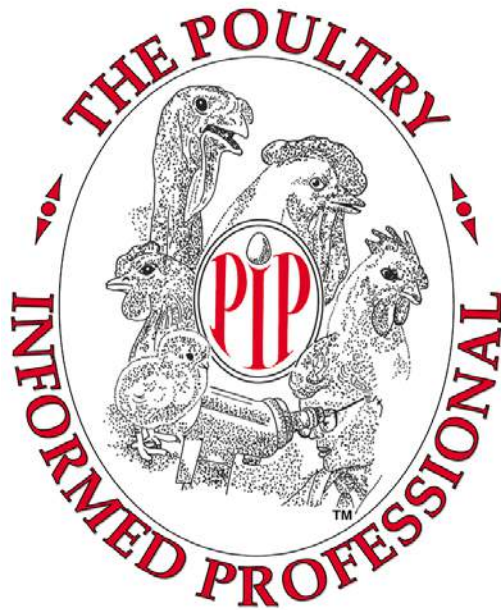


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Causes of Gizzard Erosion and Proventriculitis in Broilers

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Many field veterinarians report the presence of gizzard erosion in broilers and commercial layers as a result of propitious environmental conditions for mold growth and the use of feed ingredients contaminated with mycotoxins. Unfortunately, only in a few cases the clinical diagnosis is reconfirmed using the laboratory, therefore not much attention is placed on this finding unless the production parameters are negatively affected. Proventriculitis or inflammation of the proventriculus is not reported as frequently as gizzard erosion and has been associated with Marek's disease (MD) and Reovirus infections. In this article, the term proventriculitis will be used to name the inflammation and enlargement (proventriculosis) of the organ. Proventriculitis is a naturally occurring disease of broilers characterized by inflammation, dilatation and thinning of the organ, decreasing the speed of feed passage through the

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Broiler Performance Data (Region) – Live Production Cost

	SW	Midwest	Southeast	Mid-Atlantic	S-Central
Feed Cost/ton w/o color (\$)	198.52	190.59	200.1	201.58	199.65
Feed cost /lb meat (c)	18.18	16.19	17.92	19.06	18.56
Days to 4.6 lbs	42	40	41	41	42
Chick cost / lb (c)	4.11	4.60	4.55	3.81	4.07
Vac-Med cost/lb (c)	0.05	0.06	0.08	0.10	0.06
WB & ½ parts condemn. Cost/lb	0.16	0.10	0.10	0.16	0.15
% mortality	3.58	3.16	3.70	3.72	3.30
Sq.Ft. @ placement	0.81	0.79	0.82	0.95	0.86
Lbs/sq. ft.	7.40	6.78	6.83	7.18	7.39
Downtime (days)	15	11	14	14	11

Data for week ending September 28, 2007

lumen and predisposing to organ rupture during evisceration of the carcass. Spillage of the retained ingesta from the torn proventriculus into the body cavity causes contamination and in some cases condemnation of the whole carcass. This contamination has a negative effect on the production cost, since it is necessary to reduce the speed of the line at the processing plant to process again the broiler carcasses by washing, which represents an increase in labor cost. Several publications in the US have demonstrated a higher production cost in commercial broilers affected by this condition, with an increase of up to 10 points in feed conversion.

Anatomical and physiological characteristics of both organs.

The proventriculus or glandular stomach produces hydrochloric acid and pepsin, an enzyme that breaks down proteins. Given its small size, feed does not stay there for too long and no digestion occurs in his lumen. The gizzard is a highly muscular organ capable of producing high pressure on the feed, causing its trituration, without secreting any type of enzyme. The gizzard presents a hard lining composed of keratin, necessary for physical processing of feed and to protect the underlying mucosa against the degrading effects of the acid and pepsin produced by the proventriculus.

CAUSES OF GIZZARD EROSION

Mycotoxins. T-2 toxin, monoacetoxyscirpenol (MAS) and diacetoxyscirpenol (DAS) are three mycotoxins from the Tricothecenes group capable of causing gizzard erosion, oral lesions and intestinal hemorrhage in the gastrointestinal tract. T-2 toxin and DAS are considered the most caustic in the group and apparently produce gizzard erosion because of their caustic effect when touching the organ walls. It is important to point out that the caustic effect is more pronounced in the mouth (tongue, beak, palate) than in other anatomical areas of the digestive system (esophagus, crop, intestines, etc.) The presence of saliva in the mouth facilitates the adherence of these mycotoxins, which are capable of dissolving the cellular protoplasm of the cells located in that area.

Another factor to take into consideration in the mechanism of action of these mycotoxins is that after their absorption in the intestines they pass to the blood and later to the saliva, causing lesions in the mouth and possibly in the gizzard.

Copper. Due to its low cost in many countries, 0.5 to 1 kg of copper sulfate (25% Cu) per metric ton (MT) is added to the feed as a grow promoter or as a fungistatic agent. 0.5 Kg/MT of this product represents 125 g of copper/MT and 1 Kg/MT equals 250 g of copper/MT. The presence of gizzard erosion has been reported in birds consuming 250 g of copper/MT. An important factor to take into consideration to prevent this problem is to avoid overdosing and to choose a reliable source that will not form clumps in the feed.



Biogenic amines. These are by-products of the bacterial degradation of some amino acids, found mainly in ingredients of animal origin. The most common compounds in this group include cadaverine, tryptamine, histamine, putrescine, agmatine, tyramine, and phenylalanine. Several bacteria can transform the amino acid histidine into histamine, one of the biogenic amines mostly associated with the presence of poor performance in chickens. Histamine stimulates the receptors located in the proventricular glands increasing the hydrochloric acid secretion and causing superficial gizzard erosion. The correlation between the presence of biogenic amines and poor results in broilers has been observed in

the Southeast of the U.S., including proventricular enlargement and hypertrophy of the papillary glands. In spite of these reports, most scientific papers published until this moment has not established a direct correlation between the presence of biogenic amines in the feed and gizzard erosion or poor performance.

Fish Meal (Gizzerosine) Fish meal always has some potential of causing certain level of gizzard erosion. The lesions reported can include the presence of small cracks in the gizzard up to severe erosion and hemorrhage. When fish meal is overheated during processing, the histidine or histamine present in the meal can react with lysine forming a chemical compound called gizzerosine. Gizzerosine is not a biogenic amine and is 10 times as potent as histamine in stimulating acid production by the proventriculus. Different publications have demonstrated the interaction between gizzerosine and mycotoxins, including reports showing potentiation of the lethal effects caused by gizzerosine when the feed is contaminated with high levels of aflatoxins.

Adenovirus. Several researchers in Japan have reported the association between gizzard erosion and this group of viruses. In a study that included 18 slaughter houses in that country, 13 plants reported gizzard erosion caused by Adenovirus, as demonstrated by histopathology, immunohistochemistry, and virus isolation from the gizzard. The presence of a specific antigen against Adenovirus group 1 was detected in the epithelial cells of the gizzard.

Other factors. Other factors capable of producing gizzard erosion, but not frequently present in commercial conditions, include feed starvation and sulphur amino acids deficiency.

CAUSES OF PROVENTRICULITIS

Proventriculitis associated with Gumboro's disease. For many years several researchers considered that the proventriculitis observed in birds affected by Gumboro's disease was caused by this virus. In many cases, acute lesions in the proventriculus are accompanied by the presence of the IBD (Infectious Bursal Disease) virus as demonstrated by immunohistochemistry and viral isolation. Lately a new virus has been identified and called "viral proventriculitis virus". This type of virus is transmissible from bird to bird when administered to susceptible chickens. Some investigators speculate that the severe immunosuppression caused by IBDV is responsible for the increase in virulence of microorganisms present in the proventriculus. Several field veterinarians, including one of the authors of this article, have observed that very virulent IBDV strains cause a greater incidence of proventriculitis than classical and variant strains. Microscopically, when proventriculitis is associated with IBD, the first damage reported is the presence of acute bursal necrosis. Three to 5 days later, it becomes a chronic follicular atrophy and in the next 5 to 8 days an acute proventriculitis that later becomes a flaccid tissue is observed.

MD. This virus causes a lymphomatous lesion responsible for the enlargement of the proventriculus. When this lesion is present, the organ is enlarged and becomes rigid, because of localized areas presenting lymphocyte infiltration inside and between the organ glands. Sometimes, the presence of proventriculitis is erroneously thought to be caused only by MD. In order to reconfirm this disease it is important to look for the typical lesions caused by MD in other organs as well as the microscopic lesions that differentiate it.

Reovirus. In the 70's this type of virus was associated with the so-called "malabsorption syndrome" in broilers in several regions around the world. Proventriculitis was the most common lesion reported at that time. In the US two strains have been identified as prototype: Strain S 1133, associated traditionally with tenosinovitis and vital arthritis; and strain SS 412, associated with malabsorption syndrome and proventriculitis.

CPA. The cyclopiazonic acid is a metabolite of *Aspergillus flavus*, one of the most important molds producing aflatoxins in grains and feed. Fungi from the *Penicillium* genus also can produce this mycotoxin. In field conditions, birds presenting typical lesions caused by mycotoxins can also present proventriculitis produced by CPA. In experimental conditions, high levels of CPA (50 a 100 ppm) have caused in-

flammation of the epithelial mucosa, crop necrosis, proventriculitis and hyperplasia of the proventricular mucosa.

Other causes. Biogenic amines can also cause proventriculitis under commercial conditions.

Differential diagnosis. Grossly, it is difficult to establish a differential diagnosis among the agents causing proventriculitis and gizzard erosion. Microscopically, in the case of proventriculitis, it is theoretically possible to differentiate the lesions caused by a microbial agent from those of a non-viral etiology (biogenic amines, CPA, etc.). Usually, viral etiologic agents capable of causing proventriculitis as reovirus, MD and AE, are characterized by showing a lymphoid infiltration. However, when microbial agents do not cause the lesion, this type of inflammatory response is not detected. Among the viral agents listed, the localization of the infiltration varies according to the type of virus facilitating a differential diagnosis, as well as the presence of typical viral lesions in other organs. In the case of reovirus and AE, the infiltration is present in the muscular wall of the organ and not in the gland, as it occurs in the lesions produced by MD. Another important difference in the lesions caused by MD consists on the presence of a lymphoid infiltrate in the serosa layer of the nerves located in the external wall of the proventriculus.

CONCLUSION.

When both conditions are detected in the field, an effective differential diagnosis will allow us to determine what measures must be taken to control the problem; that is the use of IBD vaccines, reduction or elimination of feed ingredients and/or the control of mycotoxins causing the lesions by adding a mycotoxin binder to the diet. It is important to quantify the economic impact of these conditions in order to determine the incidence in the production cost. There is no doubt that feed contamination with mycotoxins play an important role in the presentation of proventriculitis and gizzard erosion. Several published experiments indicate that the association between biogenic amines and mycotoxins (Tricothecenes) cause proventriculitis, small bursas and spleens; and gizzard erosion and dilatation. In many cases, when biogenic amines were used alone, the clinical signs were not reproduced. Under commercial conditions, it is difficult to associate these lesions with only one etiologic agent, since they represent the result of the combination of several viral, nutritional and toxic agents (mycotoxins).

