# Infectious Diseases and Metabolic Syndromes Impacting Broiler Breeders

The most prevalent infectious diseases and metabolic syndromes of poultry, which can negatively impact the welfare of the birds and cause economic losses to producers, are continuing to evolve worldwide. Having good management of feeding and nutrition, as well as biosecurity and vaccination programs in place can help prevent disorders and eradicate or limit the spread of diseases. It is also important to consider the role that migratory birds, rodents, pests and the global movement of people, among other factors, play in regulating these conditions. Quickly recognizing the signs and symptoms of these disorders is essential for corrective actions and preventions to be achieved.

## INFECTIOUS DISEASES

**Mycoplasma synoviae (MS)** has persisted despite improved approaches to controlling and monitoring the disease. In addition, MS often presents as a silent infection, where broiler breeder flocks may seroconvert (test positive serologically) with no sign of illness or negative impact on performance, making it difficult to assess.

MS has re-emerged due to a decrease in antibiotic (AB) use and the occurrence of more pathogenic strains. These strains can cause the typical synovitis issues (swollen joints and footpads), secondary respiratory issues (especially in broilers) and a fairly new effect on eggs called Eggshell Apex Abnormalities (EAA) or Top Coning **(Figure 1)**. EAA more often affects commercial layers but has also been seen in broiler breeders.

**Figure 1.** Egg shell abnormalities (EAA) or “top coning” caused by MS infection in hens.

A decrease in AB use to control Mycoplasma infections coincides with more companies starting antibiotic-free programs (ABF), minimizing AB use or the ban of AB (all or specific) in some countries. Reduced AB use has led to more flocks showing seroconversion. Initially, a few live Mycoplasma gallisepticum (MG) vaccines were introduced worldwide, which seemed to work well against MG, and were cost-effective. Many producers used these live MG vaccines and subsequently stopped using continuous AB in feed to control MS. As a result, MS seemed to re-emerge and thrive. More recently, live MS vaccines have been introduced, and some broiler companies have started using these in conjunction with MG vaccines in their broiler breeders.

As companies become more conscious about biosecurity and more affordable testing becomes available, more MS infections are detected. Good biosecurity practices and management of MS-free breeding stock must be active decisions in the process to eradicate this disease.

**Coccidiosis** is most commonly seen due to improper management of birds after vaccination with live coccidiosis vaccines. Coccidiosis control starts with vaccination at the hatchery (most common) or on farm. The vaccine must be properly handled, never frozen and applied correctly, ensuring that when applied in the hatchery, that chicks are well covered, and the oocysts do not settle out of solution. Even with the best vaccine application, the farm must have the correct environmental conditions for proper sporulation of oocysts and vaccine cycling within the birds and the house. Adequate cycling of oocysts does not occur if the litter is too dry or too wet, if the bird density is too high or too low or if anticoccidial medications are used. If one or more of these occur, birds can either have too much of a reaction causing coccidiosis issues early (usually 14-30 days of age) or, if not exposed to enough vaccine and recycling in the litter, the result is typically a coccidiosis outbreak later in life (6-20 weeks of age). Coccidiosis outbreaks may also occur due to severe immunosuppression from a simultaneous or recent infection such as Marek's Disease Virus (MDV) or Chicken Anemia Virus (CAV). Further information regarding coccidiosis prevention can be found in the Aviagen Briefs - *Coccidiosis Control in Broilers with the use of Vaccines or Coccidiosis Control in Broiler Breeders with the use of Vaccines.*

**Viscerotrophic Velogenic Newcastle Disease (VVND)** is a form of Exotic Newcastle Disease (END) and is a respiratory virus recognized in many regions. In areas where it is widespread, birds must be vaccinated to protect against morbidity, mortality and egg production drops. If unvaccinated birds are exposed, the result can be similar to highly pathogenic avian influenza (HPAI), resulting in morbidity/mortality of 90-100%.

Vaccination programs for VVND are a combination of live and killed vaccines to induce very high protective titers. Poor quality vaccines, poor vaccination technique or insufficient vaccination can result in antibody titers not being fully protective against morbidity, mortality and egg drops. If birds are not fully protected, it is common to see minor egg production drops (5-15%) and a slight increase in mortality (0.5-1.0%). Symptoms can present as:

- **Torticollis (wry neck or twisted neck, especially during rear).**
- **Petechial hemorrhages in the trachea, proventriculus, intestines, cecal tonsils and sometimes the brain.**
- **A sudden increase in Newcastle disease virus (NDV) titers.**

Egg quality problems such as thin shells and white color (more noticeable in brown eggs) can also be seen with an accompanying drop in egg production. Good biosecurity is key to keeping this disease out of the flock, along with a good vaccination program to give fully protective titers.

**Histomoniasis ("Blackhead")** is primarily reported in the US but has recently been reported throughout the EU, Asia, and Latin America. In many regions, the removal of all preventative medications and effective treatments has caused an increase in the incidence of Blackhead.

The presence of dirt floors and/or re-used litter makes it more difficult to completely clean and disinfect a house between flocks and control secondary carriers, such as cecal worms **(Figure 2)** and earthworms, known to harbor the histomonas protozoan organism. There is good evidence that darkling beetles can also carry the organism, and it has been theorized that direct transmission between birds is possible. Therefore, the best way to prevent or control Blackhead is by effective cleaning and disinfection. In instances where dirt floors and/or re-used litter are present, floors should, at a minimum, be treated with one of the following on top of the floor before spreading litter material:

- **Iodine and acid with salt (such as sodium hypochlorite).**
- **A combination of salt and lime.**
- **Organic acid and iodine.**

In addition, crates used to transport birds must be cleaned, as birds have been shown to contract histomoniasis from equipment.


**Figure 2.** Cecal worms (Heterakis gallinarum) passed in feces.

Blackhead has been observed as early as 13 days of age in poultry. Therefore, frequent and early treatment for internal parasites is very effective. Treatment entails medicating as many as 4-5 times before production begins and using more than one anti-parasitic medication to prevent resistance. Treating for more than one day may also have benefits; all birds might not get an effective dose with a one-day treatment. Further information regarding histomoniasis prevention can be found in the *Aviagen Brief - Histomoniasis (Blackhead).*

**Inclusion Body Hepatitis (IBH)** is mostly reported in broilers and is often transmitted horizontally but can be vertically transmitted from parent stock (PS) and sometimes grandparents (GPs) to PS. Typically, young hens become infected during production and vertically shed the virus to their progeny for several weeks. IBH is caused by several serotypes of Fowl Adenovirus Group 1 (FADV1).

Historically, adenoviruses are present in most chicken houses, and most broiler breeder flocks naturally seroconvert to FADV1 before the onset of production. The issue occurs when pullets are placed in a new or very clean rearing house and are not exposed to FADV1 before the onset of production, leaving them without immunity when exposed to the virus in the laying house. For this reason, it is also called "new house" or "clean house" syndrome. In recent years, the risk of pullets seroconverting to FADV1 has decreased due to better biosecurity and cleaning and disinfection practices to control diseases such as Avian Influenza (AI) and avian salmonellosis.

Broiler breeders, not naturally seroconverted or vaccinated in rear but exposed during lay, usually do not show any signs of disease. Still, their progeny is at greater risk of IBH from vertical transmission for 4-6 weeks after the PS flock is exposed. If this becomes a chronic issue, birds must be vaccinated with a killed vaccine containing the specific serotypes seen in the flock.

**Avian Influenza (H9N2)** is caused by avian influenza virus serotype H9N2 and sometimes H9N3. It is seen throughout Asia, the Middle East and Europe (EU). The virus causes a mild respiratory infection with a slight increase in mortality (0.5-1.0% a week for a few weeks) in broiler breeders. If complications occur with other respiratory diseases such as NDV, IBV, MG/MS and/or bad environmental conditions (too cold or too hot, high ammonia), symptoms could worsen.

A severe egg production drop that never fully recovers to pre-infected levels is seen in unvaccinated birds. Typically, the egg production drop is approximately 30-50% over several days and takes 2-3 weeks to recover. The hens' reproductive tract is also affected, leading to eggshell quality issues such as soft-shelled eggs. If H9N2 becomes prevalent in a region, birds must be vaccinated with a killed vaccine, if an approved vaccine is available. The best protection is achieved using the local area's strains in the vaccine.

**Infectious Bronchitis Virus (IBV)** could be the most common disease seen in broiler breeders and broilers. Most companies vaccinate for IBV because minor egg drops (usually 5-15%) and cull eggs, along with respiratory signs, are likely to occur without proper protection.

There are many IBV variants seen around the world. If there is evidence of IBV variant strains in a specific area, vaccinations (usually live primers) for these variants should be included, if available, in the vaccination program to increase the spectrum of protection. If there is very early exposure in the first 2-3 weeks to a severe variant (like QX) without proper vaccination, it could lead to non-laying chickens ("blind layers" or "false layers"), caused by early damage to the oviduct.

## METABOLIC SYNDROMES AND DISEASES

Many metabolic syndromes have the following predisposing factors related to the transfer of birds from the rear to the laying phase:

- **Poor flock body-weight uniformity.**
- **Lighter than recommended body weight.**
- **Earlier than recommended transfer to the lay house.**

After photostimulation, diet formulas are often changed and feed volumes increased. As a result, the predisposing factors listed above can adversely affect bird metabolism and sexual development. Called an "overfeeding complex", it is associated with various metabolic problems that can occur individually or in conjunction, leading to lower livability of the flock and poor live production performance.

**Multiple Follicular Hierarchies** result when birds are overfed, causing changes in the ovary and resulting in the overproduction of follicles.

A typical follicular hierarchy in control-fed breeding hens consists of the recruitment and development of no more than eight follicles. This phenomenon is known as multiple follicular hierarchies **(Figure 3)**. These multiple follicular hierarchies can result in superovulation and the alteration of egg production. According to scientific research, for every extra follicle at sexual maturity, the bird produces ten fewer eggs during its productive life.

**Figure 3.** Multiple follicular hierarchies with the ovary.

As a result of superovulation, erratic oviposition and defective egg syndrome (EODES) can occur **(Figure 4)**. EODES can progress further to oviduct impaction **(Figure 5)**, setting the stage for abdominal or internal laying and salpingitis-peritonitis **(Figure 6)**.

**Figure 4.** Too many follicles as a result of superovulation (EODES).

**Figure 5.** Salpingitis with caseous egg yolk from the oviduct.

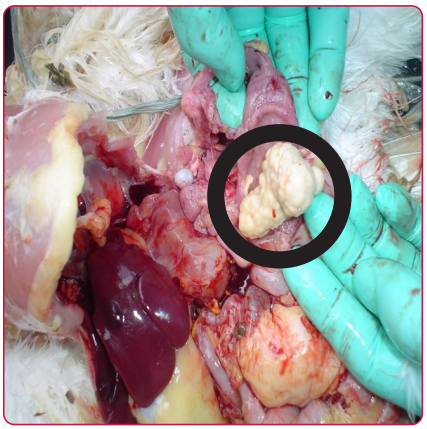

**Figure 6.** Salpingitis and impacted oviduct.

**Peritonitis** is the predominant reproductive disease causing mortality in broiler breeders. It is an acute disease with a sudden increase in mortality, especially at the beginning of egg production, and may persist until after peak. The main clinical signs are mortality, particularly in the morning hours, of hens in good condition without clinical signs of any disease. At necropsy, purulent (pus-filled) material around the ovaries and the abdominal cavity is seen. Due to internal/abdominal laying, it is sometimes referred to as Egg Yolk Peritonitis **(Figure 7)**.

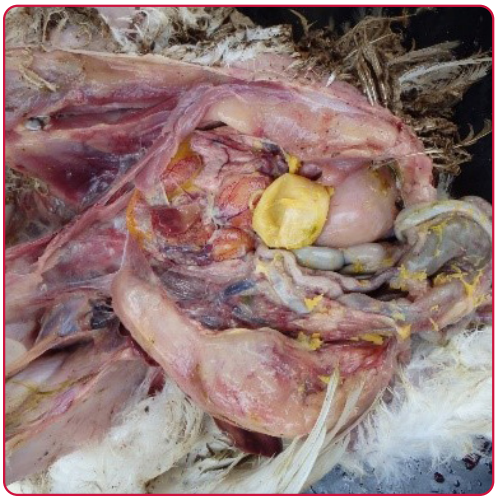

**Figure 7.** Egg yolk peritonitis, egg yolk is found around organs upon necropsy.

Peritonitis is multifactorial and caused by:

- **Poor water quality.**
- **Primary infections (E. coli, Cholera, Salmonella).**
- **Secondary infections due to immunosuppressive and respiratory diseases.**
- **Poor house/bird management.**
- **Multiple Follicular Hierarchy or EODES.**

In general, peritonitis can be prevented by:

- **Managing litter and nest conditions.**
- **Vaccinating with a live or killed E. coli vaccine.**

To prevent overfeeding and the development of peritonitis, it is critical to control body-weight gain and uniformity during rear.

**Prolapse** is commonly observed at the onset of production in flocks with poor body-weight uniformity. Prolapse and subsequent peck-out can result in cannibalism and occurs more often in Spring/Summer due to the excessive light stimulus. Feed increases that are too large after photostimulation and before peak production are associated with an earlier than desired start of production and higher rates of double-yolk eggs that can also cause cloacal prolapse. Therefore, small but frequent feed increases to peak feeding are recommended after photostimulation. Prolapse and peck-out have also been observed in flocks 40 to 50 weeks of age with excessive weight **(Figure 8)**. In this case, abdominal fat may alter the correct return of the cloacal mucosa after laying.

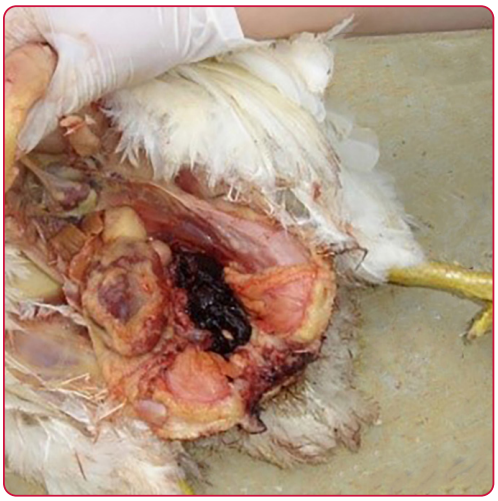

**Figure 8.** A peck-out that resulted in the extraction of internal organs. An internal blood clot is often observed upon necropsy.

**Calcium Tetany** or hypocalcemia (low blood calcium [Ca]) occurs in broiler breeders that have not started egg production and are fed high Ca levels (>1.2%). The high Ca levels trigger a metabolic mechanism (negative feedback) that limits optimal storage and Ca transport from the bone for eggshell formation. It typically occurs acutely with symptoms and clinical signs early in the morning, at the start of the day. Birds gasp and open their wings when hot, show weakness and depression, progressing towards paralysis and extension of the legs backward; sometimes, seizures can occur. As leg paralysis progresses and hens squat down in the scratching area, over-mating may occur, potentially leading to mortality. At necropsy, lesions are non-specific, often associated with active ovaries (multiple follicular hierarchies) and the presence of partially or fully formed eggs in the oviduct with follicular congestion **(Figure 9)**.

**Figure 9.** Calcium tetany - presence of partially or fully formed eggs in the oviduct with lung and follicular congestion.

Calcium tetany occurs in young broiler breeder hens between 25 and 34 weeks of age, especially flocks with poor uniformity, which have been photostimulated too soon and have been given a production diet with high Ca levels. Although less common, calcium tetany can also occur when production diets are formulated to contain lower than recommended Ca levels.

Hypocalcemia can be treated with Ca supplementation. Ca can be increased in the feed or by manual distribution of oyster shell. It is important to avoid Ca overdoses, as that would cause toxicity and lead to increased mortality. Prevent calcium tetany by correctly balancing the minerals in the diet (calcium to phosphorus ratio, [Ca:P]) and adjusting age and diet changes. It is also critical to maintain the correct body weight and uniformity during rear.

**Fatty Liver and Hemorrhaging Syndrome (FLHS)** is a syndrome that occurs mainly in commercial laying hens. However, cases are occasionally seen in broiler breeders. Sudden death from liver rupture has been reported in over-fleshed birds after peak production as they get older, gain weight, and the liver gets fatter. An enlarged, fragile and pale liver is observed at necropsy **(Figure 10)**, accompanied by breast muscle paleness resulting from internal bleeding. The liver can rupture spontaneously or due to trauma as birds enter the nests, perch or experience discomfort.

**Figure 10.** Ruptured and pale liver; blood clotting around the liver indicates rupturing.

**Sudden Death Syndrome (SDS) or Re-feeding Syndrome** is associated with the over-feeding complex mentioned earlier. Affected hens develop heart problems and changes in electrolyte balance (mainly phosphorus [P] and potassium [K]) that cause sudden death. The sudden increase in mortality occurs as the flock approaches 30% in production. It decreases when the percentage of production reaches 60 to 70%, lasting 1 to 2 weeks in mild to moderate cases. Some common clinical signs include:

- **Less flock vocalization.**
- **Very fluid fecal droppings.**
- **Increased feed clean-up times.**
- **Increased morning mortality.**

Necropsy lesions consist of pseudo-prolapse, cardiac hypertrophy, widespread internal congestion and hydropericardium with mild ascites **(Figure 11)**. In addition, birds with SDS commonly have low sexual development and immature ovaries **(Figure 12)**.

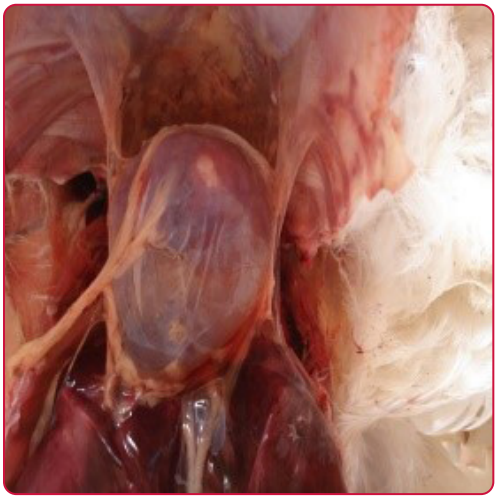

**Figure 11.** Hydropericardium associated with SDS.

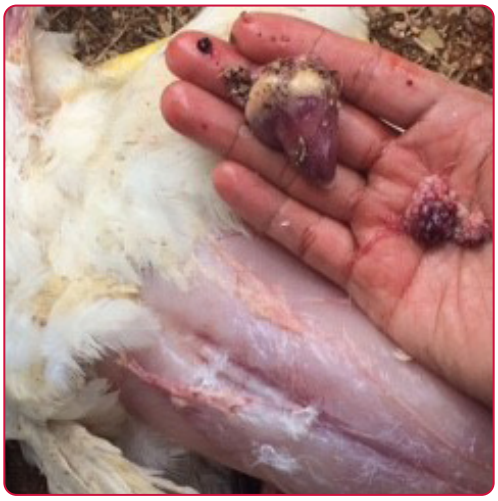

**Figure 12.** Heart with juvenile ovaries (SDS occurs in younger as well as older birds).

Cardiac hypertrophy is defined as a Heart (g)/Body Weight (kg) ratio >3.5. In hypertrophic hearts, there is increased development of the left ventricle and dilation of the right ventricle generating a notch or slit between the ventricles and the atria.

Situations that lead to the development of SDS include:

- **Large percentages of immature hens with low sexual development (small combs and wattles) and poor breast fleshing at transfer.**
- **Rapid and high volume feed increases to achieve early onset and a rapid increase of egg production.**
- **A rapid body-weight gain in conjunction with heart muscle mass gain. Necropsy reveals over-fleshed hens ("plump breast" and "double-breasted hens", (Figure 13)).**
- **Mineral requirements, especially P and K, are altered, and an electrolyte imbalance develops due to rapid body weight and cardiac muscle mass gains.**
- **This deficiency is exacerbated when light birds are fed Breeder 1 diets with higher Ca levels, resulting in heart failure and sudden death.**

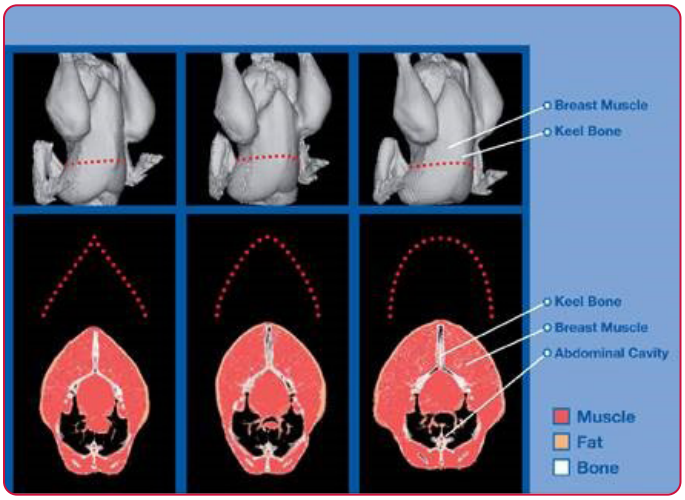


**Figure 13.** Assessing the shape of the breast in females, under, ideal, and over fleshed.

Contributing factors to SDS include:

- **Low protein diets.**
- **Excessive Ca levels.**
- **Low K (hypokalemia occurs).**
- **Low P (hypophosphatemia occurs).**

Correct diagnosis is essential as this syndrome is often confused with calcium tetany. If treated erroneously with Ca supplementation, it can create a Ca:P imbalance and hen mortality.

### PREVENTING METABOLIC SYNDROMES IN BROILER BREEDERS

1. **Improve body-weight uniformity.**
   Body-weight uniformity is critical to feed a population of birds accurately. The more uniform the flock, the greater the likelihood that a higher proportion of birds are subjected to optimal feed management and light stimulation. The more variable the weight between birds, the more variable their requirements, reproductive performance and eggshell quality. It is recommended to set targets and indicators and to propose action plans when deviations occur.
2. **Avoid pre-lighting or anticipated move to the hen house.**
   Consideration must be given to flock age, sexual maturity, uniformity, and light-proof condition of housing when planning bird move into production house and the age at photostimulation.
3. **Avoid overfeeding during critical times after photostimulation.**
   After photostimulation and the resulting increase in circulating sex hormone (estrogen) levels, broiler breeding hens become more sensitive to feed changes. Under this hormonal stimulus, birds convert feed more efficiently, and it is easy to overfeed with increases greater than 3-5 g/week from transfer to the onset of production. After photostimulation, a gradual increase in feed is recommended because the metabolic problems described herein are rooted at this stage.
4. **Adjust feeding program for egg production.**
   At the onset of egg production, a feeding program should consider small feed increases when there is low production to avoid overfeeding those birds that are not yet laying. In modern broiler breeders, it is essential to prevent excessive breast development and modulate weekly hen mortality. Therefore, it is critical to follow up by performing a field necropsy and assessing each of these syndromes' relative incidence to take corrective action at the right time.

## CONCLUSION

Many infectious diseases can be prevented with good biosecurity practices and, when available, vaccination. Metabolic diseases can be prevented by understanding the factors that predispose flocks to these conditions and how to avoid them. As the most prevalent infectious diseases and metabolic syndromes of poultry continue to evolve, good feeding and nutrition management, biosecurity, and vaccination programs must be in place for prevention and eradication to occur.