

Disease continues to impact

Health problems have always been a part of the poultry industry. Some of these can be easily controlled while others are causing more reasons for concern. The intensification of the poultry industry seems to be paying its toll.

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Despite ongoing research by universities and biologics manufacturers and the efforts of regulatory agencies, the world's poultry industries continue to be impacted by both catastrophic and erosive infections. During the current year either velogenic Newcastle disease or highly-pathogenic avian influenza has caused extensive losses in the USA, the Netherlands, Italy and Southeast China. Intensification of the poultry industry to achieve enhanced efficiency has resulted in high concentrations of broilers and egg-producing flocks creating the emergence of variant pathogens and enhanced dissemination of viral and bacterial diseases. Reduction in the intensity of vaccination and a decline in the standards of bio-security, in an attempt to reduce costs in competitive markets have also contributed to the frequency and severity of disease outbreaks.

Catastrophic diseases

Avian influenza which has resulted in extensive losses in North-central Italy over the past five years has not been eradicated. The emergence of H7N7 highly-pathogenic influenza in Holland in March 2003, with subsequent extension into Belgium and Germany, has resulted in the culling of over 25 million commercial birds. The disease has brought about a cessation of exports of breeding stock, hatching eggs and chicks in addition to disrupting the supply of commercial meat and eggs to the EU and to long-standing export markets.

An outbreak of velogenic Newcastle disease, which has in all probability become endemic in the backyard and game fowl (fighting cock) populations of Southern California, has generated control costs of over \$100 million during the first five months of 2003. Over three million hens have been depleted from infected and contact farms. As of the beginning of May 2003, the incidence rate in commercial units has declined and the Federal task force responsible for eradication has not-



Health problems cannot be hidden under the wings of a bird, no matter how wide they are spread.

ed a decrease in reports of cases in non-commercial flocks. Given the passage of time, intensity of vaccination will decline and bio-security procedures will be relaxed, in the interests of reducing cost. This will create greater susceptibility and opportunity for extension of infection into the commercial sector. Re-introduction of infection across the southern border of the United States is a real possibility as vvND is endemic in a number of border states in neighbouring Mexico. The occurrence of vvND in a small flock in El Paso, Texas, a border town, suggests a new introduction since the virus was characterized as different from the strains prevalent in California.

Low-pathogenicity H7N2 influenza has circulated among the flocks supplying the live-bird markets of the US and Northeast Seaboard over the past 15 years. This virus was responsible for an outbreak of clinical influenza in turkeys during early to mid-2002, resulting in eradication costs exceeding \$90 million.

Outbreaks of H7N2 influenza in a single large commercial egg production enterprise in Connecticut followed by a second case in Rhode Island confirm the vulnerability of egg production units. Both State and Federal authorities have agreed to

the administration of inactivated H7 vaccine using the DIVA principle, as an alternative to eradication. Affected flocks have been quarantined but washed, packed eggs can be shipped directly to retail outlets. Avian influenza is also responsible for losses in the People's Republic of China as evidenced by isolation of pathogenic virus from birds imported into Hong Kong for live sale.

Erosive infections

Relatively mild infections including coccidiosis, *E.coli* septicemia, laryngotracheitis, mycoplasmosis and infectious bursal disease continue to reduce growth rate, liveability and feed conversion efficiency in affected flocks. Emergence of variant strains of both infectious bursal disease and avian bronchitis viruses add to the problems of selecting appropriate vaccines and programs for administration. It is evident that a high concentration of poultry in close proximity allows dissemination of variants. Within three years of the emergence of the Delaware variants of IBDV, virtually the entire broiler industry east of the Mississippi was affected with these strains. There was evidence that the Delaware IBD viruses are now the predominant

the world's poultry industries



in specific regions of Brazil with negative implications for production efficiency and cost.

Emerging conditions

Deletion of growth-promoting antibiotics with activity against gram-positive bacteria in the intestinal tract has resulted in an increase in clostridial enterotoxemia. Cases of necrotic enteritis, gangrenous dermatitis and botulism are encountered frequently in countries where bacitracin and virginiamycin have been banned. The evaluation of non-antibiotic feed additives which suppress *Clostridia* and promote growth is an ongoing concern. Although mannanoligosaccharides and some botanicals have been shown to modify intestinal flora, direct replacement of bacitracin and virginiamycin as growth-promoting antibiotics is not cost-effective under a variety of commercial situations, especially when concurrent coccidiosis and stressful environmental conditions pertain.

Zoonotic infections

The emergence of zoonotic infections associated with poultry is a disquieting trend following the 1997 cases of avian influenza transmitted to human contacts of live poultry in Hong Kong. The recent HPAI outbreak in the Netherlands has so far resulted in 80 cases of conjunctivitis and one fatality attributed to infection.

Food-borne diseases associated with poultry including paratyphoid salmonellosis, listeriosis and campylobacteriosis have been the subjects of research and attempts at pre-harvest control. At the present time, modalities including vaccina-

tion, litter management, acidification of water and feed additives may only contribute to suppression of infection with no certainty of elimination of contamination. Levels of *Salmonella* on broiler carcasses have been reduced in the USA from approximately 20% to 10%, following the introduction of mandatory HACCP systems which essentially dictate chlorination of water used in spin chillers and bird washers, coupled with increased overflow rates. It is anticipated that the triad of *Salmonella*, *Campylobacter* and *Listeria* will continue to challenge both producers and regulators.

Future measures for disease prevention

Control of poultry pathogens will depend on simultaneous application of efficient biosecurity, enhancing the immune system and more effective vaccination. Fortunately the world's major biologics manufacturers are developing a range of genetically engineered vector vaccines some of which may be administered *in ovo*, in addition to a complement of inactivated oil emulsion products suitable for mature breeder flocks.

The emergence of variants of existing pathogens and changes in the epidemiology and clinical presentation of poultry diseases will continue as a result of intensification, the more extensive use of live attenuated vaccines and an increase in the movement of live birds and products both within national boundaries and in export trade. An understanding of the epidemiology of the catastrophic and erosive diseases will lead to more effective measures of prevention and control. ■

serotypes in Central America, requiring adjustment of both parent and broiler vaccination programs. A highly-pathogenic form of IBD which resulted in extensive losses in the EU and Asia now occurs

Emerging and re-emerging diseases in poultry

Poultry diseases remain of major economic and public health importance. In recent years, public health concerns for food borne bacterial infections and antibiotic resistant strains have increased, causing major political issues in several countries. The following highlights key developments in the field of emerging and re-emerging poultry diseases.

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Poultry diseases are associated with severe economic losses. Early recognition and monitoring programmes are essential in managing the infections and minimizing the economic

impact. With new and re-emerging diseases evaluating their economic impact is essential to determine whether new control measures and especially new vaccines are need. Generally, therapy or

vaccination alone is of little value unless they are accompanied with improvements in all aspects of management and biosecurity. In the long term, development of poultry lines that are genetically resistant to some pathogens should be progressed and further attention must be paid to development of efficient vaccines against bacterial infections aimed to reduce the use of antibiotics.

Worldwide the new emerging bacterial diseases are Amyloid Arthropathy and *Ornithobacterium rhinotracheale* infections. In addition, Avian Influenza, Mycoplasmosis, Erysipelas, Necrotic Enteritis, Chlamydiosis and Histomoniasis appear to be making a comeback. Reports during recent years reveal *Salmonella* (specially *S. enteri-*

tidis) and *Campylobacter* spp. are the most common causes of human food borne bacterial diseases linked to poultry products.

Avian influenza (AI)

Avian influenza (AI) caused by influenza A viruses is a disease of many kinds of poultry, wild and cage birds. It is characterised by marked variation in morbidity, mortality, signs and lesions. In addition, the infection causes periodic epidemics in humans, horses, pigs, seals, whales and a variety of birds.



Turkey poult suffering from severe sinusitis caused by LPAI-H6N2.

AI viruses are members of the Orthomyxoviridae family. Within the family there are three types of influenza: A, B and C. Type B and C affect only humans. Type A viruses can affect birds, human and mammals such as horses, pigs, seals and whales. The virus is RNA, enveloped, sensitive to ether, chloroform and different chemical disinfectants. The virus genome is single stranded, which occur as eight separate segments. Influenza A viruses are divided on the bases of the antigenic relationships in the surface glycoproteins haemagglutinin (HA) and neuraminidase (NA) into subtypes. There are at present 15 H subtypes and 9 N subtypes. The viruses are known to have a high mutation rate.

Wild and domesticated waterfowl are the major natural reservoir of influenza viruses. Representatives of all of the different subtypes of influenza A viruses have been isolated from birds, particularly from aquatic species such as ducks, geese and gulls. They may be infected with more than one type without clinical signs, excrete the virus for long periods and mostly do not develop detectable antibodies.

After the first reports on isolation of an influenza virus from turkeys with respiratory and other general disease signs in 1963, most of the major turkey-producing countries have had Low Pathogenic Avian Influenza (LPAI) problems at one time or another. Recently, several outbreaks caused by LPAI subtype H9N2, H6N2, H7N2, H7N3 were observed in many

countries worldwide.

In 1999, low pathogen Influenza A H7N1 was detected in turkey flocks in Italy, accompanied by a high mortality rate and a turn to virulence by the end of the year 2000. In addition, HPAI of subtype H7N7 visited the Netherlands in March 2003.

The disease transmitted directly through contact with infected birds or indirectly through contaminated equipments, people (shoes, clothing), crates, egg flats and egg case vehicles.

The virus can survive in the contaminated environment for long periods of time at moderate temperatures, and can survive longer in frozen material. Rodents and insects may mechanically carry the virus from infected to susceptible poultry. There is little or no evidence of vertical transmission (egg-borne infection) in poults. However, eggshell surfaces can be contaminated with the virus.

Effect on humans

In human outbreaks influenza A of subtypes H1N1, H2N2 and H3N2 appear to be the most common one. In some cases however, people with intensive contact with poultry and swine can attract infection with other subtypes. In 1996 an H7N7 virus was isolated in England from the eye of a woman with conjunctivitis who kept ducks. This virus was shown to be genetically related to a virus of H7N7 subtype isolated from turkeys in Ireland in 1995. Influenza A H5N1 was isolated from humans in Hong Kong in 1997. The virus was previously known to infect birds only. In 1998, there were 18 confirmed human cases with 6 deaths. Investigation of the first case indicated that the main mode of transmission was bird to man. At this time, there are still no definite signs of human-to-human transmission, but even if it occurs, the efficiency of transmission is low at this time. In April 1999, H9N2 AI virus was isolated from humans in Hong Kong and in China with influenza-like disease, which suggested that H9 viruses found in the region were able to infect human.

No contact with wild waterfowl

Since the avian influenza virus in nature is maintained in wild aquatic birds reservoir, it seems to be difficult, even impossible, to eradicate the infections. However, every effort must be made to prevent direct or indirect contact between domestic poultry and wild waterfowl. Persons handling wild game (especially waterfowl) must change clothes completely and bathe prior to entering poultry houses. A vaccination program, in conjunction with strict quarantine, has been used to control mild forms of the disease in commercial chicken and turkey flocks. With the more lethal forms of the disease, however, strict quarantine and rapid depopulation of infected flocks seem to be a very effective method of stopping avian influenza.

However, in some places applying of inactivated vaccine against highly pathogenic influenza viruses have revealed promising results. The success of such a program depends, of course, on the full cooperation between governments, veterinarians and the poultry industry.

In addition, since evidence collected from several influenza outbreaks indicate that HPAI viruses of H5 and H7 subtypes may emerge from LPAI, it would be necessary to limit the spread and presence of LPAI viruses of H5 and H7 subtype. This is an important reason for re-defining AI laid down in legislations.

Necrotic Enteritis (NE)

Some feed additives significantly enhance digestion of nutrients in a wide range of species. They also act to control specific diseases such as necrotic enteritis in poultry and improve welfare by increased use of nutrients and decreased undigested material deposited in the environment of the birds. A wide range of antibacterial compounds act as antibiotic digestive enhancers or growth promoters.



Necrotic enteritis causes severe inflammation and necrosis of the intestine.

The potential public health risks from vancomycin resistance in *Enterococcus faecium* and *Enterococcus faecalis* has led to the banning of avoparcin in the European Union. Currently, only 2 digestive enhancers remain approved for poultry in the EU. In response to public concerns, producers in Europe are discontinuing the use of additives. This practice is associated with an increase in the incidence of NE in poultry flocks in all European countries.

Cause of infection

NE is an acute disease caused by *Clostridium perfringens* when it migrates from the large intestine and caeca to the small intestine where it produces toxins. Toxin type A or C is responsible for damaging the intestinal lining. Dysfunction of the alimentary tract predisposes the animal to infection. Intestinal stasis, intestinal distension, coccidiosis, salmonel-

losis, crop mycosis and haemorrhagic enteritis (HE) may predispose the birds to infection.

Factors predisposing the intestinal tract to overgrowth by clostridia organisms may also be the consumption of diets high in energy, protein and fishmeal as well as the consumption of high fibre litter and a wheat based diet. The clinical signs appear suddenly; apparently healthy birds may become acutely depressed and die within hours. Mortality ranges between 2 and 10%. Affected birds show ruffled feathers, marked depression, inappetance, tendency to huddle, watery droppings and diarrhoea.

After experimental infection, the first mild clinical signs are evident approximately 24 to 36 hours after administration of a pure *C. perfringens* culture to broiler chickens in floor pens. On autopsy dehydration is the most common finding. Breast muscles are dark red and gizzards are full of litter. Severe inflammation in the duodenum and jejunum is the most predominant finding, but in some instances the entire length of the intestinal tract is involved. The intestine is distended and contains dark offensive fluid. The mucosa is covered with green or brown diptheroid membrane, which can be easily separated from the lining. Varying degrees of sloughing of the intestinal mucosa could also be observed. As the condition progresses, areas of necrosis can be recognized from outside of the intestine. A tentative diagnosis may be made from the history, signs and lesions to commence treatment. The final diagnosis is based on staining fresh smears of intestinal contents with Gram stain showing an abundant number of Clostridia organisms. Specific isolation and identification of the causative agent may be necessary in some cases.

Associated conditions

Several conditions are associated with *Clostridium perfringens* infections such as Cholangiohepatitis and Dysbacteriosis.

Cholangiohepatitis causes severe economic losses because slaughtered carcasses are downgraded and due to high liver condemnation rate on the processing line even though there are no visible clinical signs on the farm. *Clostridium perfringens* is usually isolated in association with the disease. Onderka *et al.* (1990) experimentally reproduced the condition by either tying off the bile ducts or injecting *C. perfringens* into the bile duct. It seems that clostridia either block the bile ducts or migrate into the liver and interfere with liver function.

Dysbacteriosis is defined as the presence of a qualitatively and/or quantitatively abnormal flora in the small intestine causing a clinical disorder and/or malabsorption. It is seen in broilers after 21 days of age with wet faeces and a reduction in feed intake. Panneman demonstrated in 2000 that the proximal intestine of normal birds has very low

levels of bacteria, whereas birds affected with dysbacteriosis have substantially higher bacterial numbers. *Clostridium* spp. have been shown to contribute to this overgrowth.

Treatment with antibiotics such as penicillin, amoxicillin, ampicillin, erythromycin, dihydrostreptomycin and tetracyclin provided a satisfactory clinical response. Three days is the minimum duration of treatment, however longer applications may be required. Recent investigations indicate that competitive exclusion can impact the incidence and severity of NE in poultry. In addition coccidiosis may be a contributing factor and attention must be given to an effective control program. Brennan *et al.* reported in 2001 that Narasin, when administered at 70 ppm in feed from day 0 to 41 prevents morbidity, mortality and suppression of growth and feed conversion associated with NE in broilers. Brennan also expressed that administration of dietary Tylan[®] for seven consecutive days following confirmation of an NE field outbreak reduced the NE mortality and lesion score and improved overall growth as well as feed conversion in broilers. The optimum dose of Tylan for control of NE was 100 ppm.

Ornithobacterium rhinotracheale (ORT)

Respiratory disease conditions continue to cause heavy economic losses in the poultry industry. Since December 1991 respiratory disease with different clinical courses have been observed in poultry flocks in different countries.



ORT may show severe respiratory distress and prostration.

Bacteriological examinations have resulted in isolation of slowly growing, pleomorphic gram-negative rods (PGNR). Initially, the bacterium was designated as Pasteurella-like, Kingella-like, Taxon 28 or pleomorphic gram-negative rod (PGNR) before in 1994 the name *Ornithobacterium rhinotracheale* gen. nov. sp. nov. in the rRNA-Superfamily was suggested. The infection occurs worldwide and incriminated as a possible

causative agent in respiratory disease. ORT has been isolated from chickens, chukar partridges, ducks, geese, guinea fowl, gulls, ostriches, partridges, pheasants, pigeons, quail, rooks and turkeys. It is an acute highly contagious disease of chickens and turkeys. The severity of clinical signs, duration of the disease and mortality are extremely variable and influenced by many environmental factors such as poor management, inadequate ventilation, high stocking density, poor litter conditions, poor hygiene, high ammonia level, concurrent diseases and the secondary infections. The disease spreads horizontally by direct and indirect contact. Vertical transmission is suspected since ORT has been isolated at very low incidence from reproductive organs, hatching eggs, infertile eggs and dead embryos. Currently 18 serotypes designated A to R exist. Neither the origin nor the serotype of the *Ornithobacterium rhinotracheale* strains effect its pathogenicity. Most of the chicken isolates belong to the serotype A. Turkey isolates are more heterogeneous and belong to serotype A, B and D. Reports indicate synergism between ORT and Newcastle disease, turkey rhinotracheitis, Infectious bronchitis, *Bordetella avium*, *Escherichia coli*, and *Chlamydophila psittaci*.

Identifying the causative agent

Diagnosis of ORT on the basis of clinical features and pathological lesions is often difficult since it can be confused with other infectious conditions. Proof of infection therefore must be carried out by isolation and identification of the causative agent. ORT can be identified using immuno-histochemical staining as well as polymerase chain reaction.

Serological examinations for detection of antibodies can be carried out using slide agglutination test, dot-immunobinding assay and ELISA. The serotype specificity of the ELISA depends on the method of antigen extraction used for coating the ELISA plates. Boiled extract antigens are serotype-specific, while antigen extraction with sodium dodecyl sulphate (SDS-antigen) results in more cross-reactions and is suitable for testing sera from the field. Self made ELISA (SDS-extraction) as well as two commercially available ELISA-kits (Biocheck, The Netherlands, IDEXX, USA) are able to detect antibodies against all tested ORT serotypes. Examination of serum samples collected from commercial flocks using all three systems showed similar results. The advantage of the serological tests over bacteriological examination is that antibodies persist for several weeks after infection but bacterial shedding is brief. ORT excretion and antibody response may also be affected by a number of factors such as antibiotic therapy and vaccination.

The influence of antibiotic therapy on the serological response to ORT remains unclear. In 2002 Popp and Hafez investi-

gated effects of amoxicillin drug therapy on antibody kinetics after experimental infection. Three groups of SPF layers each of 10 birds were experimentally infected with an ORT strain at 36 weeks of age intravenously. Each bird received 5×10^8 cfu. Group 1 was kept as the infected non-treated control. Group 2 was infected and treated immediately with amoxicillin at dose level of 250 ppm via drinking water for 5 days. Group 3 was infected as mentioned above and received amoxicillin for 5 days starting at 7th day post infection. An additional group (Group 4) was kept as a non-infected non-treated control. Blood samples were collected at 5 day intervals till the 50th day post infection and tested for antibodies against ORT using ELISA. The results showed that immediate treatment did not influence the course of the antibody response. While the treatment started at the 7th day post infection resulted in lower antibody response compared to the infected control.

Difficult to treat

The treatment of ORT infections is very difficult because different strains have variable susceptibilities to antibiotics. ORT acquires resistance easily against antibiotics. The sensitivity pattern depends on the source of the strain and the routinely used drugs in an area. It should be emphasized that for successful treatment an investigation of the sensitivity pattern of the isolated strain is necessary. The most published results regarding to the sensitivity tests are based on disc diffusion test and are difficult to compare. Using this method all tested ORT isolates (100%) showed high sensitivity to amoxicillin, chloramphenicol and chlortetracycline. Ninety percent and thirty six percent of the isolates were found to be sensitive to erythromycin and furazolidone respectively. In addition, only 6% of tested isolates were found to be sensitive to enrofloxacin. None of isolates were susceptible to apramycin, neomycin, gentamicin and sulphonamide/trimethoprine. The sensitivity to enrofloxacin seem to be origin-related, since most of turkey isolates from Germany and the Netherlands were resistant, while 98% of strains from France and 71% of isolates from Belgium are sensitive to enrofloxacin. Under field conditions, water medication using amoxicillin at a dose level of 250 ppm for 3-7 days gave satisfying results in most cases. Also application of chlortetracycline at dose level of 500ppm in drinking water for 4-5 days appears to be very effective. ORT is highly sensitive to chemical disinfectants. However, ORT is endemic and can affect every restocking even in previously cleaned and disinfected houses especially in areas with intensive poultry production as well as in multiple age farms.

Vaccination trials using autogenous bacterins successfully reduced the number of outbreaks of ORT infections in turkey flocks in the field. Vaccination trials us-

ing inactivated vaccine on mineral-oil adjuvant bases in broiler, broiler breeders as well as in turkey flocks were carried out. The primarily results are promising and the vaccine gives a good protection. Live vaccination is also feasible, but up to now no non-virulent strains of ORT have been found. A temperature sensitive mutant of *O. rhinotracheale* has some protective properties, but more tests are needed to evaluate the efficacy and safety of this strain.

Bacterial infections and microbial food borne diseases

Food safety is concerned with the hazards associated with poultry meat, eggs and other products that form part of the human diet. Food safety and the quality of food are currently issues of major concern in developed countries.



Feed additives or vaccines are a method to reduce salmonella colonisation.

In developing countries, however, efforts to produce sufficient food to meet the requirements of the population are accompanied by bad economic conditions that overshadow food safety. Safe food is a fundamental requirement of all consumers, rich or poor. Incidents of food borne disease in humans have increased considerably worldwide in the last few years. Although the sources of infection are mostly unknown, poultry products have repeatedly been implicated. Poultry can harbour different food borne pathogens. Many reports during recent years have shown that *Salmonella* (specially *S. enteritidis*) and *Campylobacter* spp. are the most common causes of human food borne bacterial diseases linked to poultry. Recently, verotoxin producing *Escherichia coli* O157:H7 (VTEC) has surfaced as an additional food borne pathogen causing human illness. Several other microorganisms such as *Clostridium perfringens* and some *Listeria* species can also enter the human food chain via contaminated poultry carcasses.

In addition, the development of antibiotic resistance in bacteria, which is com-

mon in both animals and humans, is also an emerging public health hazard. One such example is certain strains of *Salmonella typhimurium* DT104. Controlling these food borne organisms requires a fuller understanding of how microbial pathogens enter and move through the food chain, and the conditions that promote or inhibit growth for each type of organism. Some invasive salmonellas are the most important source of infection in poultry and appear to be vertically transmitted through contaminated eggs laid by infected carriers. Lateral spread of infection takes place through contaminated feed, water, equipment and the rearing environment. The significant reservoirs for these microorganisms are man, farm animals, pets, pigeons, waterfowl and other wild birds. *Salmonella* has been detected in different species of wild bird near hatcheries and poultry processing units and may contaminate equipment. Rodents are recognized as potential reservoirs transmitting infection between houses and contaminating stored feeding stuffs. In addition, insects are a potential source of salmonella infection in chickens.

Transmission unclear

The route of infection with *Campylobacter jejuni* / *coli* in poultry is not fully known. Generally, no evidence has been found either for transmission from one flock to the next via persistent house-contamination. However, because the organism is found in the intestines of most slaughtered poultry, the major route for campylobacter contamination of poultry appears to be the horizontal transmission from the environment. Vertical transmission is suspected.

Beef and raw milk are the main sources of verotoxin producing *E. coli* (VTEC) infections in humans. Transmission of VTEC strains from pets and poultry to humans cannot, however, be excluded. Poultry appears to be susceptible to *E. coli* O157:H7 infections. Experimental oral infections have demonstrated the ability of this organism to colonize the caeca of chickens without causing clinical signs. The incidence of VTEC in poultry and poultry meat products varies widely. The routes of infection and transmission in poultry flocks are not fully known.

Strategy for control

In general the main strategy for control of microbial food borne hazards should include: Cleaning the production pyramid from the top (in the case of invasive salmonella) by destroying infected flocks (with compensation), hatching egg sanitation and limiting introduction and spread at the farm through Good Animal Husbandry Practices (GAHPs). To achieve GAHPs, effective hygiene measures should be applied to poultry houses and their environment and the feed. In addition, reducing salmonella colonization

by using feed additives, competitive exclusion treatment or vaccines is a possibility.

Blackhead (Histomoniasis)

Histomoniasis also known as Blackhead or enterohepatitis, is a disease of chickens and turkeys with a worldwide distribution. It has been responsible for considerable economic problems to the poultry industry.



Inflamed caeca and liver necrosis are typical symptoms of Histomoniasis.

Within the past three decades the incidence of histomoniasis has been substantially decreased by improved management practices by means of separation of turkey and chicken flocks as well as the trend toward confinement rearing and effective prophylactic drug treatment as feed additive.

In Europe, the trend away from intensive rearing to alternative rearing has increased remarkably, especially in the table eggs production sector, and resulted in re-emerging of important poultry diseases of the past. In Europe infections with Histomoniasis have never played any important role for the past 20 years in poultry farms with intensive rearing system. In the last four years several outbreaks of Histomoniasis were firstly ob-

served in layer flocks kept in free range rearing systems. Thereafter, further outbreaks were detected in turkey and broiler breeder flocks as well as in commercial turkeys. During the late 1990s the broiler breeder pullet industry in the USA suffered numerous clinical outbreaks of blackhead disease, with high morbidity, considerable mortality, and lost productivity. As bacterial flora and especially *Clostridium perfringens* play a very important role in development of the histomonads and as a wide range of antibiotic growth promoters were banned in the EU or in response to public concerns, producers are discontinuing the use of additives, the incidence of NE in poultry flocks in all European countries has increased and at the same time an increase in the number of blackhead cases is observed. Recently, McDougald and HU demonstrated that blackhead diseases could be aggravated in broiler chicken by concurrent infection with caecal coccidiosis.

A protozoan parasite

Blackhead also known as enterohepatitis is caused by a protozoan parasite called *Histomonas meleagridis*. The organism is passed in the faecal material of infected birds. In many instances, the organism is shed within the eggs of the caecal worm of chickens, turkeys and game birds. In the environment the organisms do not survive long, but those in caecal worm eggs may survive for years. Transmission may also occur by the earthworm. Outbreaks in turkeys can often be traced to direct or indirect contact with ranges, houses or equipment previously used by chickens. Free-flying birds may also contribute to an infection. However, after introduction of histomoniasis into a turkey flock, it can spread rapidly without the necessity of vectors such as caecal worms or earthworms. The oral route of infection (in the absence of vectors) does not appear to be an important route of infection, but "cloacal drinking" was somewhat effective in initiating infections.

The parasite invades the caecal mucosa and the spreads via the blood to the liver. Most blackhead losses occur in young

birds (six to sixteen weeks). Among the symptoms are loss of appetite, increased thirst, droopiness, drowsiness, darkening of the facial regions and diarrhoea. Morbidity and mortality are variable, but mortality seldom exceeds 15%; however, it may approach 100% in uncontrolled turkey outbreaks. Lesions of uncomplicated cases are confined to the caecae and liver. The caecae are ballooned and walls may be thickened, necrotic and ulcerated. Caseous cores within the caecae may be blood tinged. Livers are swollen and display circular depressed areas of necrosis. Lesions are yellowish to yellow-green and extend deeply into the underlying liver tissue. Blackhead diagnosis is made readily on the basis of the lesions.

Lack of therapeutic drugs

Under field conditions the diagnosis is mainly based on post mortem lesions. In the laboratory it is mostly based on the microscopically demonstration of histomonads movement in caecal smear from freshly killed birds. In addition, isolation trials and histopathological examination can be carried out.

The biggest problem related to the control of Blackhead is the lack of therapeutic drugs. Currently in Europe there is only Nifursol as feed additive for prevention of the disease, which might be banned by March 31st 2003. Good management is the only effective method of preventing this disease since many of the effective drugs used in past years are no longer available commercially and /or by legislation's. Drugs that reduce the presence of caecal worms, and thus reduce the infection rate, are available but do not have an effect on the *Histomonas* organism. Also, antibiotics and anticoccidials have little or no influence on the course of histomoniasis in chickens, or on the interaction of histomonads with coccidia. New interventions toward diagnosis, therapy and control are required. ■

(Note: This article has been derived from a presentation given by Prof Dr HM Hafez at the annual Alltech symposium "Nutritional Biotechnology in the Feed Industry" held early May in Lexington, KY, USA)



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