perivascular and intracellular Gram-negative rods. Necrosis was accompanied by vascular thrombosis.

Liver lesions were more often degenerative than inflammatory, with centrilobular hepatocellular pigmentation (lipofuscinosis) often accompanied by mild fibrosis in 22 (36.7%) gorillas, and hepatocellular hemosiderosis in nine (15%) gorillas, mostly infants, but also two emaciated adults with infections. Hepatopathy characterized by variation in the size of hepatocytes (anisocytosis) and/or their nuclei (anisokaryosis) in six (10.35%) gorillas and binucleated hepatocytes in nine (15.5%) was observed without any histologically obvious etiology. Hepatic lipidosis in 11 gorillas (18.3%) and hepatocellular atrophy in 10 (16.7%) were additional degenerative changes. Fibrosis was recorded in 21 gorillas (35%) and was both centrilobular and periportal. Hepatitis was reported in 16 gorillas (26.7%) associated with scattered microabscesses or circulating leukocytosis/neutrophilia in eight (13.3%) and hepatocellular necrosis in three (5%). The pattern of inflammation was compatible with septicemic spread via the systemic or enterohepatic circulation. Hepatic capillariasis, often with fibrosis and/or granulomatous inflammation, as has been previously reported (Graczyk et al., 1999), was identified in 11 (18.3%) individuals (Figure 6). This lesion was sometimes quite extensive, but in most cases was seen in only a small region of the examined parenchyma. Focal periportal cholangitis with microcholelithiasis was identified in a single 35-year-old adult male. Severe suppurative cholecystitis was noted in one adult female that had a grossly abnormal gall bladder, and mild subacute cholecystitis in two others in which no gross lesions had been appreciated.

Pancreatic lesions were uncommon with the exception of zymogen depletion in exocrine acini in 18 (78.3%) individuals (15 infants and three adults) all of whom were emaciated. One juvenile female that died during a respiratory disease outbreak had multifocal acute pancreatic necrosis of presumptive viral etiology and another infant had acute pancreatitis secondary to peritonitis. Diffuse interstitial fibrosis in the pancreas of a geriatric female was of undetermined etiology.

3.4 | Lesions in the different age groups

The pattern of lesions varied among age groups. Gastritis was not present in neonates or infants but increased in frequency with age, as did nematodiasis (48% and 28%, respectively in adults). Enteritis occurred in all age groups, and was observed in more than half of the gorillas in each age class. Colitis was present in all age classes as well, but was less common in adults than in younger animals, which were also more likely to exhibit nematodes in the appendix. Cestodiasis was much more common in adults and was identified more often in the small intestine than in the colon. When present in infants and juveniles, cestodes were in low numbers (1–15, when enumerated) compared with the adults, in which "dozens" to 200 tapeworms were

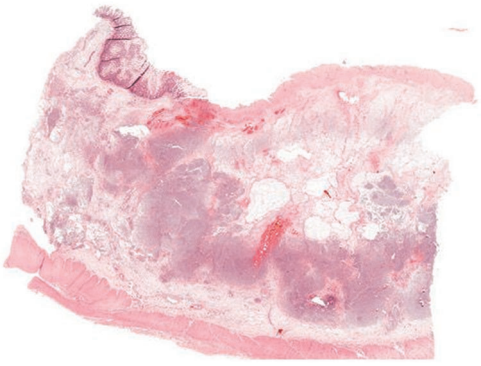

FIGURE 5 Subgross image of a histological section of the large bowel from an adult, male mountain gorilla demonstrating massive thickening due to inflammation and edema of the wall. Especially marked is the clear space (edema) in the submucosa between the lining and the wall of the colon (hematoxylin and eosin)

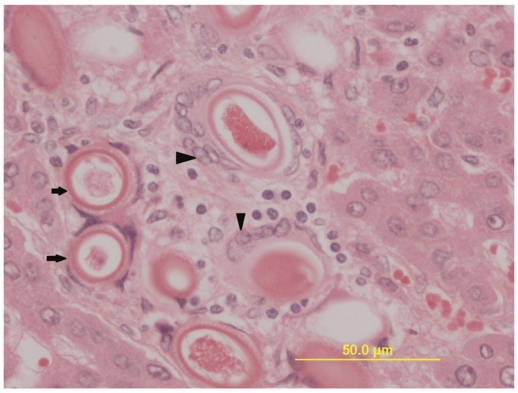

FIGURE 6 Hepatic capillariasis associated with focal granulomatous hepatitis in a young mountain gorilla. Eggs (arrows) are surrounded by multinucleated giant cells (arrowheads) (hematoxylin and eosin)

counted. Hepatic capillariasis was present in all age classes including a neonate and an old adult female estimated to be 42 years old. Centrilobular liver fibrosis and centrilobular lipofuscinosis were more common in adults. Hepatic hemosiderosis was present in animals in poor nutritional condition especially infants, as was pancreatic zymogen depletion.

3.5 | Risk factors for GI inflammation

A statistically significant association existed between gastritis and nematodiasis ($p < 0.001$; odds ratio [OR] = 11.3; 95% confidence interval [CI], 2.2–58.7) (Table 4). Gastritis was 11 times more associated with nematodiasis. There was also a strong statistical correlation between hepatitis and capillariasis ($p < 0.0061$; OR = 6.8; 95% CI, 1.7–28.0). Hepatitis was six times more likely to be observed in livers that showed concurrent capillariasis than in livers unaffected by *Capillaria*.

There was no statistically significant correlation between enteritis and nematodiasis; colitis and nematodiasis; and hepatic fibrosis and capillariasis ($p > 0.05$). However, there was a strongly significant inverse relationship between cestodiasis and enteritis ($p < 0.00014$; OR = 0.024; 95% CI, 0.002–0.134), meaning that individuals with cestodiasis were 40 times less likely to have enteritis than individuals without cestode infections.

4 | DISCUSSION

We identified a number of gross and histological digestive tract lesions in wild mountain gorillas, many of which were mild and likely not contributing to mortality, and some of which have not been reported before in this species.

The most prevalent gross lesion noted in the oral cavity was pigmentation of the teeth and tongue which was reported more frequently in adults. It was often accompanied, histologically, by bacterial colonization of the tongue mucosa, but seemed to be of no pathological significance. In a study of mountain gorilla skeletal remains, dental pigmentation was observed to be ubiquitous in adults and the author opined that it was a result of their diet (Lovell, 1990). In humans, tooth pigmentation due to dietary items, such as betel nuts, is well recognized (Reichart et al., 1985). However, other lines of evidence suggest that black pigmentation of human teeth is associated with iron protoporphyrins produced within bacterial plaque (Reid et al., 1977). Black pigmented *Bacteroides porphyromonas* and *Bacteroides intermedius* have been found in another nonhuman primate, *Saimiri scuireus* (squirrel monkey) (Clark et al., 1988). Pigmented anaerobes, such as *B. porphyromonas*, are associated with periodontal disease in humans (Mombelli et al., 1996; Saba et al., 2006). Because of this association, it would be interesting to study the oral flora of the mountain gorilla.

In a few individuals, we observed gingival regression and/or feed impaction, mandibular draining tracts, and mandibular osteomyelitis in gorilla tissues, all of which can be a consequence of periodontal disease (McGavin & Zachary, 2007). In only one individual with severe osteomyelitis was the periodontal disease considered contributory to the animal's death, as the gorilla also had acute vegetative valvular endocarditis and septicemia.

The prevalence of oral and periodontal lesions in this study was much lower than the 45%–100% of adults reported previously in reviews of skeletal remains of mountain gorillas (Elgart, 2010; Lovell, 1990). It is possible that gingival and oral lesions were underreported in gross postmortem reports in our study, as some prosectors may have considered these lesions to be normal in older animals. Dental attrition was also rarely reported in our study, likely for a similar reason. This lesion is usually seen in older adults and could be a result of the vegetarian diet, some of which is abrasive (Elgart, 2010). It has been reported that tooth wear and gingival and alveolar bone regression are higher in mountain gorillas than in other apes (Lovell, 1990).

Histological lesions associated with the oral cavity included inflammation in the minor salivary glands and in the tongue, often associated with foreign body granulomas containing plant material. The diet of wild mountain gorillas consists of many different plants, such as *Galium*, nettles, thistles, wild celery, and bamboo, the chewing of which could result in penetration of the oral mucosa by spines or splinters (Byrne, 2001). Most of the plant material observed in sections was well encapsulated and there was no evidence of the introduction of bacterial infection.

The developmental (congenital) lesions seen in the GI were cleft palate and dental malocclusion. Cleft palate was identified in two infants, one of which had been the subject of a previous report (Nutter et al., 2005). In one infant with cleft palate, the cause of death was linked to GI whereas in the other case, the carcass was decomposed and the veterinarian was only able to identify the cleft palate but not able to determine if it was the sole cause of death. While the prevalence of cleft palate in nonhuman primates is unknown, the prevalence of cleft palate in human infants ranges from

TABLE 4 Logistic regression relationship between GI lesions and parasitism

Variables					
Dependent	Independent	Odds ratio	95% Confidence limits		*p* value
Gastritis	Nematodiasis	11.3	2.2	58.7	0.001*
Enteritis	Nematodiasis	1.8	0.5	6.2	0.3184
Fibrosis	Capillariasis	2.7	0.7	10.3	0.1406
Hepatitis	Capillariasis	6.8	1.7	28.0	0.0061*
Colitis	Nematodiasis	2.5	0.7	8.7	0.1383
Enteritis	Cestodiasis	0.024	0.002	0.134	0.00014*

Note: *p* value—likelihood ratio statistics for type 3 analysis.
Abbreviation: GI, gastrointestinal tract.
*Statistically significant at 5% level.

approximately 1/700 to 1/1000 births, and the finding of two such defects in such a small population of apes, although 10 years apart, is interesting (Manyama et al., 2011). The other developmental anomaly was dental malocclusion in an adult.

Grossly, few lesions were noted in the esophagus, but in one animal the esophagus was ruptured by gunshot. Trauma (including natural events, for instance conspecific, and human interactions) is the leading cause of mortality of mountain gorillas (Cranfield, 2008). Esophagitis, noted in 11 (18.6%), was generally mild. The etiology was not determined in most cases, and it was most frequently observed in infants followed by adults. In one adult female, the presence of intranuclear inclusion bodies in cells with cytoplasmic swelling was suggestive of a herpesvirus infection. Herpesviruses identified to date in mountain gorillas by isolation or serology include Human herpesvirus-1, Human herpesvirus-2, a cytomegalovirus, and Epstein–Barr-like lymphocryptovirus (Eberle, 1992; Eberle & Hilliard, 1989; Evans et al., 2016).

In the stomach, the most common lesion, occurring in more than a third of the gorillas ($N = 24$, 40.7%) was chronic gastritis. This was often accompanied by mucosal proliferation, dysplasia, and intestinal or mucoid metaplasia. Naturally occurring gastritis in other species of primates has been associated with spiral bacteria, such as *Helicobacter pylori*, or parasitism, or maybe immune-mediated (Emerson et al., 2014; Haesebrouck et al., 2009; Narama et al., 1983; Rubio et al., 2008). No spiral bacteria were seen histologically in gastric sections from mountain gorillas, however, trichostrongylid nematodes were observed within the gastric mucosa and lumen of approximately half of the gorillas with gastritis, primarily in the adults. Two gastric trichostrongyles have been previously identified in mountain gorillas, *Hyostrongylus kigeziensis* from both Bwindi and Virunga gorillas and *Paralibyostrongylus kalinae* from Bwindi gorillas (Rothman et al., 2006; Sleeman et al., 2000). The exact identity of the nematodes seen in histological sections associated with gastritis in this study was not determined at the time of necropsy. Chronic gastritis was not considered a primary cause of death in any of these cases, but was thought to have been a contributing factor in five gorillas. Chronic gastritis in humans can lead to debility through reduced appetite due to pain and nausea, anemia due to ulceration, and blood loss or loss of intrinsic factors causing pernicious anemia, achlorhydria due to loss of parietal cells or chronic vomition, and predisposition to gastric neoplasia. Severe gastritis was the cause of death in a young gorilla with peracute gastric distension (bloat) and necrosis associated with bacterial invasion. Bloat is infrequently reported in great apes, though it is a well-known problem in captive primates in which clostridial infection is sometimes implicated (Bodkin et al., 2003; Pond et al., 1982; Yasuda et al., 2015).

Severe gastric ulceration associated with hemorrhage contributed to morbidity in an older adult female with cancer, an older adult female with valvular endocarditis and sepsis, and another adult female with congestive heart failure. Mild superficial ulceration or hemorrhage in some gorillas was similar to lesions due to sepsis, uremia or stress in other species.

The one case of gastrointestinal cancer occurred in a 35-year-old adult female from the Virunga National Park (DRC), who exhibited a malignant carcinoid (neuroendocrine cancer) arising at the gastroduodenal junction, which was too advanced to determine gastric or duodenal origin. The cancer had metastasized to regional lymph nodes and liver. This animal had chronic atrophic gastritis, which has been implicated in the development of carcinoids in humans (Vannella et al., 2012). Cancer is rare in gorillas and other apes both in captivity and in the wild (Lowenstine et al., 2015).

We observed a high prevalence of cestodiasis (tapeworms) ($N = 16$, 29.1%) of the mountain gorillas. Cestodes were found predominantly in the small intestine. Previously identified cestodes in mountain gorilla postmortem examinations were *Anoplocephala gorillae* (Fossey, 1983) and eggs of *Anoplocephala* species are commonly identified in mountain gorilla feces, occurring in up to 80% of samples (Kalema-Zikusoka et al., 2005; Redmond, 1983; Sleeman et al., 2000). Although, heavy infestations have been found in adults in both ours and previous studies (Rothman et al., 2006, 2008), the infection seems to remain subclinical. In fact, our analyses revealed that individuals with cestodiasis were 40 times less likely to have enteritis. It is not clear how cestodiasis is protective for enteritis. Recent studies have shown that immune mechanisms induced by certain nematodes and cestodes suppress enteritis and have been actually proposed as possible future remedies for human inflammatory bowel disease (Reyes et al., 2016; Smith et al., 2007). It is further argued that the development of an immunoregulatory environment likely contributes to the chronicity of helminth infection and asymptomatic disease. On the contrary, a related parasite *Anoplocephala perfoliata* in horses causes mucosal damage at sites of attachment, hypertrophy of muscular layers, and altered GI motility due to alterations in myenteric ganglia (Pavone et al., 2011). Therefore, more attention and sampling of attachment sites, if any, of cestodes in the gorillas is warranted, but may be difficult due to autolysis and postmortem detachment. Although *Hymenolepis* tapeworms have been reported previously, none were identified in gorillas in our study.

Nematodes were found grossly and histologically in all segments of the intestines in our study, but not in all animals. Exact identification was not possible in all cases. Earlier work identified nematodes (adults, larvae, and eggs) in feces as *Strongyloides fuelleborni*, *Oesophagostomum* sp., *Ternidens deminutus*, *Trichuris*, *Ascaris*, *Trichostrongylus*, *Hyostrongylus*, *Probstymaria* species, and possibly *Murshidia* (Redmond, 1983; Rothman et al., 2006; Sleeman et al., 2000). Lesions compatible with oesophagostomiasis (nodular worm) were found in the colons of seven gorillas, a prevalence much lower than that has been suggested for wild African apes, which may serve as reservoirs for human oesophagostomiasis (Cibot et al., 2015; Krief et al., 2008).

Statistically, there was no association between gastrointestinal nematodiasis and enteritis or colitis in our study. This begs the question as to what other etiologies for enteritis may be present in the population. Intestinal bacterial cultures were not routinely done at the time of postmortem examinations; this should be considered in the future. Earlier work done in this species identified bacteria of the

genera *Salmonella*, *Camphylobacter*, and *Shigella* in the feces of the mountain gorillas of Bwindi and Mgahinga in Uganda (Kalema-Zikusoka et al., 2005; Nizeyi et al., 2001). Similarly, although several studies have identified pathogenic protozoans and fungi, *Giardia*, *Cryptosporidium*, various amoeba, microsporidia *Encephalitozoon* and *Enterocytozoan* (Graczyk & Cranfield, 2003; Graczyk et al., 2001; Hogan et al., 2014; Nizeyi et al., 1999), none were identified during histological examination in this study. The failure to identify protozoans or microsporidians in this study could be due to chance or, quite likely, autolysis. The only protozoa identified in this study were endodidiomorphid ciliates seen in the ileum, cecum, colon, and appendix of a single juvenile gorilla. Ciliates of four genera (*Troglodytella*, *Prototapirella*, *Gorillophilus*, and *Gorilloflasca*) are commensal in wild apes, including lowland and mountain gorillas (Freeman et al., 2004; Imai et al., 1991; Ito et al., 2016, 2018).

Intestinal adhesions with the abdominal wall, involving either the small or large intestines were observed in 5 (10%) of the cases, the majority of which were adults. Past or current parasitic migrations is one suspected etiology. Reports of adhesions caused by *Oesophagostomum* spp. have been previously documented in humans and zoo-housed apes (Storey et al., 2000; Strong et al., 2017).

Liver lesions were more common histologically than grossly. Centrilobular fibrosis in 21 (35%) and hepatocellular lipofuscinosis in 22 (36.7%), were most common in adults. In humans, similar changes accumulate with aging, but are present to some degree after the first decade of life (Ehrlich et al., 1960; Lowenstine et al., 2015). In other species, these histologic changes which are related to oxidative stress can occur due to centrilobular hypoxia/ischemia as might occur in chronic passive congestion of heart failure. Iron accumulation in hepatocytes, seen in 15% of the livers, was mostly in infants with inanition or infections. One of the two adults with hemosiderosis was a male with septicemia and emaciation, secondary to skin wounds. Sepsis and inanition are both recognized causes of hepatocellular iron accumulation in other species (Lowenstine & Munson, 1999). Hepatocellular lipidosis or hepatocellular atrophy, also indicators of inanition, were found in 11 (18.3%) and 10 (16.7%) gorillas, respectively.

Hepatitis was seen in two contexts: hepatic capillariasis and circulating neutrophilia/leukocytosis, or microabscesses suggestive of systemic or enteric inflammation. The livers with hepatitis were over six times more likely to have *Capillaria*, but there was no significant relationship between capillariasis and fibrosis. Earlier postmortem studies revealed hepatic capillariasis in gorillas in Rwanda (Graczyk et al., 1999), and identified it as a cause of morbidity in 25% of gorilla infants (Hassell et al., 2017). Our study extends the range of this parasite to Bwindi in Uganda and in Virunga National Park in the DRC.

The histological findings in the pancreas were predominantly zymogen depletion and acinar degeneration, which can be associated with inanition or renal disease in humans, and inanition in domestic and wild mammals (McGavin & Zachary, 2007). The affected gorillas were predominantly infants and emaciated adults with chronic diseases (e.g., infections and cancer).

Gastroenteritis or colitis, historically important causes of death in approximately a quarter of captive gorillas (Strong et al., 2017), were considered to have been the cause of death in only five individuals (8%): an adult male with necrotizing typhlocolitis of bacterial origin; an adult female with severe bacterial enteritis; an infant female with severe enterotyphlocolitis associated with attaching and effacing bacteria; another infant with severe eosinophilic and neutrophilic transmural enteritis; and an infant with acute gastric tympany (bloat). In addition, three adult females had diarrhea at the time of death, but histological lesions in the intestines were mild and lesions in other organ systems were contributory to mortality. Acute diarrhea can be due to acute stress or can be an agonal event, but can also cause death due to dehydration or electrolyte imbalances. Depending on the inciting cause, diarrhea can be associated with minimal morphological alterations in both humans and nonhuman primates (Carvalho et al., 2003; Thapar & Sanderson, 2004). Digestive tract lesions were associated with death in three other gorillas: the old adult female with cancer arising from the pyloric region; an old adult female with periodontitis and osteomyelitis of the jaw leading to sepsis and heart lesions; and a neonate with cleft palate. The second neonate was too autolyzed to assign a cause of death. Gastritis was thought to be a cofactor in mortality in two cases and a likely cause of morbidity in combination with other causes of death in eight additional animals.

The study's null hypotheses were rejected in part. Although most of the parasites were innocuous, a few (i.e., gastric trichostrongylidiasis, and hepatic capillariasis) were associated with morbidity. Morbidity could not be ascribed to the majority of the oral/periodontal lesions although in one case such lesions precipitated death by being a portal for systemic infection.

Nevertheless, intestinal parasitism did not seem to be a factor in either morbidity or mortality. This study revealed that despite the presence of numerous parasites, the prevalence of morbidity and mortality due to digestive tract lesions is less in free-living gorillas than those in managed care. It also establishes a baseline of gastrointestinal lesions of wild mountain gorillas. It points to the need for a more rigorous gross and histological examination of the entire digestive tract and routine postmortem parasite identification and bacterial cultures to better understand the role of gastrointestinal disease in mountain gorilla health.

ACKNOWLEDGMENTS

This study was possible with the support of Morris Animal Foundation, the Mountain Gorilla Veterinary Project (MGVP), the Uganda Wildlife Authority (UWA), the Rwanda Development Board (RDB) (with special thanks to Dr. Antoine Mudakikwa), and the Institut Congolaise pour la Conservation de la Nature (ICCN) of the DRC. We acknowledge the work done by all the MGVP field veterinarians and technical staff on gross postmortem examinations and recording of all the observational information. Laboratory staff at UC Davis Veterinary Medical Teaching Hospital were very helpful in processing tissues, and several pathology residents were involved in preliminary histological review. Funding was provided by the Mountain Gorilla

Veterinary Project (MGVP) Inc. We are indebted to the MGVP Project Director Emeritus, Dr. Michael Cranfield who made it possible for this study to be carried out.

CONFLICT OF INTERESTS

The authors declare that there are no conflicts of interests.

AUTHOR CONTRIBUTIONS

Denis Muhangi: Conceptualization (lead); data curation (lead); formal analysis (lead); investigation (supporting); methodology (supporting); project administration (supporting); software (lead); supervision (supporting); validation (supporting); writing—original draft (lead); writing—review and editing (supporting). **Chris H. Gardiner**: Data curation (supporting); investigation (supporting); methodology (supporting); validation (supporting). **Lonzy Ojok**: Conceptualization (supporting); data curation (supporting); formal analysis (supporting); investigation (lead); methodology (supporting); project administration (supporting); supervision (lead); writing—original draft (supporting); writing—review and editing (supporting). **Kirsten V. K. Gilardi**: Conceptualization (supporting); funding acquisition (lead); project administration (lead); supervision (supporting); writing—original draft (supporting); writing—review and editing (supporting). **Antoine B. Mudakikwa**: Conceptualization (supporting); funding acquisition (supporting); investigation (supporting); project administration (supporting); resources (supporting); supervision (supporting); writing—review and editing (supporting). **Linda J. Lowenstine**: Conceptualization (lead); data curation (supporting); formal analysis (supporting); investigation (lead); methodology (lead); project administration (supporting); resources (supporting); supervision (lead); validation (lead); writing—original draft (supporting); writing—review and editing (supporting).

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

Bancroft, J. D., & Gamble, M. (2008). *Theory and practice of histological techniques* (xv, p. 52). Elsevier Health Sciences.

Bodkin, N. L., Alexander, T. M., Ortmeyer, H. K., Johnson, E., & Hansen, B. C. (2003). Mortality and morbidity in laboratory-maintained rhesus monkeys and effects of long-term dietary restriction. *Journal of Gerontology: Biological Sciences*, *58*(3), 212–219. https://biomedgerontology.oxfordjournals.org/content/58/3/B212.abstract

Byrne, R. W. (2001). Clever hands: The food-processing skills of mountain gorillas. In M. M. Robbins, P. Sicotte, & K. J. L. Stewart (Eds.), *Mountain gorillas: Three decades of research at Karisoke* (pp. 293–313). Cambridge University Press.

Carvalho, V. M., Gyles, C. L., Ziebell, K., Ribeiro, M. A., Catão-Dias, J. L., Sinhorini, I. L., Otman, J., Keller, R., Trabulsi, L. R., & Pestana de Castro, A. F. (2003). Characterization of monkey Enteropathogenic *Escherichia coli* (EPEC) and human typical and atypical EPEC serotype isolates from neotropical nonhuman primates. *Journal of Clinical Microbiology*, *41*(3), 1225–1234. https://jcm.asm.org/content/41/3/1225.abstract

Chitwood, M., & Lichtenfels, J. R. (1973). *Identification of parasitic metazoa in tissue sections*. Academic Press Inc.

Cibot, M., Guillot, J., Lafosse, S., Bon, C., Seguya, A., & Krief, S. (2015). Nodular worm infections in wild non-human primates and humans living in the Sebitoli area (Kibale National Park, Uganda): Do high spatial proximity favor zoonotic transmission? *PLOS Neglected Tropical Diseases*, *9*(10), 0004133. https://doi.org/10.1371/journal.pntd.0004133

Clark, W. B., Magnusson, I., Abee, C., Collins, B., Beem, J. E., & McArthur, W. P. (1988). Natural occurrence of black-pigmented *Bacteroides* species in the gingival crevice of the squirrel monkey. *Infection and Immunity*, *56*(9), 2392–2399.

Cranfield, M. R. (2008). Mountain gorilla research: The risk of disease transmission relative to the benefit from the perspective of ecosystem health. *American Journal of Primatology*, *70*, 751–754. https://doi.org/10.1002/ajp.20564

Eberle, R. (1992). Evidence for an alpha-herpesvirus indigenous to mountain gorillas. *Journal of Medical Primatology*, *21*(5), 246–251.

Eberle, R., & Hilliard, J. K. (1989). Serological evidence for variation in the incidence of herpesvirus infections in different species of apes. *Journal of Clinical Microbiology*, *27*(6), 1357–1366.

Ehrlich, J. C., Novikoff, A. B., Platt, R., & Essner, E. (1960). Hepatocellular lipofuscin and the pigment of chronic idiopathic jaundice. *Bulletin of New York Academy of Medicine*, *36*, 488–491.

Elgart, A. A. (2010). Dental wear, wear rate, and dental disease in the African apes. *American Journal of Primatology*, *72*(6), 481–491.

Emerson, J. A., Adkesson, M. J., Colegrove, K. M., Burdick, S. K., & Langan, J. N. (2014). Ménétrier's disease-like hypertrophic gastritis in two red-capped mangabeys (*Cercocebus torquatus*). *Veterinary Quarterly*, *34*(1), 29–36.

Evans, T. S., Gilardi, K. V. K., Barry, P. A., Ssebide, B. J., Kinani, J. F., Nizeyimana, F., & Johnson, C. K. (2016). Detection of viruses using discarded plants from wild mountain gorillas and golden monkeys. *American Journal of Primatology*, *78*, 1222–1234. https://doi.org/10.1002/ajp.22576

Fossey, D. (1983). *Gorillas in the mist*. Houghton Mifflin Company.

Freeman, A. S., Kinsella, J. M., Cipolletta, C., Deem, S. L., & Karesh, W. B. (2004). Endoparasites of western lowland gorillas (*Gorilla gorilla gorilla*) at Bai Hokou, Central African Republic. *Journal of Wildlife Diseases*, *40*(4), 775–781.

Gardiner, C. H., Fayer, R., & Dubey, J. P. (2001). *An atlas of protozoan parasites in animal tissues* (2nd ed.) Armed Forces Institute of Pathology.

Gardiner, C. H., & Poynton, S. L. (1999). *An atlas of metazoan parasites in animal tissues*. Armed Forces Institute of Pathology.

Graczyk, T. K., & Cranfield, M. R. (2003). Coprophagy and intestinal parasites: implications to human-habituated mountain gorillas (*Gorilla gorilla beringei*) of the Virunga mountains and Bwindi Impenetrable Forest. *Primate Conservation*, *19*, 58–64.

Graczyk, T. K., Da Silva, A. J., Cranfield, M. R., Nizeyi, J. B., Kalema, G. R. N. N., & Pieniazek, N. J. (2001). *Cryptosporidium parvum* genotype 2 infections in free-ranging mountain gorillas (*Gorilla gorilla beringei*) of the Bwindi Impenetrable National Park, Uganda. *Parasitology Research*, *87*, 368–370.

Graczyk, T. K., Lowenstine, L. J., & Cranfield, M. R. (1999). *Capillaria hepatica* (Nematoda) infections in human-habituated mountain gorillas (*Gorilla gorilla beringei*) of the Parc national des Volcans, Rwanda. *The Journal of Parasitology*, *85*(6), 1168–1170.

Graczyk, T. K., Nizeyi, J. B., Da Silva, A. J., Moura, I. N., Pieniazek, N. J., Cranfield, M. R., & Lindquist, H. D. (2002). A single genotype of

Encephalitozoon intestinalis infects free-ranging gorillas and people sharing their habitats in Uganda. *Parasitology Research*, *88*(10), 926–931.

Haesebrouck, F., Pasmans, F., Flahou, B., Chiers, K., Baele, M., Meyns, T., Decostere, A., & Ducatelle, R. (2009). Gastric helicobacters in domestic animals and nonhuman primates and their significance for human health. *Clinical Microbiology Reviews*, *22*(2), 202–223. https://doi.org/10.1128/CMR.00041-08

Hassell, J. M., Zimmerman, D., Cranfield, M. R., Gilardi, K., Mudakikwa, A., Ramer, J., Nyirakaragire, E., & Lowenstine, L. J. (2017). Morbidity and mortality in infant mountain gorillas (*Gorilla beringei beringei*): A 46-year retrospective review. *American Journal of Primatology*, *79*(10), e22686. https://doi.org/10.1002/ajp.22686

Hickey, J. R., Basabose, A., Gilardi, K. V., Greer, D., Nampindo, S., Robbins, M. M., & Stoinski, T. S. (2018). *Gorilla beringei* ssp. beringei. *The IUCN Red List of Threatened Species*, 2018. https://doi.org/10.2305/IUCN.UK.20182.RLTS.T39999A17989719.en

Hickey, J. R., Granjon, A. C., Vigilant, L., Eckardt, W., Gilardi, K. V., Cranfield, M., & Robbins, M. M. (2019). *Virunga 2015–2016 surveys: Monitoring mountain gorillas, other select mammals, and illegal activities*. GVTC, IGCP & Partners.

Hogan, J. N., Miller, W. A., Cranfield, M. R., Ramer, J., Hassell, J., Noheri, J. B., Conrad, P. A., & Gilardi, K. V. (2014). Giardia in mountain gorillas (*Gorilla beringei beringei*), forest buffalo (*Syncerus caffer*), and domestic cattle in Volcanoes National Park, Rwanda. *Journal of Wildlife Diseases*, *50*(1), 21–30. https://doi.org/10.7589/2012-09-229

Imai, S., Ikeda, S., Collet, J. Y., & Bonhomme, A. (1991). Entodiniomorphid ciliates from the wild lowland gorilla with the description of a new genus and three new species. *European Journal of Protistology*, *26*(3–4), 270–278.

Ito, A., Eckardt, W., Stoinski, T. S., Gillespie, T. R., & Tokiwa, T. (2016). Prototapirella ciliates from wild habituated Virunga mountain gorillas (*Gorilla beringei beringei*) in Rwanda with the descriptions of two new species. *European Journal of Protistology*, *54*, 47–58. https://doi.org/10.1016/j.ejop.2016.04.001

Ito, A., Eckardt, W., Stoinski, T. S., Gillespie, T. R., & Tokiwa, T. (2018). Three new Troglodytella and a new Gorilloflasca ciliates (Entodiniomorphida) from mountain gorillas (*Gorilla beringei beringei*) in Rwanda. *European Journal of Protistology*, *65*, 42–56.

Kalema-Zikusoka, G., Rothman, J. M., & Fox, M. T. (2005). Intestinal parasites and bacteria of mountain gorillas (*Gorilla gorilla beringei*) in Bwindi Impenetrable National Park, Uganda. *Primates*, *46*(1), 59–63.

Krief, S., Jamart, A., Mahé, S., Leendertz, F. H., Mätz-Rensing, K., Crespeau, F., Bain, O., & Guillot, J. (2008). Clinical and pathologic manifestation of oesophagostomosis in African great apes: Does self-medication in wild apes influence disease progression? *Journal of Medical Primatology*, *37*(4), 188–195.

Lovell, N. C. (1990). Skeletal and dental pathology of free-ranging mountain gorillas. *American Journal of Physical Anthropology*, *81*(3), 399–412.

Lowenstine, L. J., McManamon, R., & Terio, K. A. (2015). Comparative pathology of aging great apes: Bonobos, chimpanzees, gorillas and orangutans. *Veterinary Pathology*, *53*, 250–276.

Lowenstine, L. J., & Munson, L. (1999). Iron overload in the animal kingdom. In M. E. Fowler & E. R. Miller (Eds.), *Zoo and wild animal medicine: Current therapy* (Vol. 4, pp. 260–268). W. B. Saunders Co.

Manyama, M., Rolian, C., Gilyoma, J., Magori, C. C., Mjema, K., Mazyala, E., Kimwaga, E., & Hallgrimsson, B. (2011). An assessment of orofacial clefts in Tanzania. *BMC Oral Health*, *11*(5), 5.

McGavin, M. D., & Zachary, F. J. (2007). *Pathological basis of veterinary disease* (4th ed.). Mosby Elsevier.

Mombelli, A., Tonetti, M., Lehmann, B., & Lang, N. P. (1996). Topographic distribution of black-pigmenting anaerobes before and after periodontal treatment by local delivery of tetracycline. *Journal of Clinical Periodontology*, *23*(10), 906–913.

Muhangi, D. (2003). *Prevalence of Microsporidium and Cryptosporidium species in ruminants and mountain gorillas (Gorilla gorilla beringei) in Bwindi Impenetrable National Park* (Bachelor of Veterinary Medicine). Makerere University.

Narama, I., Tsuchitani, M., Umemura, T., & Tsuruta, M. (1983). The morphogenesis of a papillomatous gastric polyp in the crab-eating monkey (*Macaca fascicularis*). *Journal of Comparative Pathology*, *93*(2), 195–203.

Nizeyi, J. B., Mwebe, R., Nanteza, A., Cranfield, M. R., Kalema, G. R. N. N., & Graczyk, T. K. (1999). *Cryptosporidum* species and *Giardia* species infection in mountain gorillas (*Gorilla gorilla beringei*) of the Bwindi Impenetrable National Park. *The Journal of Parasitology*, *85*, 1084–1088.

Nizeyi, J. B., Rwego, I. B., Erume, J., Cranfield, M. R., Kalema, G. R. N. N., & Graczyk, T. K. (2001). *Campylobacter*, *Salmonella* and *Shigella* infections in free-ranging human-habituated mountain gorillas (*Gorilla gorilla beringei*) of the Bwindi and Mgahinga national parks, Uganda. *Journal of Wildlife Diseases*, *37*, 239–244.

Nutter, F. B., Whittier, C. A., & Lowenstine, L. J. (2005). *Cleft palate in a neonatal Virunga mountain gorilla (Gorilla beringei beringei)*. Paper presented at the 54th Annual Wildlife Disease Association Conference, Cairns, Queensland, Australia.

Pavone, S., Veronesi, F., Genchi, C., Fioretti, D. P., Brianti, E., & Mandara, M. T. (2011). Pathological changes caused by *Anoplocephala perfoliata* in the mucosa/submucosa and in the enteric nervous system of equine ileocecal junction. *Veterinary Parasitology*, *176*(1), 43–52.

Pond, C. L., Newcomer, C. E., & Anver, M. R. (1982). Acute gastric dilatation in nonhuman primates: Review and case studies. *Veterinary Pathology*, *19*(7 suppl), 126–133. https://vet.sagepub.com/content/19/7_suppl/126.abstract

R Core Team. (2016). *R: A language and environment for statistical computing R Foundation for Statistical Computing*. https://www.R-project.org/

Redmond, I. (1983). Summary of parasitology research. In D. Fossey (Ed.), *Gorillas in the mist* (pp. 271–286). Houghton Mifflin Co.

Reichart, P. A., Lenz, H., König, H., Becker, J., & Mohr, U. (1985). The black layer on the teeth of betel chewers: A light microscopic, microradiographic and electronmicroscopic study. *Journal of Oral Pathology and Medicine*, *14*(6), 466–475.

Reid, J. A., Beeley, J. A., & MacDonald, D. G. (1977). Investigations into black extrinsic tooth stain. *Journal of Dental Research*, *56*(8), 895–899.

Reyes, J. L., Lopes, F., Leung, G., Mancini, N. L., Matisz, C. E., Wang, A., Thomson, E. A., Graves, N., Gilleard, J., & McKay, D. M. (2016). Treatment with cestode parasite antigens results in recruitment of CCR2+ myeloid cells, the adoptive transfer of which ameliorates colitis. *Infection and Immunity*, *84*(12), 3471–3483. https://iai.asm.org/content/iai/84/12/3471.full.pdf

Rothman, J. M., Bowman, E. E., Kalema-Zikusoka, G., & Nkurunungi, J. B. (2006). The parasites of the gorillas in the Bwindi Impenetrable National Park, Uganda. In N. E. Newton-Fischer, H. Notman, J. D. Paterson, & V. Reynolds (Eds.), *Primates of Western Uganda (developments in primatology)* (pp. 171–192). Springer.

Rothman, J. M., Pell, A. N., & Bowman, D. D. (2008). Host–parasite ecology of the helminths in mountain gorillas. *The Journal of Parasitology*, *94*(4), 834–840.

Rubio, C. A., Dick, E. J. J., & Hubbard, G. B. (2008). The frequency of lymphocytic gastritis in baboons. *In Vivo*, *22*(1), 101–104.

Saba, C., Solidani, M., Berlutti, F., Vestri, A., Ottolenghi, L., & Polimeni, A. (2006). Black stains in the mixed dentition: A PCR microbiological study of the etiopathogenic bacteria. *Journal of Clinical Pediatric Dentistry*, *30*(3), 219–224.

Sleeman, J. M., Meader, L. L., Mudakikwa, A. B., Foster, J. W., & Patton, S. (2000). Gastrointestinal parasites of mountain gorillas (*Gorilla gorilla beringei*) in the Parc National des Volcans, Rwanda. *Journal of Zoo Wildlife Medicine*, *31*(3), 322–328.

Smith, P., Mangan, N. E., Walsh, C. M., Fallon, R. E., McKenzie, A. N., van Rooijen, N., & Fallon, P. G. (2007). Infection with a helminth parasite prevents experimental colitis via a macrophage-mediated mechanism. *Journal of Immunology (Baltimore, Md.: 1950)*, *178*(7), 4557–4566. https://doi.org/10.4049/jimmunol.178.7.4557

Storey, P. A., Anemana, S., van Oostayen, J. A., Polderman, A. M., & Magnussen, P. (2000). Ultrasound diagnosis of oesophagostomiasis. *The British Journal of Radiology*, *73*(867), 328–332. https://doi.org/10.1259/bjr.73.867.10817053. https://www.birpublications.org

Strong, V., Baiker, K., Brennan, M. L., Redrobe, S., Rietkerk, F., Cobb, M., & White, K. A. (2017). Retrospective review of western lowland gorilla (*Gorilla gorilla gorilla*) mortality in European zoologic collections between 2004 and 2014. *Journal of Zoo Wildlife Medicine*, *48*(2), 277–286. https://doi.org/10.1638/2016-0132R.1

Thapar, N., & Sanderson, I. R. (2004). Diarrhoea in children: An interface between developing and developed countries. *Lancet*, *363*, 641–653.

Vannella, L., Lahner, E., & Annibale, B. (2012). Risk for gastric neoplasias in patients with chronic atrophic gastritis: A critical reappraisal. *World Journal of Gastroenterology*, *18*(12), 1279–1285.

Watts, D. P., & Pusey, A. E. (1993). Behavior of juvenile and adolescent great apes. In M. E. Pereira & L. A. Fairbanks (Eds.), *Juvenile primates: Life history, development and behavior* (pp. 148–172). Oxford University Press.

WWF. (2019). Global mountain gorilla population count grows to 1,063. *WWF Newsletter*.

Yasuda, M., Inoue, T., Ueno, M., Morita, H., Hayashimoto, N., Kawai, K., & Itoh, T. (2015). A case of nontraumatic gas gangrene in a common marmoset (*Callithrix jacchus*). *Journal of Veterinary Medical Science*, *77*(12), 1673–1676. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4710728/

How to cite this article: Muhangi, D., Gardiner, C. H., Ojok, L., Cranfield, M. R., Gilardi, K. V. K., Mudakikwa, A. B., & Lowenstine, L. J. (2021). Pathological lesions of the digestive tract in free-ranging mountain gorillas (*Gorilla beringei beringei*). *American Journal of Primatology*, e23290. https://doi.org/10.1002/ajp.23290