

The ABC's of Airsacculitis

Steven Clark, DVM, Dipl. ACPV¹
Greg Hansen²

¹Alpharma Animal Pharmaceuticals, Fort Lee, New Jersey, 07024 USA. (ph. 201-218-5818) steven.clark@alpharma.com

²Poultry Intellimetrics Inc., 53190 532nd Ave, Paynesville, Minnesota 56362 USA. (ph. 320-276-8374)
ghansen@lkdlk.net

Airsacculitis has plagued turkey production for years. In the 1st edition of the Land O Lakes Turkey Producers Handbook (circa 1976) airsacculitis was defined as “a term used to describe a bird having exudates in its air sacs. Airsacculitis is a symptom, not a specific disease. Airsacculitis may result from *E.coli*, *Mycoplasma*, aspergillosis, avian influenza and cholera.” For treatment “get an accurate diagnosis of specific cause of airsacculitis, then follow recommended treatment for that disease.” Airsacculitis is a cost to the producer, with mortality, sub-optimal performance (morbidity), treatments (therapeutics) and excess condemnations.

Poultry Intellimetrics, Inc. is a US (United States) consulting firm that provides processing, live-haul and growout evaluations for the turkey industry. One of the many parameters that Poultry Intellimetrics measures is air sac salvage, condemnations and on-line rates. “Air sac salvage” refers to those carcasses processed off-line due to severe airsacculitis, usually involving the interclavicular air sacs and extending into the shoulder joints and wings. During the salvage process 52% of the eviscerated carcass can be lost. Parts are removed and saved (salvaged) but the shell along with some breast meat is condemned due to airsacculitis. This results in a loss to both the processor and the grower. “Air sac condemn” refers to those carcasses condemned by the USDA (US Department of Agriculture) inspector due to extensive airsacculitis and are not suitable for salvage. “On-line air sac” is a term used to describe carcasses with mild airsacculitis that the plant employees can clean (process) without removal from the processing line. On-line procedures include removal by vacuuming of kidneys and airsacculitis exudate from the body cavity. This procedure is reserved for carcasses that are mildly affected and not showing systemic lesions. Poultry Intellimetrics’ “heavy tom” category refers to those birds that weigh over 26 pounds. Prior to 1993 all toms were “heavy toms.” Not all plants that provide salvage and condemnation data can provide the on-line air sac numbers.

Processing plant data for air sac can be an indication of field conditions for those flocks several weeks prior to slaughter. Airsacculitis averages peak in the late winter and early fall, indicative of the cold/heat-stress and poor ventilation. On-line rates for airsacculitis have a seasonal trend, peaking in the fall season (August 2000 was 8.7% and September 2001 was 13.2%), and are higher than salvage and condemnation rates. In 2001 condemnations due to airsacculitis peaked in February and September at 0.13% and 0.14%, respectively, with a low in June of 0.02%. Air sac salvage follows a similar seasonal trend but is slightly higher; salvage for February, May, September 2001 was 0.76%, 0.49% and 0.1.17%, respectively. Processing plants have developed USDA approved procedures for salvaging wholesome meat and condemning the contaminated portions, from carcasses with airsacculitis. Over the past 12 years condemnations have decreased as on-line processing and off-line salvage has increased (Table). This might indicate improved salvage procedures and/or an increase in the incidence of airsacculitis.

Table. US industry airsacculitis averages (%) in heavy toms turkeys (1990 – 2001) for off-line salvage, condemnations and on-line reprocessing.

<i>Year</i>	<i>Air Sac Salvage (%)</i>	<i>Air Sac Condemn (%)</i>	<i>On-Line Air Sac (%)</i>
1990	0.19		
1991	0.37		
1992	0.31		
1993	0.40		
1994	0.47	0.18	
1995	0.60	0.16	
1996	0.61	0.16	
1997	0.60	0.14	
1998	0.69	0.10	3.47
1999	0.78	0.10	3.36
2000	0.77	0.07	4.43
2001	0.64	0.07	6.01

A for ANATOMY

An understanding of the turkey's **anatomy** provides insights into the causes of airsacculitis and how to control it. The respiratory system of birds is "the most efficient gas exchange system among all air-breathing vertebrates" (King & McLelland, 1984). But this sufficient system is also uniquely susceptible to respiratory disease, such as, airsacculitis.

As the bird inspires air, it initially enters through the nostrils to be warmed (thermoregulation) and filtered by the nasal septum and conchae. The nasal cavity is also important for smell (olfaction). From the nasal cavity the air proceeds down the larynx, trachea and syrinx. The trachea is a cartilage tube lined with epithelium and cilia. The cilia are hair-like projections critical to the bird's defense against dust and infectious agents. The trachea carries air to and from the lungs through the bronchus, bronchi, parabronchi and air capillaries. From the lungs air moves to the air sacs. Oxygen and carbon dioxide are exchanged in the lungs as the air passes over the blood capillaries. The unique anatomy provides continuous unidirectional airflow through the lung and a crosscurrent relationship between the blood and air capillaries.

There are actually seven air sacs in the turkey connecting to the lungs either directly via the bronchus or indirectly via several parabronchi. There is also some penetration of the air sacs into the bones of the breast, wing, neck and hip (sternum, humerus, cervical, ribs, pelvis, thoracic, coracoid and synsacrum). The seven air sacs include the interclavicular (1), cervical (2), cranial thoracic (2) and abdominal (2) air sacs. Normal air sacs are thin, glistening and transparent. The thoracic (rib)-cage moves in bellows-like action to compress the sacs, moving air between the lungs and air sacs.

The respiratory defenses of the bird are designed to remove particles and bacteria. Mucous secretions and active cilia (mucociliary transport) remove particles (dust, bacteria, spores) from the trachea and bronchi. Cilia are the microscopic hair-like projections that cover the inside of the trachea. Viruses, many bacteria, fungal spores and dust particles are small enough to reach the air sacs. Both Ficken and Sander (AAAP, 1994) referenced that large particles (>10 microns) are collected in the nasal cavity, those between 5 – 10 microns are retained in the upper respiratory tract, while smaller particles (<1 micron) may get inspired throughout the lungs and air sacs. Particles from 1 – 5 microns represent 90% of the dust in a poultry house (Sander, 1994). Macrophages (cellular defense mechanisms) carried by the blood and in secretions, enter the lungs, kill and remove bacteria. The air sacs have a minimal blood supply and therefore almost

non-existent defense mechanisms against bacteria and dust. When birds are open-mouth breathing, the first line of defense (nasal cavity) is bypassed and the trachea (mucociliary transport) can be overwhelmed with dust carried directly to the air sacs. Open-mouth breathing is observed during hot weather or anytime barn condition get too hot, as well as with Aspergillosis (“gasps”, “honkers”).

The respiratory system is designed to (1) carry oxygen to the lungs and blood, (2) carry waste gases, such as carbon dioxide, out of the body, and (3) dissipate heat from the body through evaporation. With any respiratory disease or insult these functions are compromised. The diseased flock is not able to handle heat stress effectively and it is more susceptible to systemic disease, such as Colibacillosis and Cholera. Growth rate is impaired in the affected birds and the immune system may be compromised.

B for BASICS (Bad Air, Bad Bugs, Bad Air Sacs)

Respiratory disease, including airsacculitis, can be divided into either “infectious” or “noninfectious” causes. Noninfectious causes are related to the farm environment, such as, air quality, ventilation, litter conditions, ammonia levels and excessive dust. Infectious causes may be subdivided into bacterial, viral, mycoplasmal or fungal etiologies.

Microorganisms (**bad bugs**) are infectious causes of airsacculitis. During the late 1940s (Hinshaw *et. al.*) “Air Sac Infection” was also known as “Infectious Sinusitis”. At that time the causative agent was suspected to “be a virus or virus-like organism”, later identified as *Mycoplasma gallisepticum* (MG). Causes of turkey bacterial respiratory disease are Cholera (*Pasteurella multocida*), Colibacillosis (*E. coli*), Coryza (*Bordetella avium*), Psittacosis (*Chlamydia psittaci*), ORT (*Ornithobacterium rhinotracheale*), or Duck Cholera (*Pasteurella anatipestifer*). Viral causes are Newcastle Disease (NDV), Avian Metapneumovirus (APV) or Avian Influenza (AI). MG (*Mycoplasma gallisepticum*) is the most common cause of mycoplasmal respiratory disease, followed by MS (*Mycoplasma synoviae*) and MM (*Mycoplasma meleagridis*). Aspergillosis (*Aspergillus fumigatus*) is a common fungal respiratory disease. To get an accurate diagnosis, submit appropriate samples for serology, culture and PCR (for mycoplasmas, NDV and APV).

Barnes (1994) addressed the pathogenesis of respiratory disease, stating that the severity of airsacculitis and whether the bird succumbs to disease is dependent upon (1) the pathogenicity and virulence of the organism, (2) the degree of exposure and (3) the susceptibility of the bird. Once the organism gains entry into the bird, colonizes, multiplies, spreads and evades the host defense (immune) system, it causes an infection. To survive, the bird contains the infection, repairs the damage and sequesters the damaged tissue. Or, the bird dies. Airsacculitis is evidence of the bird attempting to repair and wall-off (sequester) the damaged tissues (air sacs).

Ammonia and dust are two major contributors to **bad air** in a poultry house and subsequently to airsacculitis. Ammonia is (unfortunately) a common irritant present in poultry houses caused by the natural degradation of poultry feces and is affected by temperature, litter pH, moisture, aeration (oxygen) and fecal content. Ammonia is the most significant factor associated with respiratory disease and is correlated with [airsacculitis] condemnation rates at processing in turkeys (Redig). Exposure to ammonia as low as 10 ppm and as short as a few hours damages the cilia and causes excessive mucous production (exudate); 10 - 40 ppm reduce the clearance of *E. coli* from the respiratory system. Over 100 ppm of ammonia can cause blindness in poultry. The lowest level most humans detect by smell is 5 ppm, but usually farmers are de-sensitized to ammonia odor and may not smell harmful levels. Ammonia is absorbed and held in the wet litter, trapped by caking or a fresh litter covering. Tilling releases this ammonia and increases production by adding oxygen.

Dust contains broken feather barbules, skin debris, feed particles, litter particles and microorganisms. As dust levels increase in a house, air sac lesions increase and so do airborne levels of pathogens. Viruses, bacteria, fungi and endotoxins actually attach to the dust particles.

Dust plus ammonia have a synergistic effect (Sander, 1994); ammonia increases the negative effects of dust. Inhalation of fecal-contaminated dust is thought to be the cause of *E. coli* respiratory infections. Outbreaks of Colisepticemia occur soon after high levels of *E. coli* are present in poultry house air (Barnes, 1994).

Ammonia, Newcastle disease, Coryza and dust damage the (defense) mucociliary transport. Ammonia stops and destroys the cilia of the trachea. *Bordetella avium* attaches to the cilia and destroys it. Once the cilia are destroyed, it takes 7 to 14 days for the cilia to re-grow after the insult is stopped. Damaged lungs and air sacs take longer to heal. Newcastle disease virus (NDV) kills the cilia. Excess dust slows the action of the cilia.

When **bad air sacs** are observed upon necropsy, we often ask, "How long did this take?" or "When did this start?" Experimental infections show that airsacculitis can occur relatively quickly -- within 24 hours. A damaged respiratory system allows *E. coli* to enter the blood circulatory system directly through the air sacs. Pourbakhsh (1997) researched chickens inoculated with *E. coli* into one thoracic air sac. Within 3 – 24 hours, *E. coli* could be cultured from the trachea, lungs, air sacs and liver, therefore demonstrating that the respiratory infection became systemic.

- Within 3-hours the chickens were depressed and had some head shaking. The air sacs were slightly thickened.
- By 6-hours blood vessels begin to vascularize the infected air sacs and mild pus (fibrinopurulent exudates) were observed.
- By 12-hours, the airsacculitis was moderate to severe and had spread to the other (non-inoculated) air sacs. Mild respiratory sounds were observed.
- Within 24-hours severe airsacculitis with white-heart, pneumonia, enlarged spleen and hemorrhages (moderate pericarditis, marked pulmonary congestion, severe splenomegaly and presence of multiple petechial hemorrhages throughout subcutaneous tissues, visceral fat tissues and serous surfaces) was observed.
- At 48-hours post-inoculation, severe lesions persisted and the liver became enlarged and fibrin covered (mild to moderate perihepatitis). Chickens were either sick (depressed) or dead.

C for CONTROL

Controlling airsacculitis involves addressing three areas, (1) disease management, (2) litter management, and (3) air quality. Veterinary health programs designed to control disease will detail diagnosis (serology, culture, etc.) vaccinations, medications for treatments and prevention, and biosecurity.

To control infectious causes of airsacculitis, procedures can be taken at the breeder and hatchery level, as well as at the commercial farm. The National Poultry Improvement Plan (NPIP) is designed to test breeders to eliminate MG, thus eliminating vertical transmission of MG to the poults. The NPIP has active control programs for *Salmonella pullorum*, *Salmonella gallinarum*, *Salmonella enteritidis*, *Mycoplasma gallisepticum* (MG), *Mycoplasma synoviae* (MS), and *Mycoplasma meleagridis* (MM). The most common respiratory disease associated with the hatchery is brooder pneumonia caused by *Aspergillus fumigatus* or *Staphylococcus aureus*. Proper hatchery sanitation programs are essential in controlling brooder pneumonia, although some cases are attributed to contaminated brooder house conditions. Aspergillosis in the brooder is typically not associated with Aspergillosis airsacculitis in market-age flocks.

At the brooder house, from 0 to 6 weeks of age, emphasis should be placed on controlling ammonia, Coryza (*Bordetella avium*) and Colibacillosis. Good ammonia control requires the best litter and ventilation management. In the grower and finisher barns, from 6-weeks to market age, critical points are to control dust, ammonia and infectious diseases. Temperatures above 45 to

55°F in older tom turkeys cause the birds to pant, resulting in airsacculitis. Panting allows the irritating dust particles to flow directly into the air sacs where the body quickly produces exudate in an attempt to remove it.

Water sanitation is critical to minimizing the spread of diseases. Optimum sanitation is active chlorine at 3 - 6 ppm, 750 - 800 ORP (oxidative reduction potential; contact Hanna Instruments at 877-694-2662 to purchase the ORP Tester) and a pH 6.5. Conduct a water analysis to assess water quality. Gas chlorine systems are an option for effective chlorination (contact Regal at 800-327-9761). Perform a water analysis to evaluate water quality (contact your local health department or the Arkansas Water Resources Center, 501-575-7317).

Research-related Control Tips:

1. Give **vitamin D**. Vitamin D₃ supplementation resulted in lower mortality, turkey osteomyelitis complex (TOC), incidence of green livers, isolation of bacteria and lower airsacculitis scores (Huff, 2000).
2. Evaluate whether **vaccination** is necessary. Respiratory viruses may stimulate, but some inhibit the defense function of the respiratory system. Attenuated, modified live virus vaccines (such as, NDV) can also inhibit the defense system. (Toth, 2000)
3. Reduce **stocking density**. In chickens, flock size was positively associated and layout time was negatively associated with early respiratory disease complex, including airsacculitis (Tablante, 1999).
4. Control **aspergillosis**. In turkeys, research demonstrates that turkeys do not develop immunity (confer protection) against re-challenge, but may actually increase susceptibility (Kunkle, 1998).
5. Control **ORT**. ORT (aerosol administration) causes growth retardation, airsacculitis and pneumonia (van Empel, 1996). Turkeys inoculated with a live or killed (experimental) vaccine had less airsacculitis and pneumonia (Sprenger, 2000).
6. **Lower temperatures, dust and ammonia** in the barn. High dust and ammonia levels were measured in the warm environment (70°F versus 45°F), along with increased levels of aspergillus and airsacculitis in 20-week old turkeys (Noll, 1991). Ammonia concentrations above 20 ppm and dust concentrations greater than 5 mg/m³ caused lung damage (Redig; Noll, 1998).
7. Both *E. coli* (Pourbakhsh, 1997) and *P. multocida* (Ficken, 1989) can enter the blood (systemic circulation) directly through damaged air sacs.
8. Use clean **litter**. Dyar (1984) reports of treating aspergillosis contaminated litter with nystatin and copper sulfate. Aspergillosis mortality decreased and litter mold counts decreased (2,500,000 to 1,000 organisms/gram of litter).
9. Ideal **humidity** is 50 - 60%. When the relative humidity is below 50%, the respiratory system mucous membranes become dry with subsequent reduction on ciliary activity (Froyman). Dry air also increases dust. But a high humidity increases ammonia.

Collection of Control Tips from the Field:

1. **Iodine** in the drinking water, one day per week, weekly. Mix at a high dose to keep the trachea clean of particles and bacteria. Use continuously at a lower dose as a water sanitizer.
2. Add **salt** to the drinking water, one day per week, as needed to reduce dust. The birds' droppings are wetter and thereby lowering the level of dust in the barn.
3. **Organic iodine (EDDI)** has been used as an expectorant in turkeys. The use of EDDI actually induces a mild toxicity, whereas the bird responds by producing excessive seromucoid nasal excretions and intermittent coughing. Caution: do not use in an acute (early, active) infection, because the birds may actually die (suffocate, choke) on their mucous. This is typically used as a one-day-a-week preventative program.
4. To minimize **heat stress** during the summer install fans in the turkey finisher buildings. Place so fans blow directly on the birds, recommend one (1) 36-inch fan per 1,200 sq ft. This recommendation is based on field reports from the midwest and southeast (personal data). One includes a comparison of two companies; the company experiencing less

respiratory disease had twice the number of fans. Other highlights: (1) use coarse sprinklers to control dust, (2) till only when adequate ventilation is available, (3) use litter treatments to control ammonia, (4) cooler temperature to reduce panting and (5) use Clinafarm to treat litter.

5. Use litter treatments to control ammonia. Poultry litter acidifiers include **Poultry Guard**[™] (Oil-Dri Corp. of America, Chicago, IL; sulfuric acid), **PLT**[®] (Jones-Hamilton Co., Wallbridge, OH; sodium bisulfate), and **AI⁺Clear**[™] (General Chemical Inc., Parsippany, NJ; alum, aluminum sulfate). Poultry Guard, is a granular, acidified clay and requires no moisture to activate and no need for incorporation. Products such as Poultry Guard and PLT are labeled for ammonia control at 50 pounds of product per 1,000 square feet of barn floor.
6. Add **fresh litter** (shavings, straw, oat hulls, etc.) to minimize ammonia. Old litter supplemented with fresh shavings produces 300 times less ammonia per hour than old litter alone (Bennett, 2001).
7. **Tilling** the litter increases ammonia, as ammonia trapped in the moist litter is released and the exposure to oxygen produce more ammonia. Consider not tilling or tilling more frequently so as to avoid releasing large quantities of ammonia at once (Bennett, 2001).
8. In Europe (only) **Clinafarm**[®] is approved for use to spray on litter (Schering-Plough Animal Health, Union, NJ). Clinafarm is a brand of Imazalil (Enilconazole). It is highly effective against *Aspergillus* spp. organisms and spores. It is compatible with quaternary ammonium compounds, glutaraldehyde and phenolic compounds and be may be applied by fogger or sprayer. The European label reads as a spray for treatment of aspergillosis or for mold disinfection.
9. Spray the litter with **magnesium chloride** to reduce dust. This product is commercially available as Dust-Off[®] (Cargill Salt Co., Minneapolis, MN; 30% magnesium chloride) for use as a dust control agent on unimproved roads (Karunakaran, 1991). Apply between flocks then till the litter.
10. **Thiabendazole** lowers litter mold counts. Thiabendazole (TBZ) was applied before litter was added and after the litter was added. The litter was treated again at 6 and 12 weeks.
11. Spray the litter with **copper sulfate** to control aspergillus.
12. Disinfect shavings with **calcium propionate**. To control aspergillosis, spray fresh shavings with a solution of sodium or calcium propionate (Kumar, 1987). Calcium propionate is a mold inhibitor similar to TBZ.
13. Disinfect built-up litter with **formalin**. To control aspergillosis, remove the cake, spray built-up litter twice with a dilution of formalin (Kumar, 1987).
14. Control ammonia by reducing excessive **litter moisture**. Clean up and minimize water spills and wet-spots under the drinkers.
15. A lower barn **temperature** also lowers ammonia production.
16. Use **sprinklers** in the barn to control dust. Controlled fogging/spraying with a resultant litter moisture of 30 - 35% litter moisture.
17. Dusty conditions can be improved by reducing the **temperature** in the building, which increases the relative humidity and increases the weight of the dust particles so they settle out faster. It cannot be improved by increasing the ventilation rate alone, as the ventilation is increased to remove dust, the relative humidity would reduced and more dust rises into the air. (NTF, 2000)
18. **Chlortetracycline** (Aureomycin[®], Alparma, Fort Lee, NJ) has an approved label claim in turkeys at 200 grams per ton of feed, for the control infectious synovitis caused by *Mycoplasma synoviae* susceptible to chlortetracycline. Feed continuously for 7 - 14 days. In addition, for chickens it has approval at 200 - 400 grams per ton of feed for control of chronic respiratory disease (CRD) caused by MG and *E. coli*. Both **oxytetracycline** and **tetracycline** (Oxytet[®] and Tet-Sol[™], respectively, Alparma, Fort Lee, NJ) water solubles have claims for MS and CRD (read the label as approved claims can vary by brand and for species).

Control Tip Summary:

1. Biosecurity.
2. Insure NPIP certified poults.

3. Water sanitation must be constant and consistent.
4. Get an accurate diagnosis (serology, cultures, PCR).
5. Lower ammonia levels.
6. Lower dust levels.
7. Lower barn temperatures.
8. Judiciously use medications.
9. Prevent disease.

Disclaimer: this paper discusses unapproved uses. It should not be interpreted as promotion of those uses. Always read and follow label instructions. Always follow State and Local regulations.