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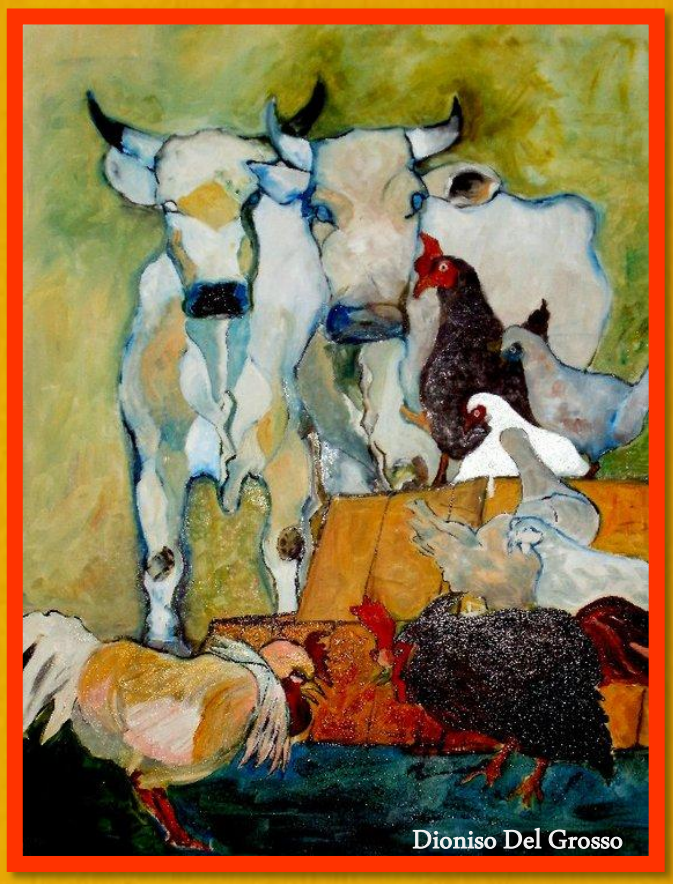

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FIRST DETECTION OF *Helicobacter canis* AND RELATED GASTRIC PATHOLOGY IN CHEETAHS (*Acinonyx jubatus*)

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Gastritis or, in general, gastro-intestinal diseases, causes significant morbidity and mortality in cheetahs (*Acinonyx jubatus*), especially in captive animals. The condition is characterized by vomit, diarrhea, anorexia, weight loss, until the death of the animal (1). In free-range cheetahs, clinical signs are weaker or even absent. Although currently a multifactorial condition is related to the pathogenesis of cheetah gastritis, four main factors interact in gastritis determinism: the lack of cheetahs genetic polymorphism; the captivity; the diet; and the presence of Gastric *Helicobacter*-like organisms (GHLOs) infection, in particular *Helicobacter acinonychis* and *Helicobacter heilmannii* (2). It is undoubted that the *Helicobacter* infection is always present in all samples of cheetahs gastric mucosa with gastritis of varying degrees and severity. Fecal samples from 18 cheetahs, with different severities of gastritis, were selected for this study. Nine wild cheetahs were located in Cheeath Conservation Fund (CCF), in Namibia, they had not evident clinical signs, with rare episodes of vomit, diarrhea and weight loss. Nine captive cheetahs, housed in different Italian Zoo Parks, had various degrees of gastritis clinically characterized by a going light syndrome, until the death of the animal. To detect *Helicobacter* species we used a highly sensitive and specific qualitative PCR assay. In addition, a subset of PCR products (= 9) was sequenced to confirm their identity: 60% of cases has been identified as *Helicobacter heilmannii* whereas 40% of cases has been identified as *Helicobacter canis*. From *Helicobacter canis* infected cases, two cheetahs showing severe clinical signs and subjected to the endoscopy, evidenced a multifocal and atrophic severe gastritis, with large numbers of inflammatory cells in both the superficial and deep regions of the lamina propria, as well as abundant intra-epithelial lymphocytes (IELs). Inflammatory cells consisted predominately of lymphocytes and plasma cells with variable numbers of large globule leukocytes. Disruption of normal glandular structure, loss of parietal cells, necrosis, and intraglandular neutrophils were also present, with a constant evidence of a heavy GHLOs colonization of the glands or free in the superficial mucus covering the mucosa. In both cases, neutrophils were a minor component of the inflammatory cell infiltrate. Atrophic gastritis characterized by large lymphoid aggregates at the base of the lamina propria, mucosal atrophy, and variable lamina proprial fibrosis were also seen in bioptic samples especially belonging to the antral region of the stomach. In our knowledge this could be the first report of *H. canis* detection from cheetahs with severe gastritis; previously this specie was isolated from feces of diarrheic or healthy dogs, cats, humans and sheep (3).

[1] Lobetti, R., Picard, J., Kriek, N., & Rogers, P. Prevalence of helicobacteriosis and gastritis in semicaptive cheetahs (*Acinonyx jubatus*). Journal of Zoo and Wildlife Medicine, 492-496, 1999. [2] Eaton, KA., Radin MJ., Kramer L., Wack R., Sherding R., Krakowka S., Morgan DR. Gastric spiral bacilli in captive cheetah. Scand. J. Gastroenterol. 26:38–42, 1991. [3] Sabry, M. A., Abdel-Moein, K. A., & Seleem, A. Evidence of zoonotic transmission of *Helicobacter canis* between sheep and human contacts. Vector-Borne and Zoonotic Diseases, 16:650-653, 2016.