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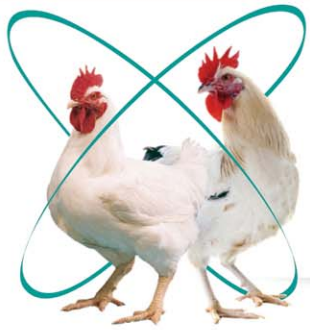

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CONTENTS

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INDEX OF ADVERTISEMENTS

Adenovirus infections in Poultry

Avantika Sharma, PhD scholar - Department of Veterinary Pathology
Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab.
Email: avantikavet123@gmail.com

Introduction: Adenoviruses are common infectious agents in poultry and wild birds worldwide. Many of the viruses replicate in healthy birds with little or no apparent signs of infection,although they can quickly take on the role of opportunistic pathogens when additional factors,particularly concurrent infectionsadversely affect the health of the avian host.

The first avian adenovirus was isolated in 1949 when material from a case of lumpy skin disease in cattle was inoculated into embryonated chicken eggs.

Most of the virus replicate readily in avian cultures derived from tissues,such as liver or kidney.

Replication take place in the nucleus and is accompanied by the development of intranuclear inclusions which may aid histopathological diagnosis.

Classification of adenoviruses:

Family: Adenoviridae

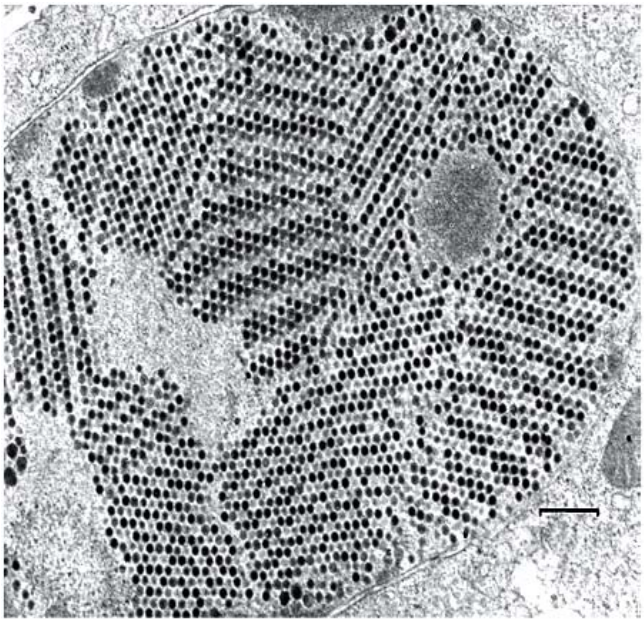
Genus: Mastadenovirus mammalian adenovirus Human, simian, bovine, equine, murine, porcine, ovine, caprine etc.

Genus: Aviadenovirus group 1 adenoviruses. Conventional adenoviruses of chicken, turkey, duck and goose. Five species A,B,C,D and E;12 serotypes

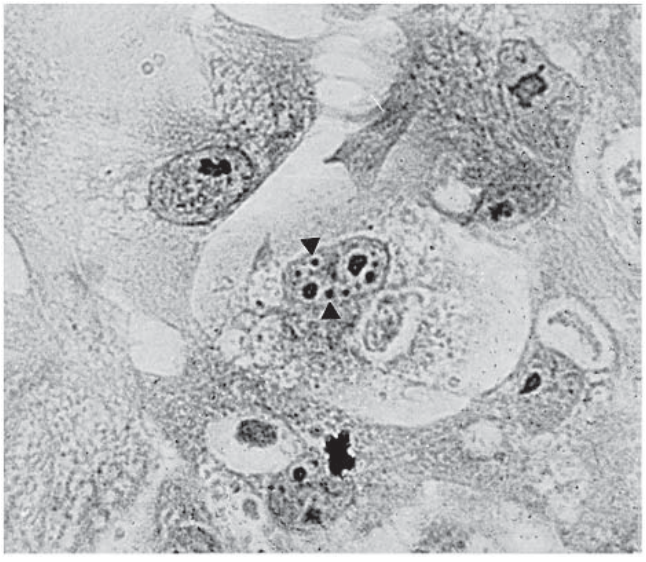
Genus : Siadenovirus group 2 avian adenoviruses, haemorrhagic enteritis virus(turkeys), Marble spleen disease(pheasants) and AASV(chicken).

Genus: Atadenovirus group 3 avian adenoviruses, Egg drop syndrome virus and related viruses.

(1) Adenovirus-infected chick liver cell culture (48 hours post-infection). Adenovirus particles almost fill the nucleus.



(2) H&E staining of chick kidney cells infected with adenovirus showing basophilic inclusions in the nucleus.



(A) Inclusion body hepatitis:

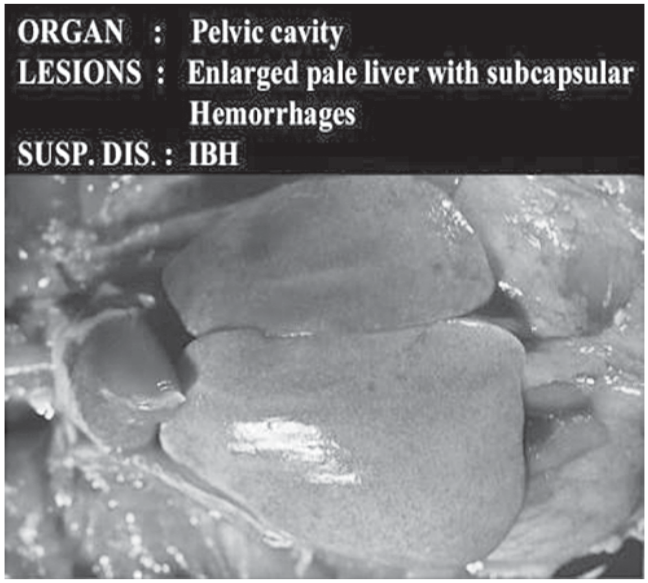
This disease is usually seen in meat-producing chickens 3-7 weeks of age but has been reported in birds as young as 7 days old and as old as 20 weeks. This disease is characterised by a sudden onset of mortality

Clinical Signs:

The disease is characterized by sudden onset of mortality. Mortality may reach 10% and sometimes as high as 30%. Anemia, jaundice of skin and subcutaneous fat and haemorrhages in various organs, especially the muscles.

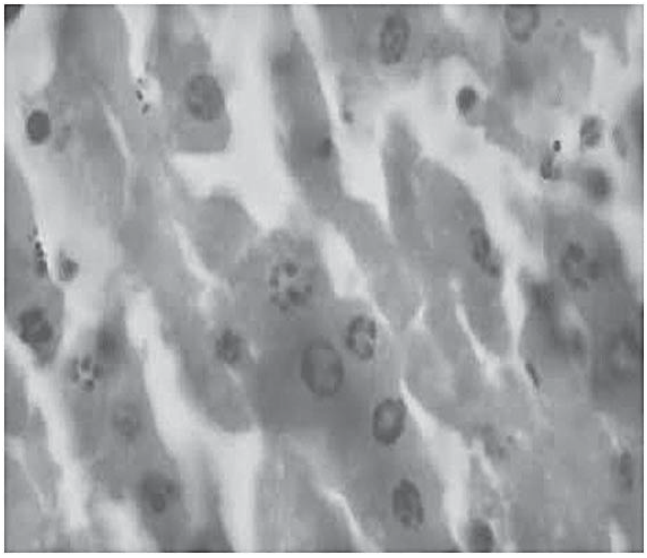
Gross Lesions:

The main lesions are pale,friable andswollen livers. Petechial or ecchymotic haemorrhages may be present in liver and skeletal muscles.



Histopathologic lesions:

There is a diffuse and generalised hepatitis with intranuclear inclusion bodies in the hepatocytes which are often eosinophilic or sometimes basophilic.



(B) Hydropercardium syndrome:

In 1987-a new condition-Hydropericardium syndrome or Angara disease-was recognized in Pakistan.

Gross Lesions:

There is accumulation of clear straw-coloured fluid in the pericardial sac,pulmonary edema, swollen and discoloured liver and enlarged kidneys with distended tubules showing degenerative changes.

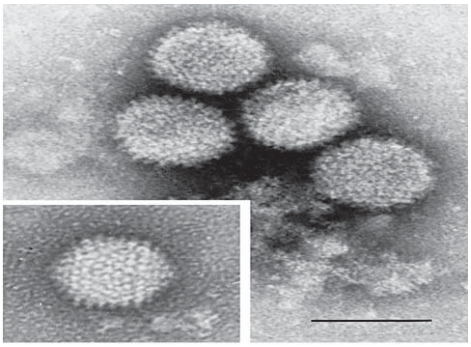
Histopathologic lesions:

Multiple areas of focal necrosis exist with mononuclear infiltration in the heart and liver. Basophilic inclusions are present in hepatocytes.

(C) Egg Drop Syndrome:

Since its initial description in 1976 by dutch workers,egg drop syndrome 1976 (EDS 76) has become a major cause of loss of egg production throughout the world.

It is caused by an adenovirus belonging to group 3.



Spread:

EDS outbreaks are divided into 3 types: 1.in the classical form,primary breeders are infected and the main method of spread is vertical through the embryonated egg.

The second pattern is the endemic form, in India 32.6% of poultry flocks were found to be infected.

The third type is sporadic form,this form results from introduction of infection from ducks,geese or any infection bird, either through direct contact or indirectly through drinking water contaminated with droppings.

Pathogenesis:

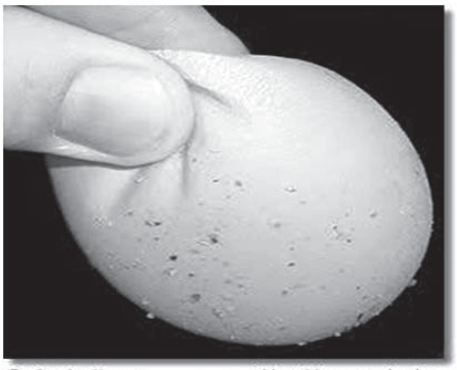
Following infection of laying hens,the virus grows to a limited extend in the nasal mucosa. This is followed by a viraemia with virus replication in lymphoid tissue throughout the body, 7u especially spleen and thymus.

The infundibulum of the oviduct is consistently affected.at 8 days after infection,there is massive viral replication in the pouch shell glands.

Clinical Signs:

Thin –shelled, soft-shelled, and shell-less eggs.There is drop in egg production.Diarrhoea may be due to an excess of oviduct secretion in droppings.Loss of colour in pigmented eggs.

Thin shelled eggs were often rough with a sandpaper-like texture or had a granular roughening of the shell at one end of the egg.



Soft-shell egg Used by permission

Gross lesions-

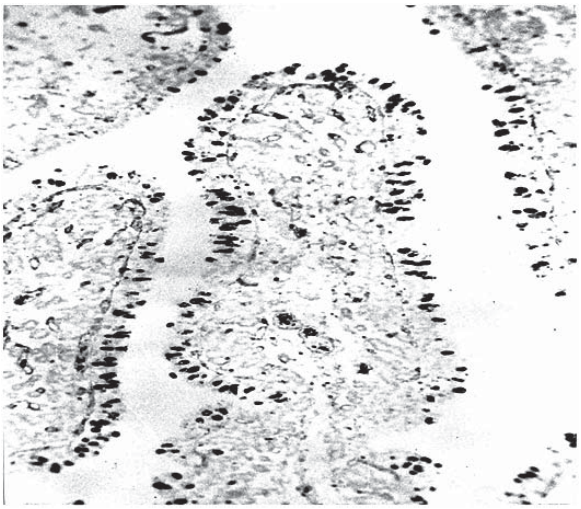
Inactive ovaries and atrophied oviducts. Uterine oedema was observed.

Mild splenomegaly, flaccid ovules and egg in various stages of formation in the abdominal cavity.

Histopathologic lesions:

The main pathological changes occur in the pouch shell gland of oviduct.Virus replicates in the

epithelial cell nuclei, and produce intranuclear inclusion bodies.Many affected cells are sloughed into the lumen.

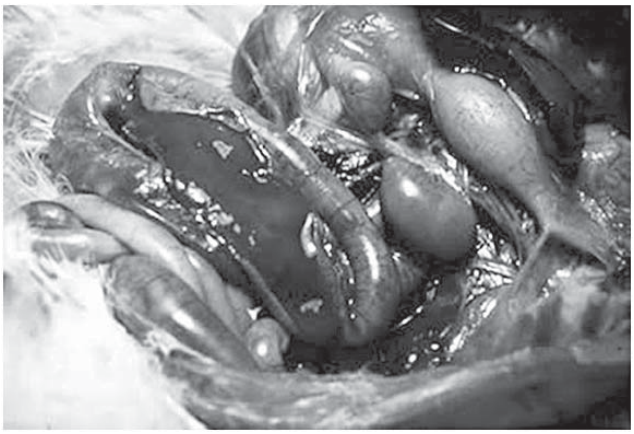


Diagnosis: The combination of a sudden fall in egg production,associated with thin-shelled and shell-less eggs in a flock of apparently healthy birds,is almost diagnostic. Others tests include HI test and ELISA

(D) Haemorrhagic Enteritis

HE is an acute viral disease of turkeys 4 weeks of age and older characteristized by depression, bloody droppings and death.

Until recently,turkey,pheasents and chickens were the only known natural hosts for HEV and related viruses. It is now thought that guinea fowl and psittacines may also be naturally infected.



Transmission: HEV can be transmitted by oral or cloacal innoculation of susceptible poults with infectious faeces.

In turkey clinical signs and mortality begin about 5-6 days after oral or cloacal.

Clinical signs:

Depression, bloody droppings and death.

Faeces containing frank blood are frequently present on the skin and feathers surrounding the vents of moribund and dead birds.

Bloody faeces may also be forced from the vent if moderate pressure is applied to the abdomen.

Gross lesions:

The small intestine is commonly distended,grossly discoloured and filled with bloody contents.

The intestinal mucosa is congested and in some individuals, covered with a yellow fibronecrotic.

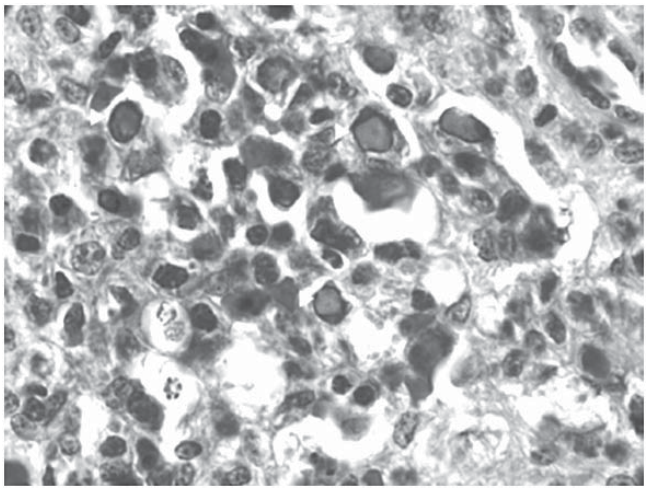
Spleen is enlarged, friable and mottled in appearance.

Histopathologic lesions:

Intranuclear inclusions can be found within mononuclear cells i.e. macrophages and lymphocytes.

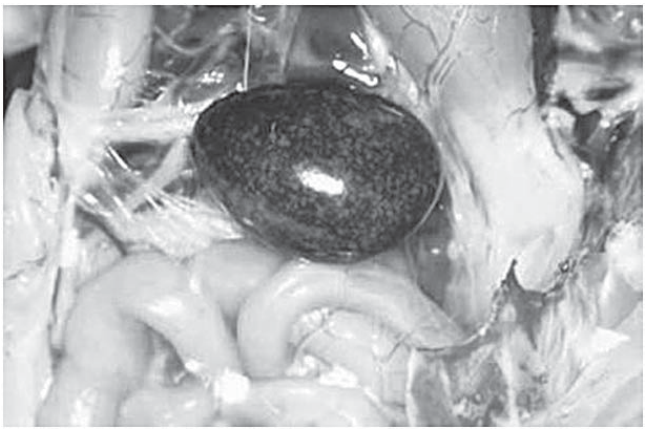
Section of small intestine from a turkey affected with haemorrhagic enteritis.

Section of spleen from a turkey affected with haemorrhagic enteritis. Nuclei of infected macrophages contain characteristic pale eosinophilic inclusions with marginated chromatin and eccentric nucleoli. H&E ×550.



(E) Marble Spleen Disease

It is an acute respiratory disease of pheasants characterized by depression,enlarged mottled spleens,pulmonary congestion and death.



Etiology:

It is a member of the family adenoviridae and the genus Siadenovirus.

It is endemic in area where turkey and pheasants are raised commercially.

The usual route of infection is oral and virus is often introduced onto previously uninfected premises via personnel or equipment contaminated with infectious feaces.

Tukey, poults and pheasants of 3-4 week old are resistant to infection because of age-related resistance.

Clinical findings:

It affects pheasants 3-8mo old.

Onset is acute with dyspnoea,asphyxiation and sudden death occurring as a result of pulmonary congestion and edema.

Mortality is commonly 2-3% but can reach 15%.

Gross Lesions:

Spleen is enlarged, friable and mottled white.

Histopathologic lesions:

Basophilic intranuclear inclusions can be seen in spleen,where lymphoreticular hyperplasia and lymphoid necrosis are noted.

Diagnosis:

By clinical signs and gross lesions. Other tests include ELISA and Agar gel immunodiffusion.

Treatment and control:

Live vaccines, regular disease monitoring and careful integration of haemorrhagic enteritis and marble spleen disease vaccines into flock vaccination protocols is encouraged.

NATIONAL EGG CO-ORDINATION COMMITTEE

DAILY / MONTHLY EGG PRICES DECLARED BY NECC AND PREVAILING PRICES AT VARIOUS PRODUCTION CENTRES (PC) AND CONSUMPTION CENTERS (CC) NOVEMBER 2020

Name Of Zone / Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	Average
NECC Prices																															
Ahmedabad	525	525	525	525	498	498	498	500	500	505	510	515	518	520	520	520	520	523	523	523	523	523	495	480	470	473	475	477	479	-	506.41
Ajmer	490	490	480	460	450	445	447	460	485	495	495	495	485	485	490	498	490	485	485	472	472	465	441	430	445	448	456	459	-	-	471.36
Barwala	493	493	480	462	454	441	441	451	471	481	481	481	481	481	483	485	485	485	485	485	485	455	441	441	443	448	451	454	457	-	468.07
Bengaluru (CC)	510	510	510	510	485	475	450	450	455	465	475	485	490	490	490	490	490	490	490	490	490	490	490	460	445	445	445	450	450	-	478.1
Brahmapur (OD)	495	495	460	460	430	430	430	430	436	448	459	460	463	463	463	463	463	463	463	463	463	463	448	448	443	443	445	448	448	-	454.69
Chennai (CC)	520	520	500	500	500	475	475	450	450	465	465	480	500	500	500	500	500	500	500	500	500	500	500	470	470	470	445	445	445	-	484.31
Chittoor	513	513	493	493	493	468	468	443	443	458	458	473	493	493	493	493	493	493	493	493	493	493	493	463	463	463	438	438	438	-	477.31
Delhi (CC)	525	525	511	505	501	485	485	475	490	490	490	490	490	490	495	501	501	501	501	501	501	501	481	461	461	475	475	475	480	-	491.79
E.Godavari	505	505	498	498	475	440	440	440	445	455	465	468	471	471	471	471	471	471	471	471	471	471	445	445	438	438	441	444	444	-	463.41
Hyderabad	470	470	470	450	450	430	430	433	441	451	461	466	469	469	469	469	472	474	474	474	474	474	445	420	420	420	425	428	-	-	453.5
Ludhiana	502	497	493	486	481	462	453	453	457	472	482	482	482	482	482	487	488	488	488	482	476	470	464	456	452	448	448	448	452	-	472.86
Mumbai (CC)	530	530	520	520	501	501	480	480	480	490	501	511	516	519	519	519	519	522	524	524	524	524	524	495	470	470	470	475	-	-	505.64
Muzaffurpur (CC)	548	538	538	529	510	505	490	490	500	524	524	529	524	524	514	514	514	514	514	514	514	514	505	505	486	500	500	500	500	-	513.14
Mysuru	510	510	510	510	485	475	450	450	457	467	477	485	490	490	490	490	490	490	490	490	490	490	490	460	445	445	445	450	450	-	478.31
Nagpur	500	500	490	490	480	460	450	450	450	470	500	505	505	505	505	505	505	505	505	490	480	480	445	440	440	440	445	445	-	-	478.04
Namakkal	475	475	475	475	450	450	430	430	440	440	440	470	470	475	475	475	475	475	475	475	475	475	450	450	450	425	425	425	425	-	456.72
Patna	538	529	529	514	500	490	486	486	490	524	524	514	514	514	514	514	514	514	514	514	514	514	500	505	486	500	500	500	500	-	508.79
Pune	520	515	510	510	500	500	480	485	490	495	501	511	516	520	520	520	520	522	525	525	510	500	500	490	470	470	472	475	480	-	501.79
Ranchi (CC)	548	543	543	538	529	519	500	500	510	524	524	524	524	524	524	524	524	524	524	524	524	519	510	510	500	505	510	510	510	-	520.41
Vijayawada	505	505	498	498	475	440	440	440	445	455	465	468	471	471	471	471	471	471	471	471	471	471	445	445	438	438	441	444	444	-	463.41
Vizag	490	490	480	480	460	440	440	445	450	460	470	470	470	470	470	470	470	470	470	470	470	470	435	435	435	435	435	435	435	-	459.31
W.Godavari	505	505	498	498	475	440	440	440	445	455	465	468	471	471	471	471	471	471	471	471	471	471	445	445	438	438	441	444	444	-	463.41
Warangal	472	472	472	452	452	432	432	435	443	453	463	468	471	471	471	471	474	476	476	476	476	476	447	422	422	422	427	430	433	-	454.72
Prevailing Prices																															
Allahabad (CC)	514	514	505	495	490	486	476	476	500	524	529	529	524	524	505	505	514	514	510	505	500	500	486	486	486	495	500	500	500	-	503.17
Bhopal	515	515	498	490	480	480	460	460	470	490	495	510	510	510	510	510	510	510	490	490	480	470	470	470	448	450	455	460	-	-	485.93
Hospet	475	475	475	475	450	440	415	415	420	430	440	450	455	455	455	455	455	455	455	455	455	455	455	425	410	410	410	415	415	-	443.1
Indore (CC)	505	505	505	480	470	460	470	480	500	500	510	500	500	500	500	505	505	505	490	480	480	480	465	465	450	450	460	465	-	-	485.18
Jabalpur	500	500	493	483	470	470	460	465	470	485	490	500	500	500	500	500	500	500	500	495	490	473	465	450	450	455	455	455	-	-	481.21
Kanpur (CC)	529	529	514	505	490	467	467	467	490	514	514	514	514	514	514	514	514	514	514	514	500	500	481	481	467	467	467	467	467	-	496.86
Kolkata (WB)	548	548	533	518	502	502	492	492	500	520	530	530	530	530	530	530	520	520	510	510	512	512	512	502	502	502	502	502	502	-	515.28
Luknow (CC)	570	570	570	570	560	550	550	540	530	530	550	550	550	550	550	550	550	550	550	550	540	530	530	530	530	520	520	520	510	-	543.79
Raipur	500	500	500	480	455	455	440	440	453	468	497	500	500	500	500	500	500	500	500	500	475	455	455	445	445	445	445	445	-	-	474.93
Surat	535	536	536	536	516	510	510	512	518	517	522	527	530	532	532	532	532	535	535	535	535	535	520	490	475	478	480	482	484	-	517.83
Varanasi (CC)	550	550	540	540	530	523	500	510	520	530	540	540	540	540	540	540	540	540	540	540	530	530	517	517	507	510	510	510	510	-	528.76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PRESS RELEASE

Raw material risk management for mycotoxins The ever-more-challenge for Indian poultry producer

Dr Susim Mukul Ray, Head - Technical & Promotion (PBU), Zydus AHL

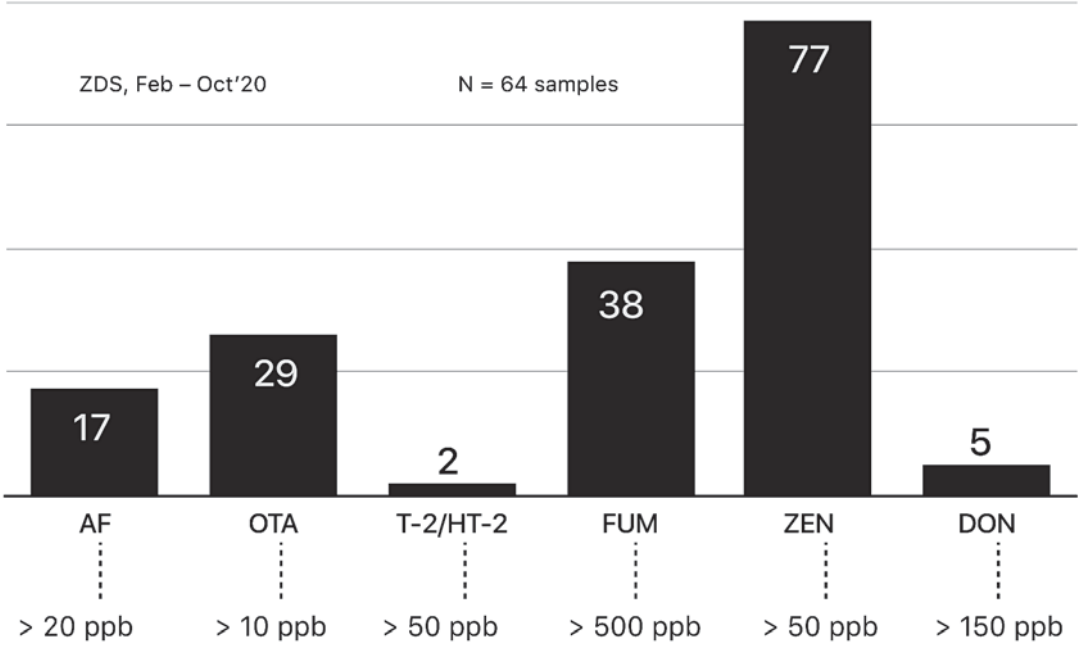
Dr Susim Mukul Ray

Amid concerns over a slowdown in the Indian economy during mid-Q3'19, worrying reports of economic distress from the poultry farm sector began hitting the headlines. During the period, the economic crisis in the sector owes largely to the hike of prices of raw materials viz., maize and rice bran, making upto 70-80% of feed formulation. The resulting hike in prices of poultry feed is speculated to be the highest in last 20 years. Short rainfall in preceding year affecting agriculture production is thought to be pivotal in precipitating the crisis. The odyssey of economic crisis was worsened by COVID 19 pandemic, until Q3'20, the reeling sector began spurting back to life with drastic reduction of maize and rice bran price- heaved a sigh of relief !

The economic growth in sector usurped the crisis apparently, while looming challenges of mycotoxicosis inflicted serious headaches to the farming community. Raw materials, especially the new strain of maize, registered higher mycotoxin levels as compared to previous year leading to various unprecedented complications in field- poultry productivity.

At Zydus AHL, our team of experts continuously strive to identify these stressors, analyse, and find right solution for the farming community. In next section, the retrospective analysis of mycotoxin levels in raw materials and finished feed studied by our laboratory is presented.

Fig 1. Contamination % of raw materials with various levels of mycotoxins in feed amples



AF = Aflatoxin, OTA = Ochratoxin A, T-2/HT-2 = Type A Trichothecenes, FUM = Fumonisin, ZEN = Zearalenone, DON = Dioxynivalenol

Fig 2a. ZEN level in feed samples

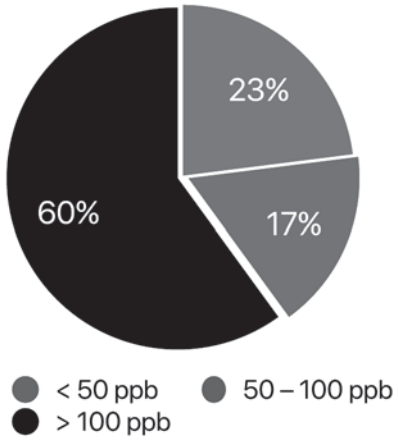
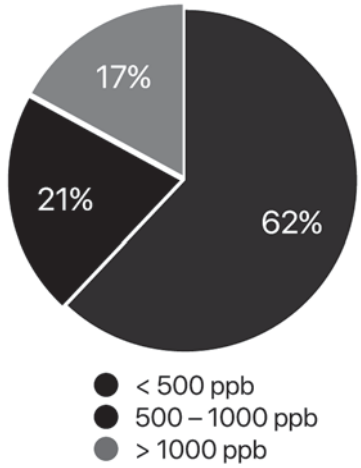
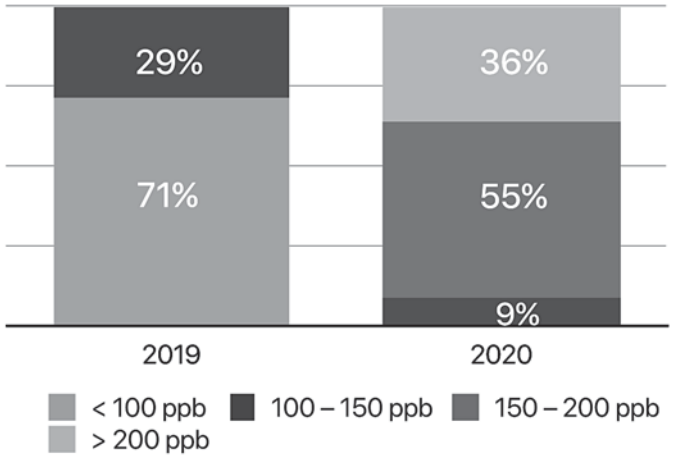


Fig 2b. FUM level in feed samples



Total 64 feed samples were analysed during Feb - Oct'20 and showed very high levels of ZEN and FUM (Fig 1). 77% feed samples had registered ZEN level > 50 ppb, while 38% feed samples had FUM level > 500 ppb (Fig 2a & 2b).

Fig 3. Comparison of ZEN level in maize (2019 vs. 2020)



N=50 maize samples in 2019 and 2020

A comparison of 50 maize samples for ZEN level in 2019 and 2020, respectively clearly highlights significantly higher contamination in 2020. In 2019, only 29% maize samples had registered ZEN level > 100 ppb, while all maize samples (100%) had ZEN level > 100 ppb in 2020 (Fig 3). Such higher ZEN level in association with FUM had visible implications on poultry productivity, as will be discussed in forthcoming sections.

Besides higher ZEN level in maize/finished feed, the co-occurrence of more than one mycotoxin - multiple mycotoxicosis had far reaching detrimental effects primarily because of the synergism existing across different mycotoxins. It means that mycotoxins exert ill-effects at significantly lower concentration than alone resulting in lowering of their threshold limits in feed e.g. ZEN level upto 500 ppb can be tolerated by broiler breeder hens while chronic consumption of AF + ZEN or ZEN + DON at 20-&-50 ppb or 50-&-150 ppb, respectively may have deleterious effect on hatchability and egg shell quality.

% Co-contamination of different mycotoxins in feed samples > 20 ppb (Fig 4a) and >50 ppb (Fig 4b)

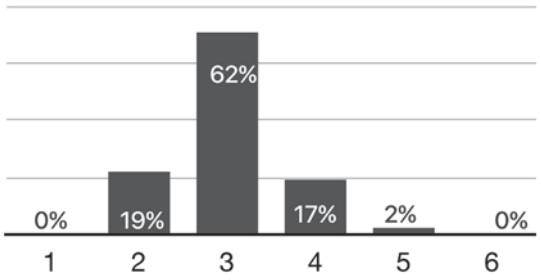


Fig 4a

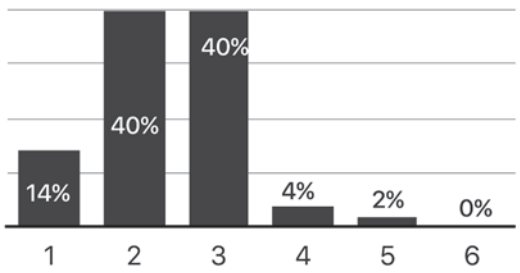


Fig 4b

N=64 samples; ZDS, Feb - Oct'20

In 2020, our laboratory data on mycotoxin analysis showed that 46% and 81% feed samples were co-contaminated with 3 or more mycotoxins >20 ppb and >50 ppb, respectively (Fig 4a & 4b).

Zearalenone (ZEN), one of the most prevalent estrogenic mycotoxins, is mainly produced by Fusarium fungi and has been proven to affect the reproductive capacity of poultry. Exposure of poultry to ZEN is a global public health concern because of its toxicity and wide distribution in poultry feeds, carry over effect in egg and meat, and being stable/ unaffected by feed/food processing conditions (150 °C for 44 h). Biotransformation of ZEN carried out by poultry liver leads to the formation of two metabolites: a-zearalenol and 13-zearalenol. All ZEN forms are estrogenic, with the a-zearalenol being the highest. It has synergistic effect with Aflatoxin (AF) and Dioxynivalenol (DON), while additive effects with Fumonisin (FUM).

In 2020, higher levels of ZEN along with other mycotoxins (AF, FUM, DON, and OTA) in maize/ finished feed samples were correlated with poultry production trend and following were our observations.

Egg laying hens (Breeder/Commercial layer)	Commercial broiler
Reduced egg egg production (3 - 8%)	Inflammation of bursa — bursitis (Feb – Jun'20) — Fig 6a & 6b
Fluctuation in egg production	Increased incidence of Gumboro disease (July – Oct'20)
Poor eggshell strength	
Watery albumin	
Reduced egg size — Fig 5	

Fig 5. Reduced egg size (right egg); normal size egg (left); Zearalenone and Fumonisin level in layer feed was 220 ppb and 540 ppb, respectively; condition reversed with broad spectrum toxin binder (AntaFerm® MT80 @ 1 kg per MT feed)

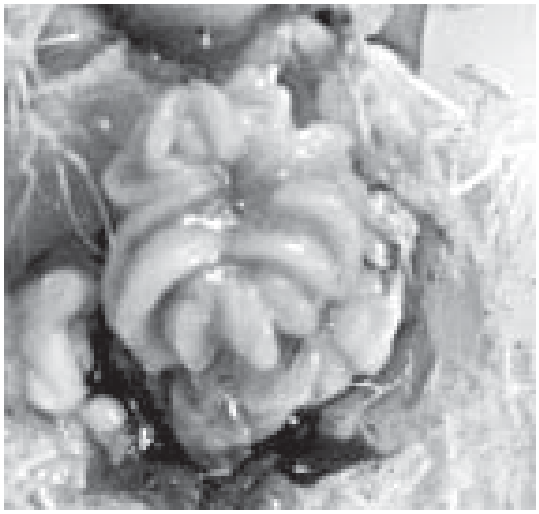


Fig 6a & 6b. Inflammation of bursa in commercial broiler flock at day 26. Flock registered mean 4500 (IDEXX) IBD titer suggesting seroconversion against vaccination only. The Zearalenone and Fumonisin level in starter feed was 260 ppb and 640 ppb, respectively.

Controlling multiple mycotoxicosis, especially ZEN, FUM and DON, is challenging for poultry producer as they are pre-harvest mycotoxins and are extremely stable in feed processing conditions. Moreover, they are produced by same genera of fungi viz, Fusarium spp., and presence of one mycotoxin e.g. ZEN potentially increase the contamination risk of other mycotoxins e.g. FUM and DON. Therefore, two way approach in checking the menace of multiple mycotoxicosis is recommended.

- First, eliminating the growth of Fusarium fungi by mould inhibitors. Combination of buffered organic acids (SCFAs) and formaldehyde (ZanitizerTM) is very effective and ensures feed sanitisation before consumption by poultry.
- Secondly, pre-formed mycotoxins should be adsorbed completely by combination of inorganic and organic adsorbents before they are absorbed by chicken GI tract. Mycotoxin adsorbents (Bentonite, (3-glucans, MOS, Diatomaceous earth, etc.) ensure that the mycotoxins are not bioavailable in systemic circulation after consumption by chicken. In this context, the right choice of mycotoxin adsorbents is critical. Mycotoxin like ZEN is non-polar in nature and require organic adsorbent (e.g. (3-glucans) for effective binding in chicken GI tract, while AF (polar) requires inorganic adsorbent (e.g. Bentonite). Furthermore, the prevalence of mycotoxins vary widely, spatially and temporally (as we have shown for ZEN level in 2019 vs 2020), and therefore, ideal mycotoxin binders for poultry use should incorporate adsorbents dedicated for both polar and non-polar mycotoxins for optimum protection.

In conclusion, multiple mycotoxicosis is a serious threat to poultry producers. These mycotoxins exert synergistic and additive action in combination and, in most part, work in significantly lower concentration in combination capable of causing deleterious effect on poultry production and significant financial losses.

Srinivasa Hy-Line

MANAGEMENT OF POULTRY DURING WINTER

In India, the winter season follows the rainy season and can be marked with cold weather. Winter season in India lasts between November to February. Northern India experiences the most severe cold season, where environment temperatures can drop below 15°C with nighttime temperatures as low as 5°C. Southern India usually experiences milder winter weather and low temperatures are not a major concern. Winter season brings unique challenges for brooding chicks. Cooler environmental temperatures can affect nutrition programs because of the bird's higher energy requirement to maintain body temperature. The effects of decreasing photoperiod and light intensity during the winter can affect sexual maturity, resulting in delays in egg production. Poor air quality can occur as farmers close curtains to maintain house temperature. Cool air slows down the drying of manure, leading to increased ammonia levels within the shed and can increase the fly menace. Poor air quality and cooler temperatures can increase the disease threat during the winter season.

Table 1. Weather Conditions in Different Regions of India during Winter

December Month Average	North India (New Delhi)	Central India (Mumbai)	South India (Hyderabad)
Maximum temperature °C	23°C	32°C	28°C
Minimum temperature °C	9°C	19°C	15°C
Average humidity %	62%	58%	57%
Average hours of sunshine per day	7 hours	8 hours	8.5 hours
Management attention	High priority	Medium priority	Low priority

Source: https://www.currentresults.com/Weather/India/temperature-december.php

The following intervention strategies should be considered during winter season:

Brooding and Growing Management:

1. Chick brooding requires special attention during winter. Brooding shed arrangements should be ready before 48 hours of chick placement. This is important because it will take a longer time to preheat the chick's environment during the winter season. Ensure the shed and equipment is heated to 35°C environmental temperature. Relative humidity should be maintained between 40–60%.
2. Be aware of low nighttime temperatures during the winter season. Cold stress usually occurs during the night and early morning. Maintaining proper chick brooding temperatures throughout the night can be challenging in the winter season. This is especially difficult for farmers using charcoal heaters or other sources of heat without thermostatic control. Thermostatic control of brooding shed temperatures is highly recommended to avoid cold stress during the night time. Low nighttime temperatures can chill chicks, which can impair their growth and organ development. Cold stressed chicks are more susceptible to infectious diseases. Use a thermometer that is capable of recording nighttime temperatures in the brooding shed.

Figure 1. Brooding management.

3. Frequently observe the activity of chicks and adjust temperatures to the comfort of the chicks. Chicks should be distributed evenly inside the cage. Under cold stress the chicks are huddled in groups, not eating and drinking and with less activity. For more information on W-80 brooding management, refer to the "Growing Management of Commercial Pullets" technical update at www.hyline.com.

4. An infant ear thermometer can be used to measure the vent temperature of chicks. This gives a good indication of the comfort of the chicks and correlates well with the chick's core body temperature. The normal vent temperature in chicks should be 39.4–40.5°C.

Figure 2. Monitoring chick temperature using an infant ear thermometer.

5. During the brooding period, place starter crumble feed on the cage paper for first 3 days to encourage feed consumption. Cage paper blocks cold drafts of air. For infrared beak treated (IRBT) chicks, place starter crumble on the cage paper for first 7 days. Checking chicks for the presence of feed in the crop helps understand feed consumption. The presence of feed in the crop is a good indication of a proper chick start (see Figure 3).

CROP FILL – ARE THE CHICKS EATING?

Hours after chick placement	Chicks with feed in crop
6	75%
12	85%
24	100%

Chick with starter feed in crop | *Chick without starter feed in crop*

Figure 3. Desired crop fill percentages.

6. During peak winter where environment temperature drops below 10°C, the drinking water temperature drops close to freezing. Drinking water temperature has a direct effect on the bird's feed and water consumption and slows body weight gains in growing chicks. Poor water consumption can also increase mortality related to dehydration and gout. The ideal water temperature to maintain good feed intake is 18–21°C.

7. The shortest daylength of the year falls on December 21st. The shortest daylength in India ranges from 10–11 hours, with North India having the shortest among all regions (see Table 1). The ideal hours of light during the rearing period for the W-80 is 11–12 hours. Rearing lighting hours need to be maintained at recommended levels for pullets in order to achieve ideal body weight gain and sexual maturity. This will be done by following the Hy-Line International / Srinivasa Farms Lighting Program Generator. This is an Excel tool which creates lighting programs appropriate for the farm location and shed style (open or EC shed). These customized lighting programs can be provided to commercial customers with their chick placements. For further information, see https://www.hyline.com/ViewFile?id=d14081e1-8af8-49f1-a752-71720d4b5680 or contact Srinivasa Farms' technical service team or Hy-Line India's technical service team.

8. Provide adequate ventilation in brooding house for 24 hours in the winter. Do not close the brooding area too tightly while maintaining brooding temperatures during the winter. Always provide a continuous supply of fresh air to the birds by maintaining some opening of the curtains. The minimum ventilation rate during the winter must be sufficient to remove moisture and prevent the build-up of noxious gases in the brooding area. Ammonia greater than 25 ppm is harmful to chicks and can promote respiratory disease outbreaks. Coal heaters are commonly used in India as a heat source in brooding sheds and they produce large amounts of carbon dioxide (CO_2), carbon monoxide (CO), and other undesirable gases inside the house. It is recommended to have a minimum opening (one foot) at the top level of the curtains to provide minimum ventilation even during nighttime. During the middle of the day, the side curtains can be adjusted according to temperature and chick comfort.

9. Allowable levels of gases at the bird level in the shed are: ammonia (NH3) <25 ppm; carbon dioxide (CO_2) <5000 ppm; carbon monoxide (CO) <50 ppm.

10. Bird transfers from brooding to rearing sheds and rearing laying sheds should be completed no later than 7 weeks and 16 weeks, respectively. Timely transfers give the birds enough space to continue proper growth and development and enough time to adjust to the new environment. During the peak winter season, schedule transfers to occur during mid-day when the temperature is more comfortable for the birds.

Layer Management:

1. Feed intake is generally higher in winter months as a result of increased demand for energy to maintain body temperature. Protein and amino acids should be balanced based on the actual flock feed consumption. Overconsumption of energy, protein and amino acids beyond the recommended level can lead to deposition of extra fat which predisposes bird to fatty liver / hemorrhagic syndrome (FLHS), as well as increases egg weight. Energy requirements tend to be slightly higher during winter, so it is important not to decrease the energy levels at the same proportion of the feed intake increase. See the W-80 flock book provided by Srinivasa Farms for the nutritional recommendations of the W-80.
2. Increased feed intakes during winter could lead to increased egg weights. Overconsumption of energy, methionine + cystine, other digestible amino acids, linoleic acid, and total fat can directly increase egg size. Egg weights should be monitored every week during winter and appropriate adjustments to the diet made to control egg weight.
3. Stone grit management may help in controlling feed intake and maintains eggshell quality if egg weights increase. Vitamin D supplementation during winter may be needed due to poor brightness of sunlight. Follow the W-80 recommended levels of vitamin D3 (3,300,000 IU per ton of feed – Rearing and laying phases).
4. Decreasing the feed particle size of less than 700 microns and including fibrous ingredients to the feed formulation is the best way to control feed intake.
5. In addition to shorter daylength, foggy conditions with lower light intensity are common in winter. Average hours of sunshine are less during winter months (November to February). North India records the lowest hours of sunshine compared to other regions (see Table 1). Increased use of curtains during the winter to protect birds from cold stress blocks sunlight and further reduces the light intensity inside the shed. With lower brightness inside the shed, it is good practice to use the house lights to maintain recommended light intensity (30 lux) inside the layer shed.
6. Keep light intensity optimum by cleaning dirty bulbs and replacing faulty bulbs. This work should be done before the arrival of winter.
7. Adult laying birds are also susceptible to cold stress. In open-sided laying houses, it is recommended to use side curtains to protect birds from direct exposure to cold stress. The side curtains are managed in such a way to protect birds from cold stress as well as to provide minimum ventilation to remove excess ammonia buildup. Curtains should be allowed minimum opening (one feet) at the top level of the shed even during nighttime, and during the middle of the day, partial opening at the side can be practiced based on bird comfort (see Figure 4).

Figure 4. Lowering the curtain at the top creates better ventilation.

8. Decreasing day length during the winter may delay pullets from coming into egg production. Timely shifting of the flock to the laying shed and on-time light stimulation at the correct body weight (1100g with 85% uniformity) prevents a delayed start of egg production. A timely transition from the developer or pre-lay diets to the peaking diet ensures that egg production begins properly, avoiding egg production delays.
9. Cold air slows down the drying of manure and removal of moisture from the shed. This can cause excess ammonia gas build-up in laying sheds in the winter. High ammonia is also caused by nipple leakage and lack of ventilation due to closed side curtains. This problem will be more pronounced in farms where the height of the manure is close to bird level. Remove manure and replace faulty nipples prior onset of winter to avoid conditions of high ammonia.

10. Cold weather and reduced air quality favors multiplication of pathogens, especially respiratory pathogens. Incidences of avian influenza, Newcastle disease, Gumboro (IBD), fowl pox, colibacillosis (E. coli), infectious coryza, gangrenous dermatitis, salmonellosis, and coccidiosis are more common in winter. Following good winter management with good biosecurity and timely vaccinations to control disease outbreaks.
11. Vaccinations should be carried out in the daytime during peak winter (December and January) when the temperature is ideal. In case of water vaccination, water holding time before vaccination should be increased from 30 minutes to 1 hour since water consumption is normally lower during winter. Water volume used for water vaccination should be matched with actual water consumption.

Management Chart:

Management Practices	North India Farms	Central India Farms	South India Farms
Brooding management	High attention	Medium attention	Medium attention
Water management	High attention	Medium attention	Low attention
Feed management	High attention	Medium attention	Medium attention
Lighting program	High attention	Medium attention	Low attention
Ventilation	High attention	Medium attention	Low attention
Manure management	High attention	Medium attention	Medium attention
Disease control	High attention	High attention	High attention
Bird transfer	High attention	Medium attention	Medium attention

Srinivasa | Hy-Line

TU WINTER MGMT INDIA 11-2020

Nearly half a million poultry deaths: there are 3 avian influenza outbreaks in Victoria. Should we be worried?

As we navigate a global human pandemic, avian influenza (or "bird flu") has been detected in domestic poultry across Victoria.

When scientists discuss avian influenza, we're usually referring to the diverse subtypes of influenza that primarily infect birds. Avian influenza viruses are commonly found in healthy wild birds and can also cause illness and death among domestic poultry including chickens, turkeys and ducks.

Humans can contract it if they come into close contact with infected birds (not from eating cooked chicken or eggs). But these viruses don't easily infect us and their health risk is considered low.

Between 2003-2019, there have been about 2,500 human cases of avian influenza globally (mainly caused by the influenza subtypesH7N9 and H5N1).

There's also no evidence of people becoming infected as a result of the current outbreaks in Victoria. Nonetheless, avian influenza viruses can mutate, so we must carefully monitor and deal with them as they arise.

How we classify avian influenza

These viruses are classified in two ways. The first is based on the HA-NA subtype system. On the surface of the virus are two proteins: haemagglutinin (HA) and neuraminidase (NA). Of these, there are 16 and 9 types respectively.

So when we talk about the subtype H5N1, for example, we're referring to type 5 of the HA and type 1 of the NA. Due to their mix-and-match nature, there are 144 potential HA-NA subtype combinations. The vast majority of these never cause disease in birds.

Avian influenza viruses are also classified by how "pathogenic" they are, which refers to their ability to cause disease in domestic poultry. Low pathogenic viruses are common in wild birds and

may cause limited disease in poultry, but highly pathogenic viruses cause high mortality in poultry.

Occasionally, when an H5 or H7 low pathogenic avian influenza virus crosses from wild birds to poultry, changes in the virus genome can occur, transforming it into a highly pathogenic virus.

Avian influenza outbreaks in Victoria

In Victoria, there have been three outbreaks of avian influenza since July this year: two low pathogenic avian influenza viruses, H5N2 and H7N6, in domestic turkeys and emus, respectively, as well as a high pathogenic H7N7 virus in chickens.

The simultaneous detection of different virus subtypes in chickens, emus and turkeys is unusual. In the past, outbreaks in domestic birds have mostly been caused by a single subtype. This highlights the importance of stringent biosecurity practices, to prevent the introduction of avian influenza into farmed poultry.

Victoria's current outbreaks are causing substantial economic loss and are considered emergency animal diseases. They have resulted in:

the deaths of about 450,000 domestic birds across six farms, of which the vast majority are egg-laying chickens

a potential loss of export markets for poultry products

significant response costs and loss of income for affected producers, requiring permits to move eggs, equipment and birds from affected areas.

The good news is Australia has successfully eradicated high pathogenic avian influenza viruses in the past. We will almost certainly eradicate these too.

Agriculture Victoria, the lead agency for emergency animal diseases in the state, is responding to the outbreaks in a number of ways.

Firstly, a housing order requires all bird owners in the affected areas to keep their birds inside. This measure, along with other movement controls, helps limit spread to other farms.

Second, infected birds on the farms are destroyed, with the farms thoroughly decontaminated. These procedures are key to preventing the continued spread of avian influenza.

How we're tracking the spread of the viruses

Outbreaks of avian influenza in Australian poultry are infrequent. The last outbreak of high pathogenic avian influenza in Victoria's poultry (before this year) was in 1992. Low pathogenic avian influenza, however, is detected in our wild birds regularly.

Past testing has found different groups of wild birds can have infection rates ranging between 0.1-40%. The variation depends on which species make up the group, the group's predominant location and also what season it is. The most common virus subtypes found in wild birds are H1, H3 and H6.

Data used to understand and monitor avian influenza in the wild is generated by the National Avian Influenza Wild Bird Surveillance program, which screens samples directly from captured birds, or indirectly through their faeces.

Not an 'imported' virus

Unlike contact tracing with people, birds can't tell you who they have been socialising with. That's why genomic sequencing is crucial in tracking, tracing and monitoring avian influenza viruses.

Each virus has a unique genomic sequence, like a genetic fingerprint. Using genetic analysis, the different genomes can be compared. This offers insight into how closely related certain viruses are and how wild birds may be spreading them across the country.

This method helped us to discover Victoria currently has three distinct outbreaks – and to connect the farms within each outbreak.

Also, a critical component of our response is the collection of virus genomes already available to us from past surveillance efforts. These data have revealed the viruses currently in Victoria are not imported from Asia, or elsewhere.

Rather, they're similar to low pathogenic avian influenza viruses currently circulating in wild Australian waterbirds, as well as viruses that have caused past outbreaks in poultry.

Courtesy: theconversation.com

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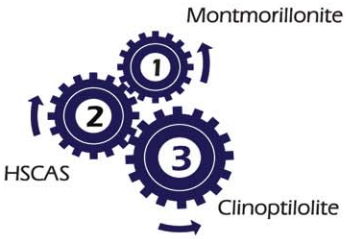

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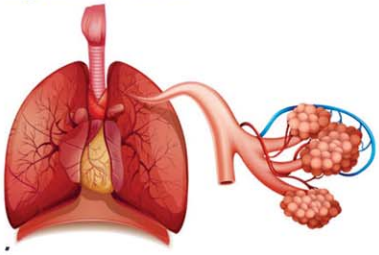

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Why Mycotoxins Matter in Broiler Production

By Lorran **BaeumleGabardo**

The negative impacts of mycotoxins in poultry can be far-reaching, decreasing gastrointestinal integrity, immunity and performance in broilers and resulting in economic losses.

Given the high rates of mycotoxin in poultry feed, a mycotoxin risk management strategy is needed to protect fattening animals in all phases in order to reduce the challenges in the animal and ensure profitability of production.

Poultry producers frequently ask: What is the real impact of mycotoxins on the broiler's productivity? What sounds like an easy question is, in the case of mycotoxins, unfortunately not so easy to answer.

Three key considerations can help you evaluate the impact of mycotoxins on your broilers' productivity:

1. Contamination levels in the finished feed
2. Direct and indirect effects of mycotoxins in the animal
3. Performance parameters

Contamination in the broiler's feed

Mycotoxins are frequently found in **poultry feed**. According to the BIOMIN World Mycotoxin Survey, more than 8,000 samples of finished poultry feed tested positive for mycotoxins in the last five years (2016 – 2020). A closer analysis of this data shows that 92% of these samples are contaminated with more than one mycotoxin, it can be founded as much as 40 to 50 mycotoxins in the same sample. Therefore, it is of utmost importance to interpret the whole picture and not just look at the effects of a single mycotoxin if you need to evaluate the risk in a broiler production (Figure 1).

Testing the finished feed and/or their ingredients helps to evaluate the risk for the animal's health. The frequent presence of mycotoxins triggers the immune system of the animals, consequently reducing the performance of the flocks, what can be translated as a loss of profit for the poultry producers.

Mycotoxins in the broiler itself

Visible clinical signs in poultry such as **fatty liver**, beak erosions or cystic oviduct are not always clearly identifiable in broilers under field conditions, for two main reasons: First, the short life cycle of broilers means that problems may lurk within the animal but not have time to manifest outwardly. Second, the combined effects of more than one mycotoxin make the diagnosis of a mycotoxicosis more difficult. However, the major impacts of mycotoxins on immunity, inflammation, oxidation and gut health in poultry husbandry has been clearly proven in scientific trials.

Special attention should be paid to:

Mycotoxins' impact on gut health: It is well proven that mycotoxins, especially deoxynivalenol (DON) and fumonisins (FUM), affect several aspects of intestinal integrity. A meta-analysis (Grenier and Applegate, 2013) showed a clear influence of DON and FUM:

- On the morphological structure of intestinal epithelium by destroying the villus;
- By reducing the tight-junctions between the intestinal cells, opening up the intestinal barrier to the bloodstream;
- By modulating the local immune response and the microbiota profile.

Collectively, these effects can compromise several intestinal functions, mainly reducing the absorption surface for nutrients and consequently harming digestion. Moreover, by increasing the permeability of the intestinal barrier, the entrance for pathogens, anti-nutritional factors and other toxins into circulation is facilitated. (Figure 2).

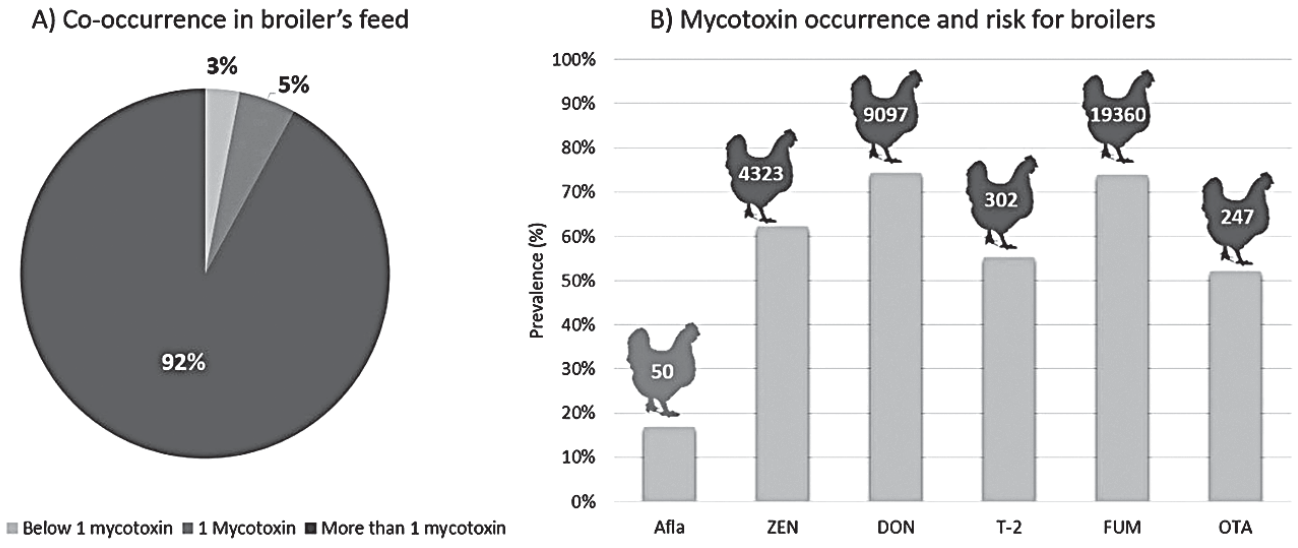


Figure 1. Mycotoxin presence in poultry finished feed (2016 -2020). A) Co-occurrence of mycotoxins. B) Mycotoxin occurrence and risk for broiler: % of positive samples for the respective mycotoxin are shown in the bars while maximum contamination found is expressed inside the broiler image (ppb). The colours express the risk level for broilers, red being high risk and orange indicating moderate risk. (Source: BIOMIN World Mycotoxin Survey)

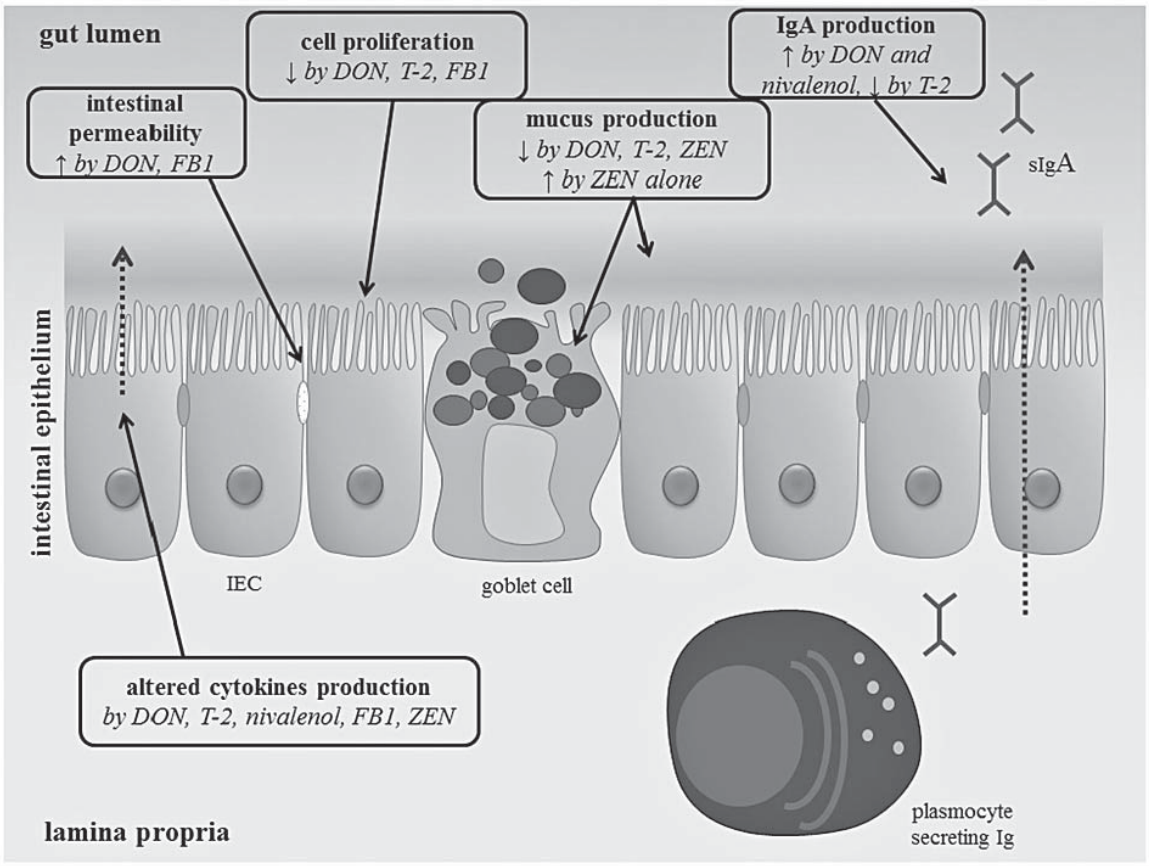


Figure 2. The effect of DON, nivalenol, fumonisin B1 (FB1), T-2 toxin and zearalenone (ZEN) on the intestinal epithelium. They alter the different intestinal defense mechanisms including epithelial integrity, cell proliferation, mucus layer, immunoglobulins (Ig), and cytokine production. (Source: Antonissen et al., 2014)

Mycotoxins as predisposing/trigger factors for health issues: Antonissen et al (2014) evidenced that low to moderate doses of different *Fusarium* mycotoxins (DON, nivalenol, fumonisins, t-2 toxin and zearalenone) are predisposing factors to several important diseases in poultry production such as coccidiosis, salmonellosis, necrotic enteritis and colibacillosis. Combinations of DON, fumonisins and zearalenonewere identified as reducing the Eimeria-induced immune response and the efficacy of the anti-coccidial treatment. The necrotic enteritis challenge is also increased when fumonisins and DON are present (Antonissen et al., 2012 and Antonissen et al., 2015). Evidence of a higher susceptibility to *Salmonella typhimurium* was also reported in the presence of DON and T2, suggesting that these compounds may modulate the bacterial metabolism. *E. coli* pathogenesis is influenced by fumonisins, stimulating intestinal colonization and translocation of *E.coli*.

Mycotoxins triggering the immune system and stressing the liver, with a cost on nutrients and energy for growth: It is known that the liver is directly affected by most mycotoxins, causing a loss of function of the hepatocytes. In broilers, a visible fatty degeneration and an increase of approximately 15% in the liver weight were reported. This disturbance reflects an increased cost of nutrients and amino acids, specially methionine which is the first limiting amino acid for broilers. To overcome this challenge a possible action would be to adjust the nutritional levels of the diet to the mycotoxin presence. By adjusting the formulation, you do not overcome the problem. You may alleviate the issue in the short term, however, this strategy results in a higher cost for the diets. Moreover, the dynamic profile of the mycotoxin contamination means it is not feasible long-term. The right approach is to use a mycotoxin deactivator in order to prevent nutritional losses without trying to guess how to manage nutritional changes in the diet.

Mycotoxins inducing vaccination failure due to interference with the immune system: Modulation of immune response is one of the main modes of action of mycotoxins. They silently interfere with other sanitary aspects, such as the **vaccination efficacy**. Mycotoxins act as a contributing factor to reduce the immunity for viral diseases in broilers (Kamalavenkatesh et al., 2005; Hanif and Muhammad, 2015 and Yunus et al., 2012). Ochratoxin, DON, T2, cyclopiazonic acid significantly reduced the antibody titer for Newcastle disease virus (NDV), infectious bronchitis virus (IBV), infectious bursal disease virus (IBDV), and hydropericardium syndrome (HPS). This vaccination failure increases the susceptibility of birds to infectious diseases that could be avoided under normal conditions.

Mycotoxins' ability to facilitate bacterial contamination of carcasses: As previously described, mycotoxins are able to reduce the thickness of the intestinal epithelium resulting in a 'leaky gut'. Once the junctions between intestinal cells are disrupted, an exchange of molecules happens between the intestine and the bloodstream. This condition can influence the carcass contamination in the slaughter house by:

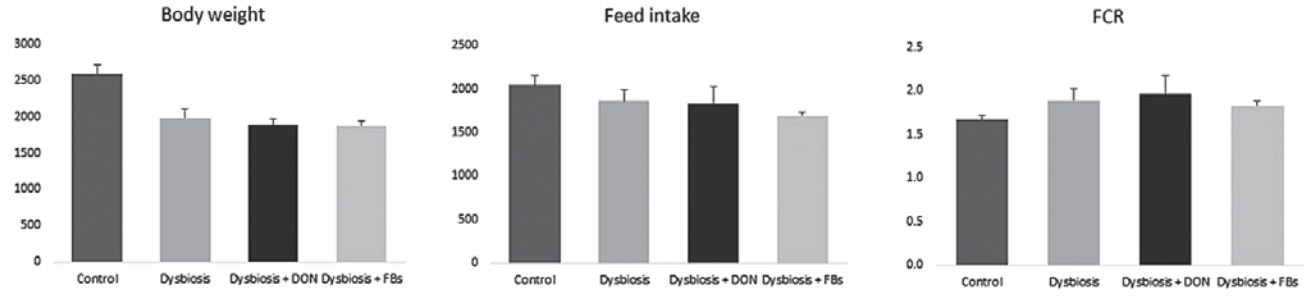


Figure 3. Performance parameters in day 39 of broiler chickens fed either a negative control, dysbiosis control, deoxynivalenol contaminated dysbiosis or a fumonisin contaminated dysbiosis diet. Bars represent means for the 7 replicates (pens) per treatment ± SD. Within the same period, bars with different letters (a-b) differ significantly (P d" 0.05). (Source: Antonissen et al., 2018)

- The excess of proteins in the intestinal lumen supporting the proliferation of pathogenic bacteria such as *E.coli*, *C. perfringens* and *Salmonella* sp,
- The higher amount of water in the gut lumen leading to more leaky excreta and higher contamination in the slaughterhouses,
- The damaged intestinal junctions letting pathogenic bacteria enter into blood circulation.

Therefore, preventing the establishment of a 'leaky gut' can be correlated with the reduction of economic losses and enhancement of food security in the poultry industry.

Impact of mycotoxins on broiler's performance parameters

Performance is still considered one of the most important parameters in evaluating the success of the poultry production. Through this perspective, it is proven that mycotoxins can negatively impact zootechnical parameters in a broiler farm. A data compilation of scientific trials has shown that the presence of mycotoxins in poultry diets significantly reduced ($P < 0.05$) feed intake by 12% and body weight gain by 14%, resulting in an impaired feed conversion ratio of 7% when compared with non-contaminated groups (Andretta et al, 2011). This is mainly attributed to a diminished feed intake resulting in a lower protein deposition efficiency.

Furthermore, preliminary results from a cooperation project with the University of Ghent (Antonissen et al., 2018) show a synergistic negative effect between a dysbiosis challenged diet and additional DON (5 mg/kg) and fumonisins (20 mg/kg) on broilers' performance. Interestingly, this data also evidence that the decreased performance even is more evident in the final phase, reinforcing the impact of mycotoxins in the growth parameters in the last period of broiler's life cycle (Figure 3).

An example of a realistic outcome also comes from recent data of Kolawole et al. (2020). In his long-term evaluation (18 successive trials) of a commercial farm, it was shown that natural contamination with levels below the EU recommendations for mycotoxins has a significant impact of 2.5 points in FCR (Figure 4), confirming the synergistic effects of the mycotoxins (fumonisins, zearalenone, DON and DAS) and the decreased broiler performance in commercial conditions.

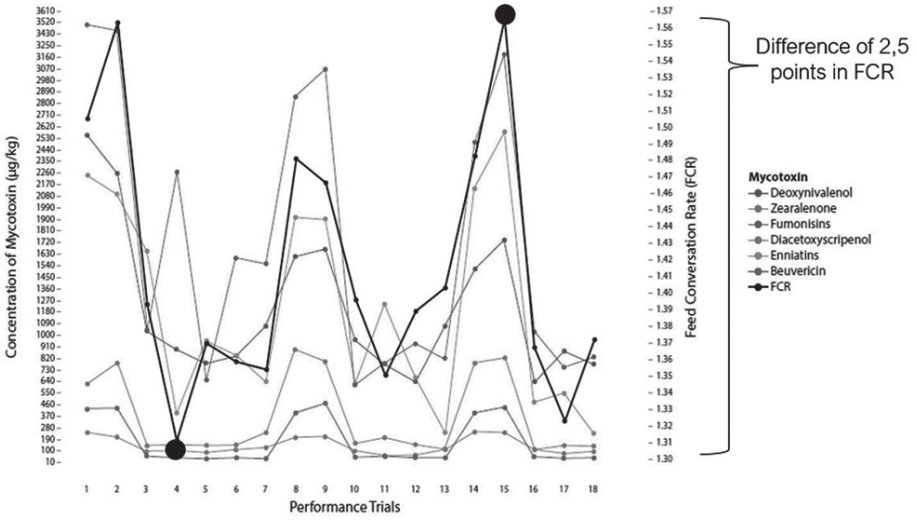


Figure 4. The difference between FCR (black line) in high and low contaminations of mycotoxins (colored lines) in broilers (Adapted from Kolawole et al., 2020).

- The negative impacts of mycotoxins can be far-reaching, decreasing gastrointestinal integrity, immunity and performance in broilers and resulting in economic losses. Due to the frequent occurrence of mycotoxins in poultry feed, a mycotoxin risk management strategy is needed to protect fattening animals in all phases in order to reduce the challenges in the animal and ensure profitability of production.

PRESS RELEASE

Impact of Mycotoxin on gut health in Poultry

Dr B C Dutta, *Poultry Consultant*
https://sites.google.com/view/drbalaichandradutta/homeduttabalaiss@yahoo.co.in

Mycotoxins in feed pose a constant threat to the poultry industry globally. The common feed ingredients forfeed formulation can be contaminated by many mycotoxins.

Mycotoxins are secondary metabolites of common moulds/ fungi produced during their growing period. Some fungi produce mycotoxins on the field, while other produce mycotoxins during the harvest or storage of grains and in finished feed.The most common feed ingredients that can be contaminated by mycotoxins areMaize&its by-products, Wheat &its by-products, Rice &its by-products, Soybean meal, BarleyandGroundnut Cake.

Dr B C Dutta,
Poultry Consultant

There are two Mycotoxin Surveys done by two leading Poultry Feed Additive Manufacturer, Biomin & Trouw,reveals the terrible condition Asian Poultry Industry is facing.

Trouw Nutrition Survey from samples taken between January to August 2020

Table 1. Number of Analysis performed and Percent contamination (Global & Asia)

Mycotoxin/ Parameter	AFB1	DON	FB1	ZEA	OTA	T2	Total
Global No	6232	3066	2741	1790	764	700	15293
Asia No	491	128	194	115	199	109	1236
Asia % Contamination	70	73	90	72	67	2	

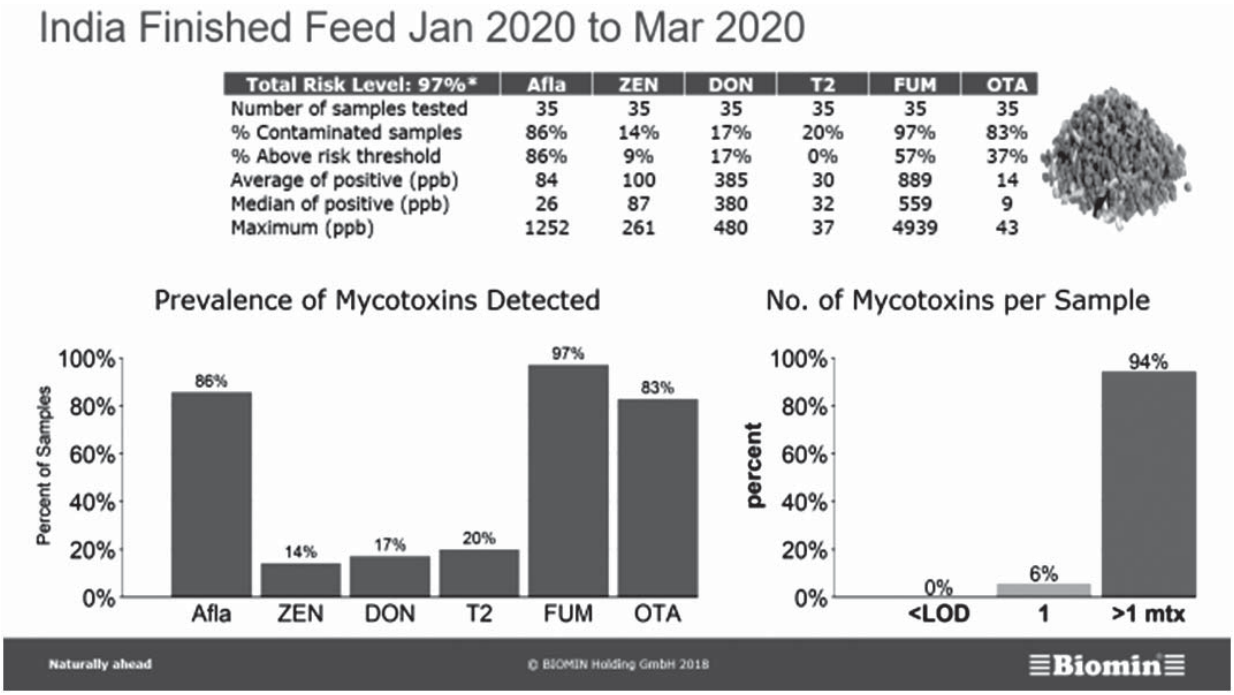
Table 2. Mycotoxin Distribution in Asian countries

Country	AFB1	DON	FB1	ZEA	OTA	T2
India	98 (31)	0 (0)	98 (2101)	67 (30.30)	93 (13.3)	0 (0)
Indonesia	60 (8.9)	65 (213)	85 (2144)	75 (40)	20 (1.0)	1 (0.36)
Bangladesh	56 (4.2)	82 (2214)	67 (3283)	63 (101)	80 (2.7)	0 (0)
China	48 (3.3)	95 (603)	90 (1386)	65 (41)	50 (2.8)	6 (1.80)
Myanmar	47 (5.8)	100 (150)	75 (804)	100 (55)	NA	NA

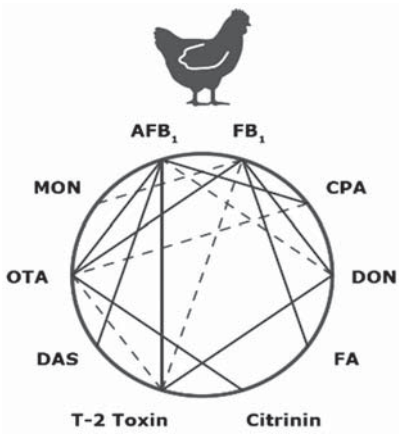
NB: NA = Not Analysed. The Values are % contaminated samples & Concentration in ppb inside brackets

Table 3. Mycotoxin concentrations (ppb) in various commodities in Asia

Commodities	AFB1	DON	FB1	ZEA	OTA	T2
By-products	9.3	1717	613	76	3.4	1.4
Cereal Grains	17	144	1956	29	0.74	0.13
Protein sources	12	472	356	64	13	1.3
Poultry Feed	19	259	2571	40	9.5	0.74
Pig Feed	7.7	125	580	44	1	0
Ruminant Feed	18	NA	1000	NA	4	NA



Poultry have heterogeneous sensitivity to mycotoxins; ducks and turkeys are more sensitive than chickens. Young chickens are more sensitive to the mycotoxins. The effects of mycotoxins in poultry are complex and varies greatly according to their mechanism of toxicity affectingdifferent organs which may lead to death in case of high contamination level. Presence of mycotoxins in combination in feed may have synergistic or additive effects.Even at low levels of mycotoxins in feed, during sensitive period of production cycle or when exposed for longer periods, can impair the immune system leading to the immune-suppression.



Additive (dashed black line) and synergistic (red line) effects of different mycotoxin in poultry (Source: Biomin website)

- Aflatoxins (AFB1), ochratoxin (OTA), trichothecenes, and fumonisins (FB1) are known to induce immune suppressive effects in chickens, enhancing their susceptibility to diseases (Singh et al., 1990, Ghosh et al., 1991). Low level of mycotoxins can have an antimicrobial effect and can cause feed passage (Devegowda and Murthy, 2005).
- The presence of multiple mycotoxins today, particularly in complete feed, is a rule and not an exception.When occur together, many mycotoxins act in a synergistic or additive manner inside animal's body leadingto unexpected and high toxicity.
- Unlike microbes, mycotoxins are heat resistant and survive common feedprocessing operations such as pelleting and extrusion. As a result of all these, the exposure of animals tomycotoxins is unavoidable today and hence prevention strategies should be applied at crop production,feed production and at animal facility levels

Impact of Mycotoxin on Gastrointestinal Tract (GIT) Function

- The two survey reports reveal that almost 98% feed ingredients are contaminated with AFB1

& FB1 and 93% are with OTA. Further, Finished Feed were worse than ingredients with 100% are contaminated with Mycotoxins and 94% are with more than one mycotoxin.

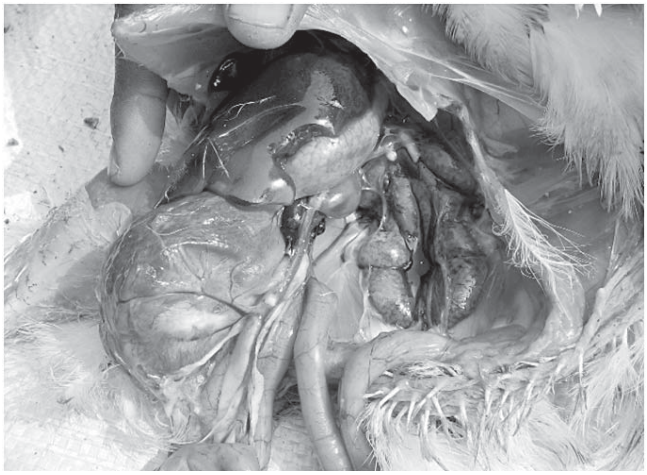
- Gastro Intestine Tract (GIT) is the biggest organ surface exposed to foreign materials including feed, Mycotoxins.
- GIT is responsible for digestion & absorption of all feed materials including water and its ability to function is directly linked to poultry productivity.
- GIT is the biggest Immune organ in chicken's body.
- Any mycotoxins present in feed are delivered straight to the GI tract of the birds; the organ most affected by mycotoxins.

Among the major mycotoxins, DON (deoxynivalenol), ZEN (zearalenone) and FUM (fumonisins) are often overlooked because their impact on poultry health and productivity is not clearly visible. However, many scientific and commercial trials prove that these *Fusarium* mycotoxins are closely related to some important poultry diseases.

Picture 1. Damage of Intestinal Mucosa

Picture 2. Gizzard Erosion

Picture 3. Fatty Liver

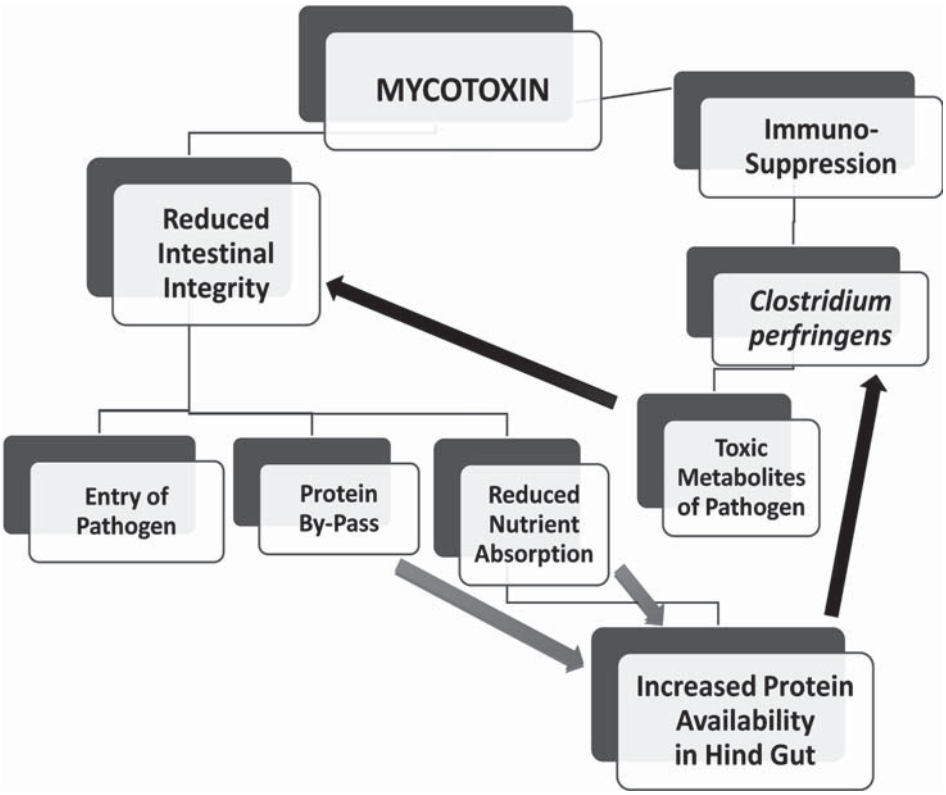


Picture 4. Swollen& damage Kidney

- **AFB1** causes Fatty Liver and Malabsorption due to reduced production of Bile Salts.
- **AFB1** damages the Tight Junction Integrity of Intestinal Epithelial cells resulting leakage of nutrients and facilitates entry of pathogen through damaged mucosa
- **AFB1** reduces the size of Bursa, spleen & thymus and thus affects production of both B cells & T cells, leading to immunosuppression, which ultimately results increased susceptibility to other enteritic diseases like Salmonellosis, E coli, Necrotic Enteritis, Coccidiosis, Adenovirus, Rotavirus, Astrovirus infections.
- By damaging epithelial cell integrity, AFB1 directly damages the gut associated lymphoid cells (GALT) and indirectly making the passage

open by destroying the barrier to facilitates entry of many more infections.

- **AFB1** reduces Enzyme activity of digesting Starch, Protein & Lipids in chicken.
- **FB1** affects proliferation of Intestinal Epithelial cell, reduces villi height & villus to crypt ratio and thus affects the normal atmosphere of intestinal epithelium and intestinal microbial homeostasis resulting increase incidence of NE & Coccidiosis.



- **FB1** reduces functional activity of intestine resulting nutrient leakage, diarrhoea, poor digestive output, etc
- **OTA** impacts Tight Junction Integrity and damage intestinal mucosa affects digestive functions
- **T2 Toxins**disturbs Intestinal epithelial cell proliferation, Mucous production & Immunoglobulin production and thus affects Intestinal health & nutrient utilization
- **DON**impaired Nutrient absorption and affects Tight Junction Integrity of Intestinal epithelial cell

Present Mycotoxin contamination scenario compel us to conclude that:

- Almost 100% of common Feed ingredients & finished feed in this subcontinent are contaminated with one or more mycotoxins, and the contamination become arule now due to unseasonal rains, draught, hot & humid weather, crop damage by insect and storage inefficiencies.
- Most mycotoxins damages Gut epithelium & Tight junction integrity even in suboptimal level, which usually been neglected at Feed Manufacturingpoint due to the absence of distinct clinical signs
- Combination of bare minimum level of mycotoxin (combined mycotoxicosis) may not produce any clinical signs but can damage Intestinal Epithelium & Tight junction integrity leading to poor gut health; which is a fact but not considered.
- Mycotoxin negatively impacts of both Humoral & Cell mediated immunity, damages GALT to further deteriorate immunity, damages Gut Barrier to facilitate entry of pathogen into the system and hence the major, if not the main predisposing factor for all enteric & other diseases resulting not only to Poor Gut Health but also mortality leading to huge loss in poultry business

PRESS RELEASE

Proteon Pharmaceuticals Unveils 'BAFACOL™', a bacteriophage-based feed additive for protecting poultry against *E.coli* infections

- ***E.coli* infections are among the most common infections affecting poultry of all ages and categories.**
- **Overuse of antibiotics has led to emergence of antibiotic resistant *E.coli*strains in poultry which can have aserious consequence on human health.**
- **Bacteriophage technology helps preventing bacterial infections naturally, improving the health of poultry and reducing usage of antibiotics**

October, 2020 – Leading global biotechnology company Proteon Pharmaceuticals, a leader inbacteriophage technology for livestock, has launched BAFACOL™, a new poultry feed additive which provides a natural guard against avian pathogenic *E. coli* bacteria.The product offers an innovative solution for Indian poultry farmers to improve the health and safety of their flocks.

Poultry sector is currentlyone of the fastest-growing sectors in agriculture in India. However,bird's health (and consequently bird's productivity) can be affected by rampant threat of bacterial infections such as colibacillosis which is caused by avian pathogenic *E. coli*(APEC). There are different strains of *E. coli* present in the intestines of flocks . While most of them are harmless, certain strains arespecialized in pathogenicity due to the acquisition and expression of virulence genes. *E. coli* strains which are major cause of infections in birds are calledAPEC or avian pathogenic *E. coli*.

Colibacillosis is a major cause of mortality and morbidity in poultry species. Indiscriminate antibiotic usage to treat a variety of bacterial infections has led to a higher prevalence of antibiotic-resistant *E.coli* strains in chicken.Considering that poultry is one of the most popular meat and that avian *E. coli* are more resistant to antibiotics than bacteria from other animal species, it can be extremely dangerous to human health.

Proteon Pharmaceuticals' BAFACOL™is an innovative feed additive comprising five lytic bacteriophages that selectively target APEC.

Introducing the new product in Indiaduring an online session, Ms. Justyna Andrysiak, Chief Product Development Officer at Proteon Pharmaceuticals said, "BAFACOL™ is a cocktail of five lytic bacteriophages that are highly effective only against virulent avian pathogenic *E. coli* bacteria without affecting negatively the beneficial microflora of the gut"

"BAFACOL™ is an environmentally sustainable product that ensures healthy growth of poultry without the need to overuse antibiotics. It can be used in both organic and industrial poultry production to reduce the mortality rate occurring due to avian pathogenic *E. coli,*" **she added.**

Recently, a *in vivo*trial was conducted with BAFACOL™ at Agrivet ConsultancyP Ltd., Kolkata, on broiler chickens for a period of 35-days. The results showed that the product is effective in controlling colibacillosis which showedlower mortality rates compared to the untreated groups.**Dr. Sudipto Haldar, R&D Director at Agrivet Consultancy** said "A controlled experiment was conducted to evaluate the effects of BAFACOL™ as a therapeutic and prophylactic agent against APEC infections. The test induced positive results confirming that BAFACOLmay be considered as an alternative to antibiotic treatments in poultry production to combat colibacillosis"

With the poultry market projected to grow to INR 4,340 billion by the year 2024, it is obvious that the demand for poultry products is set to rise in India. The rampant antibiotic usage in poultry and the wide consumption of poultry products threatens to increase antibiotic resistance in human beings.

Bacteriophages are naturally occurring organisms that attack only specific bacteria, while remaining completely safe for animals, humans and the environment.Phage preparations are a new hope for modern agricultural industry and can help the farmers to prevent bacterial diseases.In nature they co-evolved with bacteria, and every single bacterium has a phage opponent that can control its population. Phages outnumber bacteria by a ratio of 10 to 1, and also play an important role inrecycling the carbon in bacteria. Bacteriophage-based preparations are increasingly gaining interest in the globalmarket as they help farmers raise safe and healthy livestock.

About Proteon Pharmaceuticals

Headquartered in Poland, Proteon Pharmaceuticals S.A. is a leader in bacteriophage (phage) technology for livestock farming and aquaculture. Proteon's products modulate the microbiome, enhancing sustainability and improving performance on the farm. They have created a precision phage development platform that uses omics technologies, molecular biology, bioinformatics and artificial intelligence (AI) to create effective, reliable and safe antibacterial solutions for animal health. Proteon was the first company to develop precision bacteriophage-based feed additives to combat bacterial infections in poultry.

Lamba's

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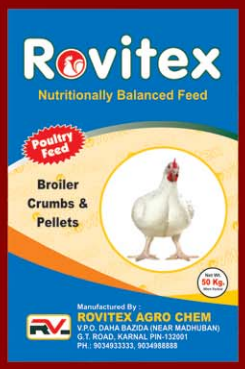
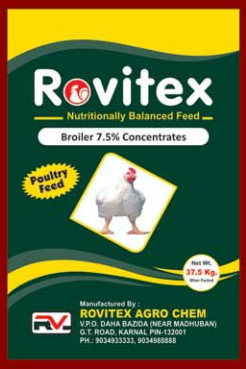
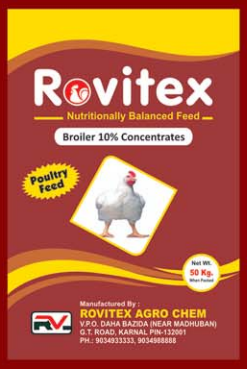
- ❖ Broiler 7.5% Concentrates
- ❖ Broiler 5.5% Concentrates
- ❖ Broiler 3.5% Concentrates
- ❖ Broiler 2.5% Concentrates
- ❖ Broiler 1.5% Concentrates

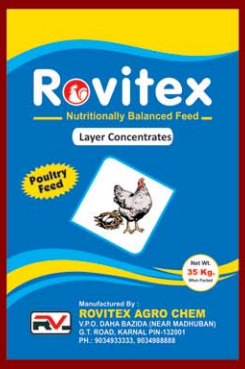
Layer Concentrates:
- ❖ Layer 5% Concentrates
- ❖ Layer 10% Concentrates
- ❖ Layer 25% Concentrates
- ❖ Layer 35% Concentrates

Broiler Crumbs/Pellets:
- ❖ Broiler Pre-Starter Crumbs
- ❖ Broiler Starter Crumbs
- ❖ Broiler Finisher Pellets









ROVITEX AGRO CHEM

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Dealers enquiries solicited from unrepresented areas

Prevention of Egg drop syndrome (EDS 76) in layer flocks

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Tamil Nadu Veterinary and Animal Sciences University

Introduction

Egg drop syndrome is a viral disease affecting laying birds. It is characterized by a sudden drop in egg production or a failure to achieve a normal peak in egg production, as well as its eggshell quality in apparent healthy laying birds.

Incidence: EDS '76 was first identified in Netherlands in 1974. In the autumn of 1976, a distinct egg drop syndrome was first identified in Northern Ireland. Apparently a similar disease had been seen over a 4-year period in broiler parents in Holland. The cause has been identified as Adenovirus BC14, 127, first isolated in Northern Ireland in 1976.

Occurrence: Worldwide, it became a problem in European countries such as Ireland, Holland, France, and the United Kingdom. Other places which have had outbreaks include India, Bangladesh, South America, Indonesia, Japan and Africa. Since then, EDS '76 has been recognized as a global threat for the breeding and laying companies. The virus has been isolated from non-symptomatic migratory ducks in the US.

Species affected: The natural host of Duck atadenovirus A are wild and/or domestic waterfowls such as ducks, geese and swans. More susceptible in chickens of all ages and breeds, especially broilers and brown egg layers. All chickens are susceptible, but brown layers are most susceptible. Guinea fowl, Japanese quails, and turkeys are also susceptible.

Age affected: Adult

Causes: It is caused by the double stranded-DNA virus, called Duck Atadenovirus-A, an avian adenovirus, which is 70-75nm in length. The other name of this virus may include duck adenovirus 1 (DAdV-1), EDS '76 virus (EDSV) and adenovirus 127 (AV-127). The virus grows to high titer in embryonating eggs of ducks or geese, or cell cultures derived from ducks, geese, or chickens—especially well in duck kidney, duck embryo liver, and duck embryo fibroblasts.

Incubation period: 7-9 day.

Transmission: Three patterns of disease are recognized in chickens:

1) **Classical EDS '76** occurs when primary breeding stock are infected and the virus is transmitted vertically through the egg. The virus often remains latent until the progeny chick reaches sexual maturity, at which time the virus is excreted in the eggs and droppings, infecting susceptible contacts.

2) **Endemic EDS '76** is the result of horizontal infection of the flock during lay. It is usually seen in commercial egg layers. Contaminated egg collection trays are one of the main vehicles of horizontal transmission between flocks, and outbreaks are often associated with a common egg-packing station.

3) **Sporadic EDS '76** has been recognized occasionally in flocks. This is due either to direct contact with domestic ducks or geese or, more often, to use of a water supply contaminated with wildfowl droppings. Although infection by this route is uncommon, there is always a risk that these introductions of the virus could form a starting point for endemic disease.

The main method of horizontal spread is through contaminated eggs or equipment such as trays, crates, trucks, or personnel. Droppings are also infective. The virus can be transmitted by bleeding or vaccination needles. Insect transmission may be possible but has not been proved. After horizontal infection, the virus grows

to low titers in the nasal mucosa. This is followed by viremia, virus replication in lymphoid tissue, and then massive replication for ~5 days in the pouch shell gland. Changes in the egg shell coincide with viral replication in the shell gland. Both the exterior and interior of eggs produced between 8 and ~18 days after infection contain virus. Exudate and secretions from the oviduct are rich in virus and pass into the droppings, which may become mildly to moderately watery for 2–3 days. Unlike other fowl adenoviruses, there is little, if any, virus growth in the epithelial cells of the intestine. Interestingly, the massive viral replication in the pouch shell gland occurs after seroconversion, a fact that is useful diagnostically.

Chicks hatched from infected eggs may excrete virus and develop antibody. More often, the virus remains latent, and antibody does not develop until the bird starts to lay, at which time the virus reactivates and grows in the oviduct, repeating the cycle. The infection is commonly present in ducks and geese but does not cause disease.

Clinical signs:

Birds remain generally healthy. **Atadenovirus replicates primarily in lymphoid tissues and massively in the pouch shell gland region of the oviduct, producing various eggshell changes.**

a. **Inappetance, anaemia,dullness and diarrhoea may occur.**

b. Poor egg production and egg quality characteristics such as

- Loss of shell colour in pigmented eggs.
- Failure to reach a peak egg production or egg drop at peak. Drops may be of 5 to 50% and last for 3-4 weeks.
- Poor external quality- Reduction in egg size.Rough, chalky, thin or soft-shelled eggs and shell-less eggs.
- Poor internal quality- Watery albumin
- Lack of signs in the birds themselves.
- There is no effect on fertility or hatchability of those eggs with a shell quality that is satisfactory for setting.
- In cage units, spread can be slow, and the signs may be overlooked or perceived as a small depression (2%–4%) of egg yield.

Post-mortem lesions

- No specific lesion - only a slight atrophy of ovary and oviduct.
- Histopathology - it may be possible to demonstrate degenerative changes in the epithelial cells of the magnum of the oviduct.

Postmortem lesions

The lesions are confined to female reproductive tracts. Lesions include inactive ovaries and atrophied oviducts, uterine oedema, exudate in the shell gland, flaccid ovules and a mild splenomegaly.

Diagnosis

- History
- Clinical signs/ Gross lesions.
- Microscopic lesions- Presence of inclusion bodies in the epithelial cells of the shell gland are diagnostic.
- Isolation of haemagglutinatin agent in duck eggs or cell culture, group antigen distinct from classical adenoviruses (white cells, throat swabs, oviduct).
- Immunofluorescent staining of fluids with specific conjugated antisera, and the Haemagglutination-inhibition test and ELISA to check for antibody in blood are also helpful. It simulates IB.

Differential Diagnosis: EDS '76 can largely be distinguished from other poultry diseases, such as Avian Influenza, Infectious Bronchitis and Newcastle Disease, by the clinical findings alone ie., the absence of respiratory signs, the absence of ridged and malformed eggs, and the absence of poor internal egg quality. Laboratory testing is still needed to eliminate doubts for a definitive diagnosis. Identification of the virus can also be detected by Polymerase Chain Reaction-based test.

Serology: Haemaglutination-Inhibition(HI), Serum Neutralisation (SN), Double Immunodiffusion test (DID), Enzyme-linked Immunosorbent Assay (ELISA). It is important to rule out other possible reasons for egg drop, which can be caused by a large number of factors acting individually or in combination.

Management problems:

- Inadequate water supply
- Extremes of temperatur
- Inadequate lighting programme;
- Sudden changes in feed.

Nutritional deficiency: vitamins E, B_{12}, and D as well as calcium, phosporus, selenium.

Diseases: Diseases in which egg drop occurs, may be infectious or metabolic.

- Infectious diseases include Infectious Bronchitis, Infectious Laryngotracheitis, Avian Encephalomyelitis, Newcastle disease, Marek's disease/Leukosis or any infectious disease causing a significant systemic disturbance (CRD, Coryza, Cholera, Parasites, Diphtheritic Fowl Pox).
- Metabolic diseases include Fatty Liver Syndrome, intoxication by sulphonamides, insecticides or nicarbazin.

Prevention

This disease has been eradicated from primary breeder flocks in most countries. Its entry into layer flocks is further managed by:

(1) preventing contact with other birds, especially waterfowl;

(2) disinfecting all equipment regularly;

(3) chlorination of water.

Biosecurity; All kinds of biosecurity measures are to be adopted.

Hygiene: Cleaning all areas, such as breeding and laying areas, and equipment may mitigate the risk of getting EDS '76. Shared egg trays have to be cleaned and disinfected prior to use. Healthy and uninfected flocks should be kept away upon a contact with those affected birds and the virus natural host. Potential contaminated water should also be chlorinated.

Vaccination:

An inactivated vaccine with oil adjuvant can be administered. This vaccine is given before hens start to lay eggs, usually between 14 and 20 weeks of age. Available vaccines are: Izovac EDS, Nobilis EDS, AVIVAC-EDS-76 and other vaccines. Sentinel chickens may be placed along with vaccinated chickens and periodically checked for antibodies, which would allow detection of the presence of virus in the flock.

Treatment

Prevention is better than cure. Soluble multivitamins may be recommended as a non-specific measure.

PRESS RELEASE

Strategies to combat *E.coli* in poultry farms

Justyna Andrysiak – Chief Product Development Officer _ Proteon Pharmaceuticals SA Poland
Kishore Gedam – Techno commercial Manager – Proteon Pharmaceuticals India
https://ehp.niehs.nih.gov/doi/10.1289/ehp292

Justyna Andrysiak

What is *E. coli*?

E. coli is a gram-negative bacterium that belongs tothe intestinal microflora of livestock, including poultry. These bacteria are capable of surviving long periods outside the host and are present in almost all bird environments, particularly the litter and house dust. Opportunistic infections may occur under certain conditions (stress, weakened immune system, accompanying diseases and infections), however, pathogenic bacteria may also enter the body from the external environment. Poultry feed & water is often contaminated with coliforms and are the most common route of infection with new serotypes. Outbreaks often occur in broilers, layers & breeders causing enteritis, affecting the fallopian tube causing inflammation &colisepticemia are the most common cause of birds'mortality.

Economic losses and estimates

The economic losses due to pathogenic *E. coli*infection can be both: direct and indirect. Weight loss, decreased egg production, increasing mortality and secondary infections affect the livestock production systems. Moreover, disinfection, cleansing, disposal, and excessive use of antibiotics can lead to additional expenses for poultry farmers. The indirect effects comprise the influence on the domestic economy, including interference with major industries, increase in antibiotic resistance, and impact on other sectors.

Susceptibility in poultry farms

Not all age of birds is equally susceptible to the bacterium. When chickens are18 to 30 weeks old, egg production is at its peak. They are still developing, and their bodies are under a lot of stress, making them more prone to various infections. Laying hens that are more than one year old are also quite vulnerable. They breathe in the Avian Pathogenic *Escherichia coli*-laden dust that is quite prevalent in dried out faeces, which tend to accumulate in the layer house in most Indian poultry farms due to poor farm management practices. Pullets are susceptible when their bodies begin to produce hormones that are necessary for egg production. It is a stressful time and their immune systems are not functioning at full capacity, making them an easy target for the colonization of pathogenic bacteria such as *E. coli*. In broilers when reared in deep litter system the prevalence of *E. coli* infection increases due to more exposure to contaminated litter.

Kishore Gedam

Control measures

Biosecurity measures play a key role in controlling the spread of *E. coli*. Keeping the bacteria out of the flock is not practical or possible since intestinal colonization is common in warm-blooded animals. Fortunately, external infections can be limited through feed, water, and environmental sanitation,

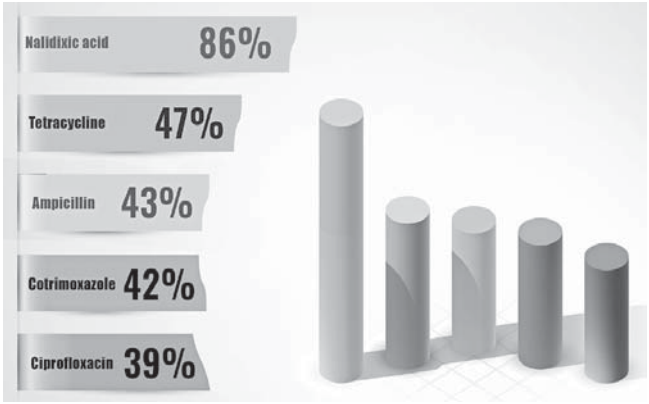

Figure 1: Levels of antibiotic resistance

Nalidixic acid	86 percent
Tetracycline	47 percent
Ampicillin	43 percent
Cotrimoxazole	42 percent
Ciprofloxacin	39 percent

as well as good air quality. Pelleted feed has a lower percentage of *E. coli*bacteria compared to mash feed. Rodent faeces are a ubiquitous source of *E. coli*. Furthermore, contaminated water supply can also contain high numbers of bacteria. One of the possible way to curb the spread of pathogenic microorganisms is to chlorinate the drinking water and use closed watering systems.

Maintaining litter and air quality can greatly reduce the risk of colibacillosis infection. The damage caused to the respiratory mucosa of the flock has a direct correlation to the degree of ammonia exposure. Dust also increases the risk of an infection. The combination of ammonia and dust results in the inhalation of bacteria in high numbers, making it difficult for birds to clear them from their respiratory tract.

Treatment

Although *E. coli*infection is commonly treated with antibiotics, a survey of commercial poultry producers found that chickens raised for eggs and meat have high levels of antibiotic-resistant bacteria. The survey found that more than half of the *E. coli* isolates were resistant to multiple drugs and nearly 60% of them contained broad-spectrum beta lactamase, an enzyme that provides resistance to beta-lactam antibiotics. Broiler farms are twice as likely to be exposed to antibiotic-resistant bacterial strains compared to layer farms due to the high level of antibiotic usage. Independent farms are more likely to developantibiotic-resistant *E. coli* than contracted farms, that are mostly owned by large producers and have to follow strict production protocols, including better veterinary care and hygiene methods. On the other hand, independent farms misuse antimicrobials.

The problem will only get worse. An increase in income and an increase in demand for poultry products would cause an exponential increase in the use of antibiotics in food production. Poultry producers ~~Farm owners~~ must take rigorous action and implement government regulations to control the massive use of antibiotics on poultry farms in India. The transition to a more sustainable way of production should also be promoted by setting up funds to subsidize biosecurity measures at farm level. Poultry farmers should switch to feed additives containing bacteriophages. Since they target specific pathogenic bacteria without affecting the host, they are the most valuable tool in the arsenal of poultry producers in the fight against multi-drug resistant bacteria. Bacteriophages are being adopted successfully by poultry producers around the world, recently introduced to the market in India by Proteon Pharmaceuticals and it is time to mainstream this solution.

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LYSOPHOSPHOLIPIDS Role in Nutrient Absorption Enhancement

Bird performance in the commercial poultry industry has shown a consistent improvement over the past few decades. This change has occurred due to improved genetics, well balanced nutrition and better farm management practices. The industry has undergone a remarkable change and growth over the last 30 years, such that today we see 3 Kg broilers before 40 days of age and white egg layers are capable of producing nearly 340 eggs in 52 weeks of lay. Among the above factors, nutrition plays a vital role in supporting the desired growth and production performance of birds. Provision of a good quality feed with all essential nutrients must be ensured. Also, the nutrients supplied through feed have to be effectively digested, absorbed and assimilated. If the nutrients are not absorbed within the time limit, they are attacked by the bacteria in the large intestine or excreted as waste, defeating the purpose for which they are fed and is reflected in terms of inefficient growth and productivity.

Feed accounts for 65-70% of the total costs in animal production. With the rise in feed costs internationally; the birds' ability to absorb nutrients optimally is a very important aspect of overall performance efficiency. Nutritionists are therefore increasinglyemphasizing tooptimise the feed efficiency and reduce feed cost.

Besides having a superior emulsification property, Lysophospholipids (LPLs) are proven to be very effective in enhancing the flux rate of nutrients across the gut membrane, thereby improving the absorption and reducing the nutrient loss through feces.

Phospholipids &Fluidity of Bilayer Membrane

Membranes define the boundaries of the cell and its organelles and act as permeability barriers.One remarkable feature of all biological membranes is their flexibility; their ability to change shape without losing their integrity and becoming leaky. The"Fluid-Mosaic" model (Singer & Nicolson, 1972)of cell membrane indicates that membranes are made up of lipid bilayer and membrane proteins (peripheral and integral).

The lipid component of the membrane is a two dimensional fluid in which the constituent molecules are free to move laterally. TheLipid bilayer functions as both; a solvent for integral proteins and as a permeability barrier.Peripheral membrane proteins are anchored to the surface of the membrane, while integral membrane proteins contain trans-membrane regions that pass completely through the bilayer.These two classes of membrane proteins contribute to the "mosaic" aspect of the fluid-mosaic model.

Phospholipids (PLs) are the most abundant lipids found in membranes. These are amphipathic in nature, having hydrophilic as well as lipophilic properties. PLs are characterized by a glycerol backbone to which a polar hydrophilic Phosphodiester(head) group and two lipophilic hydrocarbon tails are linked. The tails are usually fatty acids derived acyl residues thatcan differ in length (normally contain 14 to 24 carbon atoms).

Along with phospholipids, the animal cell membrane also contains significant amounts of sterols, mainly cholesterol, which is necessary for maintaining and stabilizing the membrane by acting as a fluidity buffer. Though the lipid bilayer structure is quite stable, its individual phospholipid and sterol molecules have some freedom of motion. Lysophosphatidylcholine (LPC) like molecules affect the fluidity of the cell membrane by modifying cholesterol levels in the membrane and thereby dynamics of the membrane.

Lysophospholipids: The Fluidity Modulator

Lysophospholipids (LPLs) are glycerophospholipids in which one acyl chain is lacking as compared to PLs and therefore only one hydroxyl group of the glycerol backbone is acylated. LPLs which include lysophosphatidylcholine (LPC), lysophosphatidy lethanolamine (LPE), lysophosphatidyl inositol (LPI) and Lysophosphatidylserine (LPS) are prepared by enzymatic hydrolysis of natural soy lecithin by phospholipase A2.

Each membrane at equilibrium contains pores and holes – these are best thought as gaps or vacancies where phospholipids are missing from the lattice structure. Sometimes there are clusters of these vacancies of various sizes. When additional LPLs are introduced, it is this distribution that is affected, which results in an increase in both the number and the size of pores. Through the normal passive transport processes, nutrients of larger molecular weights can then pass more readily across the membrane. When the membrane comes into contact with a certain ratio of LPLs, these exogenous LPLs quickly get interdigitated into the bilayer membrane. The close packing between the PLs is disrupted and the lipids go from an order to disorder state and the membrane becomes more fluid i.e. the gaps or pores in the membrane form big clusters or larger vacancies in the matrix causing an increase in the number and size of pores. This means that the nutrient absorption profile of the gut is beneficially altered with the passive flux hurdle temporarily lowered.

Also, LPLs have the ability to change the attraction between lipids and displace calcium ions. With this increased freedom of movement, lipids can aggregate closer together making existing holes larger so that larger molecules are easily absorbed.A pre-determined ratio has to be maintained between PLs and LPLs, and also between different LPLs to observe a consistent and desired end result.

The above describes how Lysophospholipids (LPLs) act as a membrane fluidity modulator. They increase the number and size of pores by altering the mechanical properties of the membrane, thereby enhancing the flux rate at which nutrients of various molecular sizes pass across the membrane of the gut and thereby act as an absorption enhancer. This is one of the key applications of LPLs in animal nutrition as it is possible to extract more nutrient value from every kilogram of the diet and thereby optimize feed efficiency and therefore feed cost.

PRESS RELEASE

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Avitech launches Avitriol - Natural Bioactive instant Vitamin D_3

Avitech Nutrition is pleased to announce the launch of Vitamin D_3-Avitriol.Avitriol is a unique form of Vitamin D_3 that has been derived from the Waxy Leaf Nightshade plant. Avitriol is a 100% natural productcontaining $1,25(OH)_2D_3$, the metabolically active form of Vitamin D_3, in a glycosidic form. Avitriol represents a form of Vitamin $D_3$with increased bioactivity and fastermechanism of action. Avitriolis readily absorbed through the intestine and, unlike other Vitamin D_3 brands available in market, does not require any activation by liver and kidney of animals. Avitriol maximizes the calcium-phosphorus mobilization in bones and muscles, thereby enhancing animal performance.

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place	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31
Hyderabad	110	105	98	100	103	112	120	125	126	126	126	126	126	119	110	106	111	117	122	122	122	117	111	114	117	120	120	115	107	99	104
Karimnagar	110	105	98	100	103	112	120	125	126	126	126	126	126	119	110	106	111	117	122	122	122	117	111	114	117	120	120	115	107	99	104
Warangal	110	105	98	100	103	112	120	125	126	126	126	126	126	119	110	106	111	117	122	122	122	117	111	114	117	120	120	115	107	99	104
Mahaboobnagar	110	105	98	100	103	112	120	125	126	126	126	126	126	119	110	106	111	117	122	122	122	117	111	114	117	120	120	115	107	99	104
Kurnool	110	105	98	100	103	112	120	125	126	126	126	126	126	119	110	106	111	117	122	122	122	117	111	114	117	120	120	115	107	99	104
Vizag	115	115	110	110	112	112	120	125	126	126	126	126	126	126	118	113	113	113	116	116	116	111	105	108	111	114	114	114	109	103	103
Godavari	115	112	107	109	112	112	120	125	126	126	126	126	126	126	120	115	115	118	123	123	123	118	112	115	118	121	121	121	116	110	110
Vijayawada	115	112	107	108	112	112	120	125	126	126	126	126	126	126	120	115	115	120	123	123	123	118	112	115	120	123	123	123	115	107	110
Guntur	115	112	107	108	112	112	120	125	126	126	126	126	126	126	120	115	115	120	123	123	123	118	112	115	120	123	123	123	115	107	110
Ongole	115	112	107	108	112	112	120	125	126	126	126	126	126	126	120	115	115	120	123	123											

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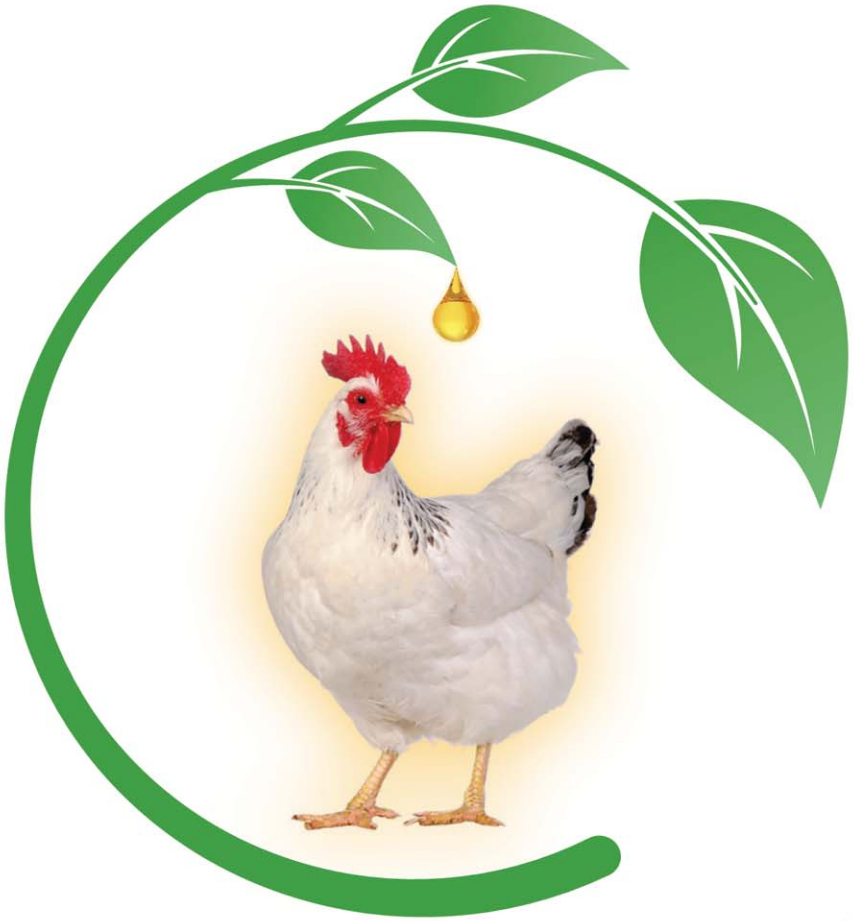

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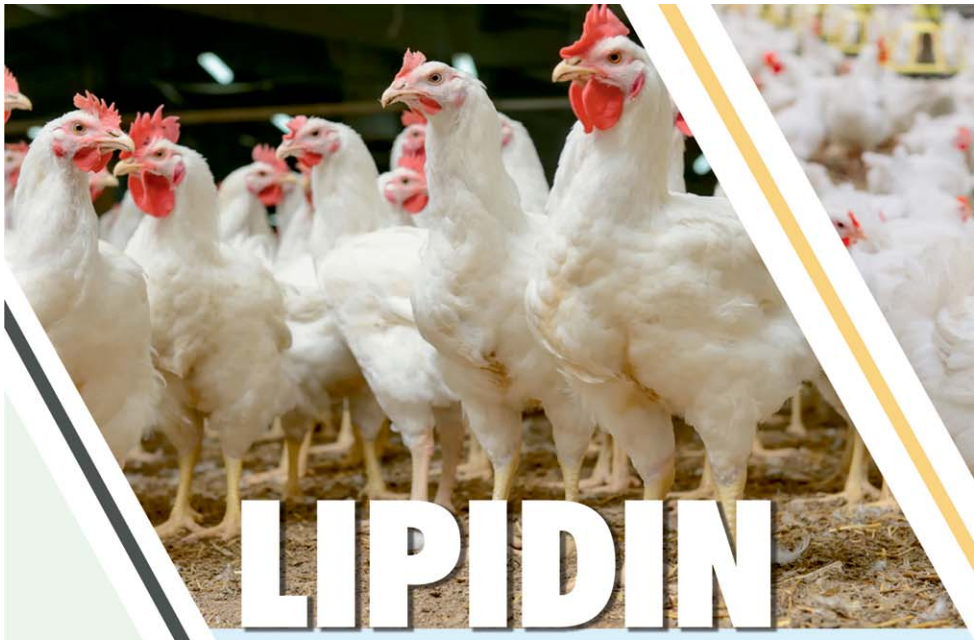

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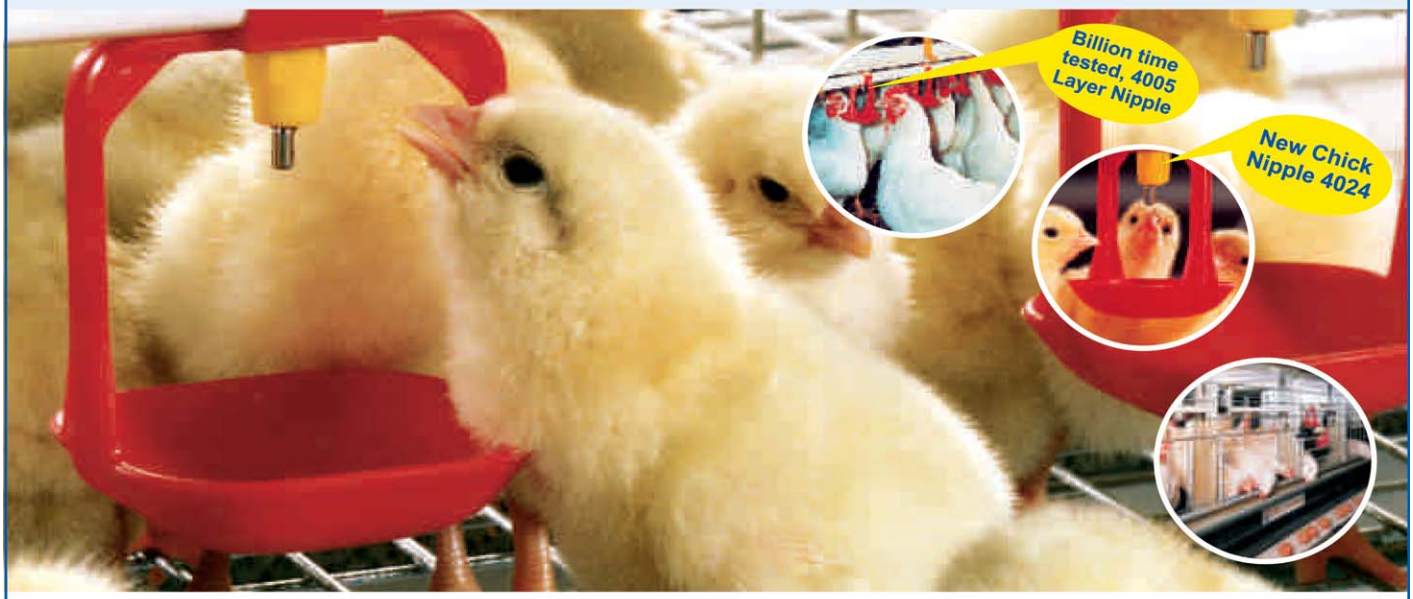

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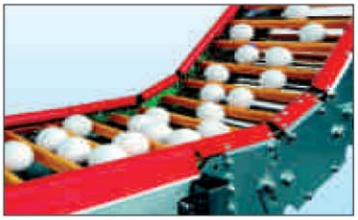

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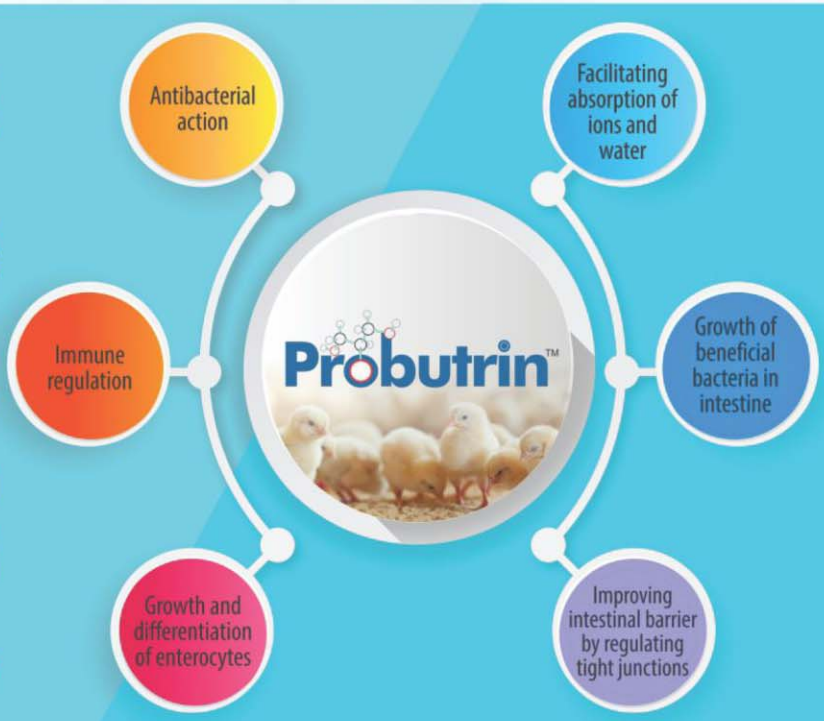

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