

Isolation of *Escherichia fergusonii* from the feces and internal organs of a goat with diarrhea

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Abstract — A fecal sample from a 42-year-old goat with a 2-month history of poor weight gain and diarrhea yielded a moderate growth of an organism resembling *Salmonella* spp. on MacConkey agar. The organism was identified as *Escherichia fergusonii*. The animal was euthanized. Samples of intestine, lung, liver, and kidney yielded the same organism, *E. fergusonii*.

Résumé — Isolement d'*Escherichia fergusonii* à partir des fèces et des organes internes d'une chèvre diarrhéique. Un échantillon fécal d'une chèvre âgée de 4,5 ans présentant une histoire de faible gain de poids et de diarrhée a été cultivé sur gélose MacConkey et a révélé une croissance moyenne d'un organisme ressemblant à *Salmonella* spp. L'organisme a été identifié comme étant *Escherichia fergusonii*. L'animal a été euthanasié. Les échantillons d'intestins, de poumons, de foie et de reins contenaient le même organisme : *E. Fergusonii*.

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Diarrhea in goats may be produced by a number of pathogens, toxic substances, and nutritional causes. The most common causes of diarrhea in adult goats are parasite, including coccidia, *Clostridium perfringens* type D, and *Salmonella* spp. infection, nutritional factors; toxic agents; liver disease; and copper deficiency. An uncommon cause is Johne's disease, but diarrhea may occur in the terminal stages of the disease (1). This case in a goat with diarrhea and wasting yielded *Escherichia fergusonii* in feces and internal organs. This organism, a potential pathogen, resembles *Salmonella* organisms on culture plates, particularly on MacConkey agar.

A 4 1/2-year-old, mixed breed, male goat, weighing 25 kg, was donated to the Atlantic Veterinary College (AVC). There was a 2-month history of poor weight gain and diarrhea. There were approximately 30 goats on the farm. Over the previous few months, several deaths had occurred within the herd and the goats had similar clinical signs. The only new addition to the herd had been a boar purchased at same time as this goat's initial problems began. The entire herd was treated, PO, with a double dose of the injectable form of ivermectin (Ivomec; Merck Agvet, Kirkland, Quebec) at that time for suspected parasitism. During the previous winter, coccidiosis on the farm had been treated with amprolium (Amprol; Merck Agvet), 20 mg/kg bodyweight (BW), PO, q24h for 7 d. The goats were fed barley twice daily

and had access to free choice hay and pasture. The goats continued to lose weight despite an increase in feed consumption.

On physical examination (PE), the goat reported herein was recumbent but alert and responsive with a body condition score of 1/5. Mucous membranes were pale and tacky with a prolonged (> 2 s) capillary refill time. Dehydration was estimated to be ~7%. The goat was tachycardic with a heart rate of 132 beats/min, and a grade 3/6 systolic cardiac murmur was auscultated. Thoracic auscultation also revealed harsh lung sounds dorsally but no sounds ventrally. A cough could be elicited easily. During the PE, the goat experienced diarrhea that was foul smelling. The owner elected euthanasia and the carcass was submitted for postmortem examination.

At necropsy, the animal was found to be in poor body condition with moderate muscle wasting and serous atrophy of perineal, mesenteric, and coronary fat. Grossly, the mesenteric lymph nodes were enlarged (2×) and edematous. The walls of the small intestine were flaccid and the lumen contained a considerable amount of liquid ingesta. Longitudinal sections of long bones revealed thin cortices and a notably reduced number and thickness of bone trabeculae in the epiphyses. The right ear showed signs of mild otitis externa, but close inspection of temporal bones did not show any evidence of inflammation in the inner or middle ears. Samples of intestine, liver, kidneys, and lung were submitted for bacteriological culture, and feces were submitted for parasitologic examination. Samples of lung, liver, heart, kidneys, rumen, abomasum, intestine, mesenteric lymph nodes, skeletal muscle, and brain were fixed in buffered formalin and processed for histopathologic examination.

Microscopically, the lungs had moderately extensive intrapulmonary hemorrhages, without evidence of erythrophagocytosis. The axial interstitium was distended with protein-rich

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edematous fluid. The liver had a few focal areas of necrosis and inflammation, characterized by coagulation necrosis and infiltrates of neutrophils. Periacinar hepatocytes were slightly vacuolated and the portal triads had mild fibrosis with occasional mononuclear cell infiltrates. The mesenteric lymph nodes were edematous with moderate sinus histiocytosis and many of these macrophages were filled with a yellow pigment that stained positive for hemosiderin with Prussian-blue stain. Some subcapsular lymphatic vessels contained large (> 100 µm), oval, immature *Eimeria* spp. schizonts. The mucosa of both large and small intestines was diffusely infiltrated with mononuclear cells, largely lymphocytes and plasma cells. Some sections of the small intestine had developing oocysts of *Eimeria* spp. in the cytoplasm of the enterocytes. Mycobacteria were not observed in acid fast-stained sections of intestine and mesenteric lymph nodes.

Low to moderate numbers of protozoan cysts (*Eimeria* spp. oocysts, *Giardia* spp. cysts) and nematode eggs (*Trichostrongyle*-type and *Trichuris* spp. eggs) were detected on sugar centrifugal flotation and examination of the fecal sample.

The initial fecal sample from the live animal was submitted for bacteriologic study. Culture on blood agar and MacConkey agar yielded moderate growth of nonlactose fermenting (NLF) colonies resembling those of *Salmonella* spp., mixed with the growth of *E. coli*. Culture of the fecal sample in modified semi-solid Rappaport Vassiliadis medium (Oxoid, Nepean, Ontario) did not yield *Salmonella* spp. The NLF colonies gave an alkaline/acid (K/A) reaction in triple sugar iron agar (Oxoid), a positive reaction for indole, and a negative reaction for urease and oxidase. The isolate was inoculated into a bacterial identification strip (API 20E strip; Analytab Products, BioMerieux Canada, St. Laurent, Quebec), which identified it as *E. fergusonii* with 98.9% probability. Antimicrobial susceptibility tests were done by using the Kirby-Bauer disk diffusion method (2), and the zone sizes were interpreted according to the National Committee for Clinical Laboratory Standards (NCCLS) for bacteria isolated from animals (3). The isolate showed susceptibility to ceftiofur, oxytetracycline, tilmicosin, trimethoprim-sulfa, and florfenicol. It was moderately susceptible to streptomycin, and resistant to erythromycin and penicillin. The postmortem intestinal sample yielded a heavy growth of *E. fergusonii*, with light growth from lung, liver, and kidney. The growth of *E. fergusonii* from liver was almost pure, with only 1 additional colony of different morphology, which was identified as *E. coli*. Identification by bacterial identification strip (API 20E), and the antimicrobial susceptibility results were identical to those from the isolate from the live animal.

Escherichia fergusonii, a member of the family *Enterobacteriaceae*, is closely related to *E. coli*, but it does not ferment lactose; therefore, the colonies on MacConkey agar resembled those of *Salmonella* spp. Funke et al (4) isolated *E. fergusonii* from gall bladder fluid, blood cultures, and feces

of a human patient with pancreatic carcinoma and cholangiosepsis. Bain and Green (5) reported isolation of this bacterium from an adult cow with severe scour and rapid weight loss. The isolation of *E. fergusonii* from a diarrheic cow, which was clinically suggestive of salmonellosis (5), indicated that this organism may be significant in diarrheic conditions in animals. In a study of 50 cases of human diarrhea, 4% of isolates contained *E. fergusonii*, all of which were enterotoxigenic, as evidenced by fluid accumulation in rat ileal loops (6). Recently, *E. fergusonii* has been reported as a pathogen causing enteritis in ostriches (7). Parasites may affect the balance of the bacterial flora of the intestines, and intestinal coccidial infection has been reported to influence intestinal microflora composition in both poultry and mammals (8–10). Infection with a mix of 5 *Eimeria* spp. was reported to decrease the gram-positive and increase the gram-negative bacterial fauna in Nubian goats (11). Increased enterobacteria fecal shedding associated with *Eimeria* infection has been reported in rabbits, sheep, and goats (12,13). To what extent the coccidial infection present in the goat in this case affected the composition of the gut fauna and predisposed the animal to *E. fergusonii* infection remains speculative. CVJ

References

1. Matthews J. Diseases of the Goat, 2nd ed. London: Blackwell Sci, 1999:224–225.
2. Barry AL, Thornsberry C. Susceptibility tests: Diffusion test procedures. In: Lennette EH, Balows A, Hausler Jr WJ, Shadomy HJ, eds. Manual of Clinical Microbiology. 4th ed. Washington, DC: Am Soc Microbiol, 1985:978–987.
3. National Committee for Clinical Laboratory Standards. Performance Standards for Antimicrobial Disk and Dilution Susceptibility Tests for Bacteria Isolated from Animals; Approved Standard, 2nd ed. NCCLS document M31-A2. 2002:22(6).
4. Funke G, Hany A, Altwegg M. Isolation of *Escherichia fergusonii* from four different sites in a patient with pancreatic carcinoma and cholangiosepsis. J Clin Microbiol 1993;31:2201–2203.
5. Bain MS, Green CC. Isolation of *Escherichia fergusonii* in cases clinically suggestive of salmonellosis. Vet Rec 1999;144:511.
6. Chaudhury A, Nath G, Tikoo A, Sanyal SC. Enteropathogenicity and antimicrobial susceptibility of new *Escherichia* spp. J Diarrhoeal Dis Res 1999;17:85–87.
7. Herraiz P, Rodriguez AF, Espinosa de los Monteros A, et al. Fibrinonecrotic typhlitis caused by *Escherichia fergusonii* in ostriches (*Struthio camelus*). Avian Dis 2005;49:167–169.
8. Turk DE, Littlejohn VP. Coccidial infections and gut microflora. Poultry Sci 1987;66:1466–1469.
9. Baba E, Wakeshima H, Fukui K, Fukata T, Arakawa A. Adhesion of bacteria to the cecal mucosal surface of conventional and germ-free chickens infected with *Eimeria tenella*. Am J Vet Res 1992;53:194–197.
10. Yvore P. Interaction between coccidia, gut microflora and intestinal parasites in mammals, INRA: Coccidia and Intestinal Coccidiomorphs — 5th Int Coccidiosis Conf, Tours, France, 1989:183–192.
11. Mohammed RA, Idris OA, El Sanousi SM, Abdelsalam EB. The effect of coccidian infection on the gut microflora of Nubian goat kids. Dtsch Tierarztl Wochenschr 2000;107:414–416.
12. Licois D, Guillot JF. Evolution du nombre de colibacilles chez des laperaux atteints de coccidiose intestinale. Recl Med Vet 1980;156:555–560.
13. Vasilkova Z, Krupicer I, Legath J, Kovalkoviova N, Petko B. Coccidiosis of small ruminants in various regions of Slovakia. Acta Parasitol 2004;49:272–275.