Gangrenous Dermatitis (GD) is a serious bacterial disease affecting poultry. It generally is fatal, often within 24 hours. It sometimes seems to appear almost out of nowhere because suddenly you will have healthy birds and dead birds, but not many birds in between. While GD has been reported in commercial layers, turkeys, and broiler breeders, it usually occurs in fast-growing broilers between 4 and 8 weeks of age.

In recent years, there has been an increase of this disease in commercial broiler operations across the U.S., and GD now results in significant economic losses to the poultry industry. The decision by many integrators to move away from antibiotic use in poultry feed because of consumer pressure may contribute to an increase in the number of cases of GD. Economic losses are substantial because much of the mortality occurs late in the growout, just before harvesting the flock. Death losses that range from 50 to 400 birds per house per day are not uncommon. The disease is known by several other names, including necrotic dermatitis, avian malignant edema, gangrenous cellulitis, “blue wing,” “red leg,” and “wing rot.”

What Causes Gangrenous Dermatitis?

A wound to the skin is usually the initial culprit that sets a bird up for GD; followed by a secondary bacterial infection with *Clostridium prefringens* type A, *Clostridium septicum*, or *Staphylococcus aureus* (either alone or in combination). Bacteria involved in the occurrence of GD are usually not able to penetrate intact skin. However, the infectious agent can be ingested if live birds peck at dead birds that have died with the disease or if the litter and feces are contaminated with large numbers of disease-causing bacteria. Milder forms of GD are usually associated with *Staphylococcus aureus*. When *Clostridium* is the cause, GD cases are generally much more severe, with higher mortality occurring much more rapidly. Clark et al. (2008) indicates that for GD to occur and affect large numbers of birds, generally three things are required:

1. Some type of injury to the skin,
2. The disease organism (*Clostridium* or other species) present in sufficient numbers to cause disease, and
3. Some type of immune suppression

Even on the best-managed farms, there is no shortage of skin injuries. Chicken toenails are responsible for many cuts and scratches that broilers receive during the growout period. However, growers should constantly monitor their houses and eliminate everything possible that could be a potential injury risk (ladders left in the house, exposed nails at bird level, sharp edges, etc.).

Both clostridia and staphylococci are ubiquitous (present everywhere) in the poultry house environment and in the intestine and on the skin of the birds. However, presence alone of the organisms does not automatically result in a disease challenge. Other contributing factors are thought to play a role in development of clinical disease within a flock. Because *Clostridia* are extremely hardy and capable of surviving very harsh environmental conditions for long periods of time, it is unlikely that they can ever be totally eliminated from a poultry house environment. *Clostridia* are spore-forming bacteria often
found in soil, but they may be found in feed, feces, dust, and numerous other places. When spore-forming bacteria detect that the environment is becoming unfavorable, they will form a spore that helps them survive difficult times. The spore is resistant to desiccation, almost all disinfectants, heat, and cold.

Birds with a strong, or even competent, immune system generally do not seem to be affected by GD. Therefore, it is often believed that other diseases that trigger immunosuppressive effects such as infectious bursal disease, inclusion body hepatitis, reovirus, reticuloendotheliosis, Marek’s disease, and chick infectious anemia may set a flock up for GD. In addition, mycotoxins (particularly aflatoxin) in the feed can cause immune suppression, as can birds subjected to stressful situations such as overcrowding, wet litter, or heat stress. Other stressors such as environmental extremes, coccidiosis, nutritional deficiencies, and management issues may also suppress the immune system and lead to GD. Often, farms that have had GD before will experience repeat outbreaks unless corrective action is taken. A complete clean-out and disinfection of the house is usually the best course of action to reduce spore counts after an outbreak. However, a complete clean-out may not totally eliminate the problem on farms with a history of GD.

In addition, a complete clean-out is not always possible. Short downtime between flocks, availability and cost of new bedding material, and weather conditions may prevent a total clean-out from being an option. As an alternative, some field reports indicate varying degrees of success using litter treatments to produce a dramatic shift downward in pH of the litter, which may reduce microbial growth. Aluminum sulfate, sodium bisulfate, salt, and other treatments have been tried in an attempt to reduce the occurrence of GD. A NOTE OF CAUTION: Growers should not apply any product to their litter or pad without first consulting their service technician and having a green light from their integrator that the product in question is safe to use. Furthermore, growers should be aware of all consequences associated with any product they apply and how that product may affect the litter for future use.

Another alternative to a total clean-out is to windrow litter between flocks in an effort to reduce the pathogen load in the litter. When properly done, in-house windrowing can eliminate almost all of the pathogens present in the litter, including *Clostridium* (Macklin et al., 2007). Windrowing takes advantage of primarily heat to kill microorganisms, although high ammonia levels and competitive exclusion (“good” bacteria overwhelming “bad” bacteria) may also play a role in reducing pathogen numbers.

Many pathogens grow most effectively at a temperature range near the body temperature of their host; in the case of chickens, this is around 105–107°F. However, if the temperature can be raised a few degrees, the pathogen’s growth can be inhibited. If the temperature can be increased by 20–30°F, most microorganisms can be killed. The target temperature to shoot for inside windrows is 130–135°F. This is hot enough to kill most pathogens. It is thought that the litter temperature increase associated with windrow composting is sudden enough that it may catch *Clostridium* unprepared to form a spore in time to save itself. However, litter moisture is critical to getting the windrow to heat. If the litter is too dry, the windrow will not heat. If litter moisture is less than 30 percent, the windrow will have a difficult time achieving a temperature of 130°F, and bacterial growth will only be inhibited until conditions improve, but the bacteria will not be killed. Too much litter moisture can have the same effect of not allowing the windrow to heat. Litter moisture greater than 60 percent may prevent the windrow from heating because the litter is too wet.

**What to Look For**

Gangrenous dermatitis has characteristic lesions that help identify it if you know what to look for. It usually begins with small pimples on the skin, but it soon progresses to involve much larger areas. Look for small spots on the top of the wings that look like raw sores or bloody spots.
In the early stages, these will be small, perhaps the size of a dime or nickel. Unfortunately, there is usually much more of the bird affected than just the wing. However, the top of the wing is easy to see as you walk through the flock, and you will be able to pick up on the fact that something isn’t right when you see this wing damage. If you are fairly perceptive, you may notice this even before the mortality starts to spike. If you miss the wing damage, you certainly won’t miss the rapid and severe increase in mortality as the disease begins to spread through the flock.

Birds with the disease do not show clinical signs for long because they die very rapidly, generally within 24 hours. Lesions on the skin are dark reddish to purple to green. These are most often on the abdomen, breast, wings, and/or legs. Gas or gelatinous fluid may accumulate under the skin. The bird’s skin may have a “spongy” feel to it because of increasing numbers of bacteria producing gas between the muscle and skin. Muscle tissue is also affected and may appear as raw, moist areas where the skin has deteriorated. Dead birds decompose extremely rapidly. You may pick up birds this morning and go back in a couple of hours and find more dead birds that look like they have been dead for several days instead of only a couple of hours.

If you have an outbreak of GD on your farm, you MUST pick up dead birds several times each day. Live birds pecking dead birds can contract the disease, and live birds having the disease (even though they won’t live long) can contaminate healthy birds via feces, feeding equipment, etc. Generally, a fairly accurate diagnosis can be made by the rapid and acute increase in mortality and the characteristic gross lesions. If you ever see a case of GD, you won’t forget it. So, should you ever have it a second time (which often does happen), you will recognize it immediately. It can be tracked from house to house, so take precautions and practice sound biosecurity when working your houses. If you have an outbreak in one house, work that house last to help prevent tracking it to your other houses.

Management factors are critical to preventing and controlling a GD outbreak. Reports of outbreaks seem to be greater in summer and fall than in winter and spring. Daily mortality collection is critical throughout the year but especially during hot weather. This is easier said than done, but it is very important: do your best not to miss a single dead bird from one day to the next. Collection of ALL mortality at least once daily is critical to disease prevention. Poor farm management practices, unsanitary conditions, and stressful environmental conditions (wet litter and high humidity) may predispose flocks to GD. Chicken house pads with a soil pH > 6 may also be at a higher risk of dermatitis infection. Wet litter (> 60 percent moisture) may also be a risk factor for GD. High humidity in the house during the summer and fall seasons when cool cells are operating much of the time may increase the moisture content of the litter and lead to a greater risk of GD.

Growers should closely monitor feed inventory. Do not let your birds run out of feed. Contact your service technician or the feed mill if you run low on feed. Bird activity increases when hungry chickens receive feed, resulting in an increased number of cuts, scratches, and skin damage. Also, be sure that migration fences are in place on time to prevent overcrowding in some areas of the house that may increase the GD risk. Avoid loud noises that may disturb birds and increase likelihood of cuts, scratches, and skin damage. Follow a lighting program that helps calm the birds and control activity level. Remember that anything you can do from a management standpoint that reduces stress for the birds and/or reduces the possibility of a skin injury will reduce the risk of a GD outbreak.
Summary

Gangrenous dermatitis is a serious health concern for many broiler operations. The fact that it has a somewhat come-and-go nature can make it difficult to determine all the factors involved in its occurrence. Prevention should be high on every grower’s to-do list because managing an outbreak is labor-intensive, time-consuming, and expensive. Because *Clostridium* and other disease organisms that cause GD are everywhere in the environment, it is unlikely they can be totally eliminated. Therefore, the goal should be to keep their numbers as low as possible. By doing so, fewer birds will be exposed to disease-causing pathogens, and the ones that are exposed will not be exposed to overwhelming numbers of pathogens, decreasing their chances of becoming infected. **Growers should focus on farm hygiene, sound management practices, and ideal litter and house environment conditions** to lessen the GD threat.

References
