Reportable Diseases in Poultry

Emphasizing HPAI & vND

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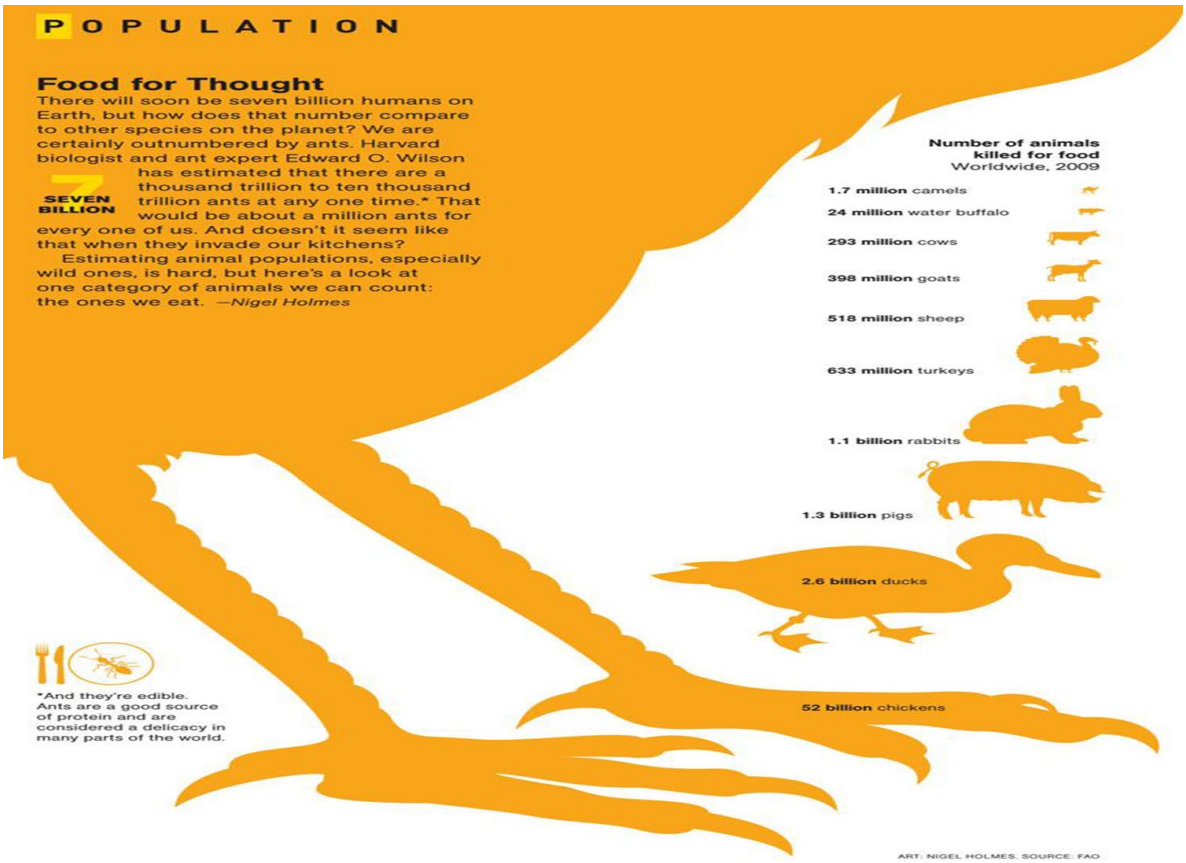
There has been a tendency to for VS epidemiologists to neglect the Reportable Disease List for Poultry as opposed to the lists for Cattle (FMD, etc.), swine (ASF, etc.), and equine (AHS, etc.).Our mission is to perpetuate the productivity and marketability of all components of Animal Agriculture, INCLUDING poultry. As you know, the immediate diagnosis of a disease is the most important step in the control and eradication of a foreign animal, or regulatory, a FAD incursion.

It is important to recall that the last several foreign animal disease (FAD) incursions into the United States have affected poultry; and, the most costly and severe (in terms of the size of the geographical area, and number of flocks, affected) was the 2014-15 H5N2 HPAI outbreak. Accordingly, an immediate diagnosis of a suspicious disease into poultry should be a TOP PRIORITY. For that reason, the USDA's Reportable Disease List for Poultry is very important.

The importance of the poultry industry (as compared with other components of Animal Agriculture) in the United States is demonstrated by the following data. The **number** of poultry and ducks processed annually dwarfs all other components of Animal Agriculture:



(as per Dr. Alberto Torres, Cobb-Vantress Export Veterinarian)

The poultry industry (broiler and turkey) processed 9.241 billion animals in 2019 as compared with 0.1554

billion of the other segments of Animal Agriculture (or 98.3% versus 1.7%). These data points clearly demonstrate the importance of poultry as a protein source that must be perpetuated.

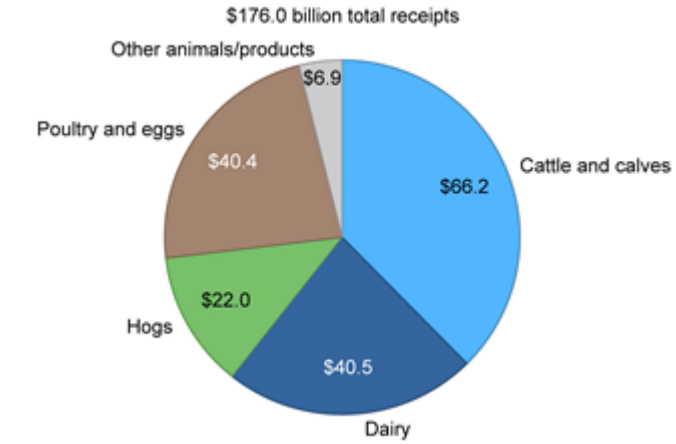
Feeding 326 Million Americans

The meat and poultry industry is the largest segment of U.S. agriculture. U.S. meat production totaled 52 billion pounds in 2017 and U.S. poultry production totaled 48 billion pounds in 2017.

(as per North American Meat Institute)

The poultry industry (broiler and turkey) processed 9.241 billion animals in 2017 as compered with 0.1554 billion of the other segments of animal agriculture (or 98.3% versus 1.7%). (as per North American Meat Institute)

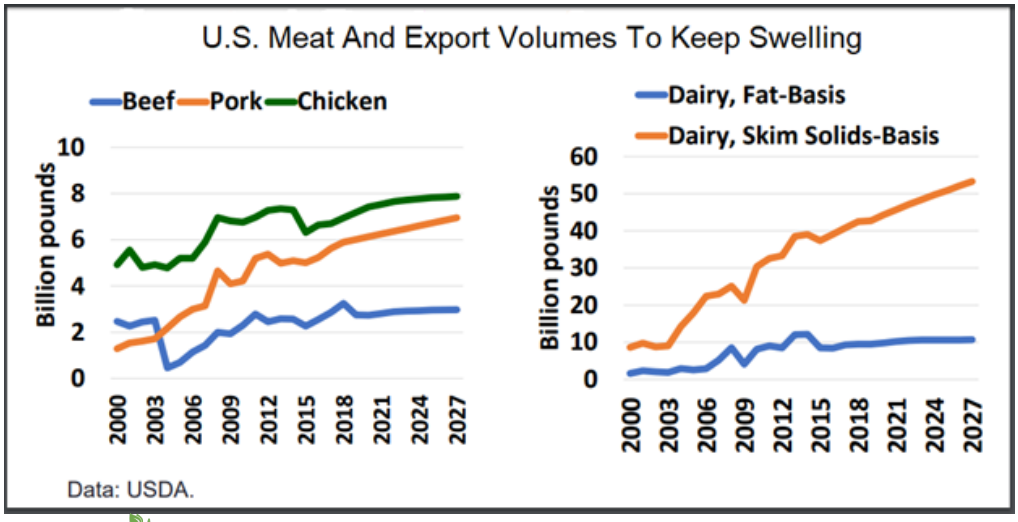
2019 animal and animal product cash receipts ($ billion)



Note: Components may not sum to total because of rounding. Data as of September 2, 2020.
Source: USDA, Economic Research Service, Farm Income and Wealth Statistics.

Cash receipts for animals and animal products totaled $176.0 billion in 2019. Cattle/calf receipts accounted for 37.6% of that total, while dairy receipts accounted for 23%, and poultry and eggs receipts accounted for 22.9%.

To a large extent, U.S. agricultural exports are a story of producing a significant amount of protein for the rest of the world. For more than two decades, the U.S. pork, beef and poultry industries have been expanding exports rapidly so that 11 percent of U.S.-produced beef is now sold abroad, as is 16% of chicken meat, 10% of turkey, 19% of dairy products, and 22% of pork.



(as per Agri Pulse)

Therefore, the health and vitality of the poultry industry is extremely important as a protein source for the American consumer, as well as a revenue source in Animal Agriculture.

The Reportable Disease List for the USDA, APHIS

Avian Diseases

Disease	Status	Date of Last Occurrence / Notes
Avian chlamydiosis	Present	Sporadic (wild birds, pet birds, backyard)/limited distribution/no commercial production flock detections reported in 2020
Avian infectious bronchitis	Present	
Avian infectious laryngotracheitis	Present	Sporadic (primarily vaccine-related)/limited distribution
Avian mycoplasmosis (*M. gallisepticum*)	Present	Sporadic/limited distribution/all commercial poultry breeding flocks are under a surveillance program to confirm infection-free status. Commercial table-egg laying may be vaccinated.
Avian mycoplasmosis *(M. synoviae*)	Present	Sporadic/limited distribution/all commercial poultry breeding flocks are under a surveillance program to confirm infection-free status.
Duck viral hepatitis	Free	1998
Fowl cholera (*Pasteurella multocida*)	Present	
Fowl typhoid (*Salmonella gallinarum*)	Free	1981
Highly pathogenic avian influenza	Free	2020: H7N3 in South Carolina commercial turkey flock and identification of H5N2 in a wild mallard duck.
Low pathogenic avian influenza (poultry); H5 and H7 are notifiable.	Identification of the presence of infection/infestation	Disclosures of many strains (not H5 or H7) in migratory are sporadic. Identified sporadically in backyard poultry and in live-bird-markets that serve local ethnic communities. Low pathogenicity H5 was detected in a live-bird market and low pathogenicity H5N2 was detected in backyard poultry. No commercial production flock infection was detected from 7/2017-12/2017
Infectious bursal disease (Gumboro disease)	Present	Sporadic/limited distribution

Marek's disease	Present	
Newcastle disease (Neurotropic and viscerotropic strains)	Free	2018-20, California
Pullorum disease (*Salmonella pullorum*)	Present	Sporadic/limited/distribution Identified sporadically in backyard poultry. No commercial production flock detections since 1991, considered absent in them.
Turkey rhinotracheitis	Present	Sporadic/limited distribution Type C is detected in poultry sporadically and with limited distribution.

A comprehensive review of the poultry diseases on the USDA/APHIS Avian Reportable Diseases List follows:

Avian Chlamydiosis

Text adapted from "Chlamydiosis in Birds" brochure, provided by the Association of Avian Veterinarians (AAV). Please contact AAV for copies at: AAV Publications Office Phone: 817-428-7900, Fax: 817-485-4800, Email: aavpublication@aav.org

Chlamydiosis, formerly called ornithosis, and most commonly known to physicians as psittacosis, or (lay terminology) parrot fever, is a common disease of many bird species and is caused by the organism *Chlamydophila Psittaci*. Owners should be fully informed of the implications for their pet birds and the potential for transmission to humans. This disease most often is disclosed in a pet shop environment.

TRANSMISSION
Transmission of the disease between birds is primarily through inhalation of contaminated fecal or feather dust. Risk of infection is increased by close contact with infected birds that are shedding the organism. For this reason, birds that are stressed through shipping, overcrowding, reproductive activity, or malnutrition have a greater tendency to shed the organism. Infected birds may shed the organism even if no clinical signs of disease are observed.

CLINICAL SIGNS
The most common visible clinical signs of avian chlamydiosis involve the respiratory or gastrointestinal systems of birds. Lime-green diarrhea is a common clinical manifestation and is associated with liver disease caused by the *C. psittaci* infection. Some birds may show general signs of illness: lack of appetite, weight loss, depression, diarrhea, discharge from the eyes or nares, or even death. The same signs are not unique to chlamydiosis and may represent a number of other diseases. Some birds that are actively infected with *C. psittaci* may be mildly affected or show no signs of illness. Immunosuppression in most cases results in a more severe clinical disease. Breeding birds can pass the organism to their young. Baby birds are more susceptible to severe infection than adult birds and may die in the nest or soon after weaning.

DIAGNOSIS
A confirmed diagnosis of chlamydiosis in a live bird is sometimes difficult and depends on the species of bird, length of time since exposure, and general condition of the bird. The most commonly used diagnostic tests include the polymerase chain reaction (PCR) assay, serology, and culture of the organism.

TREATMENT

If chlamydiosis has been diagnosed, or if treatment has been recommended by your veterinarian, all exposed birds in the household should be treated at the same time to reduce the spread or recurrence of the disease. It is imperative that infected birds be isolated during treatment and that certain sanitary measures be employed to prevent spread or reinfection of the disease. The success of treatment depends on all of the medication being given in the recommended dosage and time frame. Antibiotic dosage and treatment should be directed by your veterinarian to ensure the appropriate course of therapy is undertaken and followed. There are several ways to administer medication to the birds: mouth (oral), injection, mixture of the antibiotic in soft foods or drinking water, or through commercially available medicated pellets. Depending on the condition of the patient, other supportive treatment may also be recommended. Your veterinarian will discuss the most appropriate treatment for your bird. Antibiotic treatment must be continued for a minimum of 45 days to be effective. During treatment the owner must:

→clean the premises with an appropriate disinfectant such as a bleach & water (1:32 dilution), 1% Lysol®, or quaternary ammonia compounds;

→use caution when handling droppings and cage debris, take care not to aerosolize dust while cleaning, keep dust and feather circulation to a minimum to avoid potential exposure to humans or other birds;

→separate/isolate and seek medical care for other birds showing signs of disease;

the elderly, pregnant, sick or very young children and people that are immunosuppressed or on anti-rejection drugs should avoid bird contact;

→remove all mineral supplements containing calcium as calcium interferes with the effectiveness of the recommended medication;

→reduce stress in the bird's environment as much as possible.

TRANSMISSION TO HUMANS

Avian chlamydiosis is transmissible from birds to humans, although the incidence of transmission is rare considering the high incidence of infection in birds. If anyone in the household with an infected bird develops persistent flu-like symptoms, respiratory distress, fever, chills, headache, weakness, or fatigue, that person should seek the advice of a physician as soon as possible and tell the physician of any recent history of bird exposure. Treatment is simple and most often successful in humans, but neglect of the symptoms or delayed diagnosis may result in serious illness, especially in people that are immunocompromised or those with other underlying medical conditions. *Chlamydophila Psittaci* is not the same organism that causes sexually transmitted chlamydia infection in humans.

PREVENTIVE MEASURES

The following recommendations help reduce the incidence of chlamydiosis in flocks or companion birds:

→immediately after purchase, take all newly-acquired birds to an avian veterinarian for chlamydiosis screening tests;

→buy birds from suppliers who routinely screen their birds for the presence of *C. Psittaci* or who guarantee the health of their birds in some manner;

→isolate and quarantine all newly acquired birds for a minimum of six weeks;

→maintain appropriate preventive health management as recommended by your avian veterinarian.

[as extrapolated from "Chlamydiosis in Birds" brochure, provided by the Association of Avian Veterinarians (AAV).]

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Avian Laryngotracheitis

*By **Maricarmen Garcia** PhD, University of Georgia*

Infectious laryngotracheitis (ILT) is an acute, highly contagious, **herpesvirus** infection of chickens and pheasants characterized by severe dyspnea, coughing, and rales. It can also be a subacute disease with nasal and ocular discharge, tracheitis, conjunctivitis, and mild rales.

ETIOLOGY

The disease is caused by *Gallid herpesvirus I*, commonly known as infectious laryngotracheitis virus (ILTV). It has been reported from most areas of the USA in which poultry are intensively reared, as well as from many other countries.

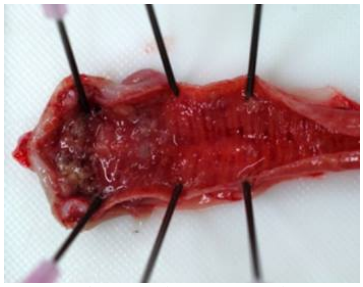
CLINICAL SIGNS

→In the acute form of infectious laryngotracheitis virus, gasping, coughing bloody mucoid exudate, rattling, and extension of the neck during inspiration are seen 5–12 days after natural exposure.

→Broiler chickens coughing bloody exudate and gasping for air, characteristic of severe forms of the disease.
→Moderate conjunctivitis due to infectious laryngotracheitis virus in a broiler.

Courtesy of Dr. Maricarmen Garcia.

→Reduced productivity is a varying factor in laying flocks.
→Affected birds are anorectic and inactive.
→Mortality varies but may reach 50% in adults and is usually due to occlusion of the trachea by hemorrhage or exudate.
→Signs usually subside after ~2 weeks, although some birds may show signs for longer periods. Strains of low virulence produce little or no mortality, with mild respiratory signs and a slight decrease in egg production.
→The most prominent lesion is that is hemorrhagic tracheitis:



Courtesy of Dr. Maricarmen Garcia.

→After recovery, birds remain carriers for life and become a source of infection for susceptible birds. The latent virus can be reactivated under stressful conditions. Infection also may be spread mechanically. Several epidemics have been traced to the transport of infected birds or contaminated equipment and litter.

DIAGNOSIS
PCR and histopathology of trachea and conjunctiva

VACCINATION:
Three types of ILT vaccines are available:
1. **Tissue culture origin (TCO)** vaccines have a relatively low level of infectiousness and are administered by an eye drop. A disadvantage of TCO vaccines is that the level of immunity is limited; the advantage of this is that it causes a less severe reaction and the birds do not shed the virus.

2. **Recombinant ILT** vaccines have been developed. These recombinant ILT vaccines can be administered as a single dose, by subcutaneous injection, on day 1 or in older bird. It is reported to provide ILT immunity for up to 60 weeks. The recombinant vaccine does not cause shedding of the virus; therefore unvaccinated birds are not placed at risk. It has also limited level of immunity and birds may develop mild clinical signs of ILT. These vaccines are also more expensive.

3.**Chicken embryo origin (CEO)** vaccines can be administered through an eye drop or mass vaccination, such as spray or water. While these CEO vaccines result in a better immunity, **it can cause severe clinical signs and disease due to the increased level of infectiousness. Also, chickens treated with CEO vaccine can become carriers of the virus, putting unvaccinated flocks at risk.** In most states chick embryo origin vaccine is only for sale and use by permit only issued by the State Veterinarian. (as per Dr. Lyndon Badcoe, Washington State University)

CONTROL
In endemic areas and on farms where a specific diagnosis is made, infectious laryngotracheitis virus is controlled by implementation of biosecurity measures and vaccination. Vaccination is done with live attenuated vaccines and viral vector recombinant vaccines. Live vaccines originated from virulent isolates that were attenuated by consecutive passages in embryos or tissue culture.

(as per Merck Veterinary Manual)

Avian Mycoplasmosis (M. Gallisepticum)

M Gallisepticum infection is commonly termed "**chronic respiratory disease (CRD)"** in chickens and as **infectious sinusitis** in turkeys. Infection may also be seen in pheasants, chukar partridges, and peafowl. Infection in pigeons, quail, ducks, geese, and psittacine birds can also occur.

M Gallisepticum is the most pathogenic avian mycoplasma; however, strains may differ markedly in virulence. Primary isolation is made in enriched broth medium containing 10-15% serum, then plated on agar. Typical colonies are identified by immunofluorescence.

CLINICAL SIGNS
In chickens, infection may be inapparent or result in varying degrees of respiratory distress, with slight to marked rales, difficulty breathing, coughing, and/or sneezing. Morbidity is high and mortality low in uncomplicated cases. Nasal discharge

and frothiness about the eyes may be present.

In turkeys, the disease is generally more severe than in chickens, and swelling of the paranasal sinus is common. Feed efficiency and weight gains are reduced. Broilers and market turkeys may suffer high condemnations at processing due to air sacculitis.

Infectious sinusitis in a turkey due to Mycoplasma Gallisepticum. (Courtesy of Dr. Jean Sander)

TRANSMISSION
M Gallisepticum is egg transmitted (transovarian), but the infection rate in breeder hens is low, and some hatches of progeny may be free of infection. Horizontal transmission is primarily via the respiratory tract, with direct and indirect routes.

In the USA, most breeder flocks are free of *M Gallisepticum* , and outbreaks are due to lateral transmission from infected chickens; however, in some parts of the world, egg transmission is a major source of infection. The incidence of egg transmission is highly variable, ranging up to 30-40% during the first 2 months after infection of susceptible birds in production. The transmission rate then lessens and is inconsistent (0-5%) until the end of production.

Birds infected before the onset of production transmit through the egg at a much lower rate, if at all. The infection may be dormant in the infected chick for days to months, but when the flock is stressed, aerosol transmission occurs rapidly and infection spreads through the flock. In addition, the infection may be carried by personnel (especially from an infected to a clean flock), fomites, or introduction of infected birds. In many flocks, the source of infection cannot be determined.

VACCINATION
Live virus vaccination, natural virus infection, cold weather, or crowding may initiate the spread.

DIAGNOSIS
→If by serology, ELISA or hemagglutination inhibition.
→If from oropharyngeal swab samples, PCR.

TREATMENT
M. Gallisepticum infection responds to many types of antibiotics (macrolides, tetracyclines, fluoroquinalones) but not penicillins (for those act on cell walls, which Mycoplasma organisms lack). Antibiotics may alleviate the clinical signs and lesions, but not the infection. When those birds are stressed, the clinical signs may return. For this reason, in many states, affected flocks remain under quarantine or are depopulated to eliminate the infection.

CONTROL
The National Poultry Improvement Plan coordinates control and serology-based surveillance program for *M. Gallisepticum*. These programs have resulted in eradication of the infection in most primary breeder flocks of chickens and turkeys in the USA. Chicks and poults should be obtained from *M Synoviae*–free breeders and raised with biosecurity to prevent introduction.

In backyard/non-commercial flocks in some areas of the U.S., *M. Gallisepticum* infection is of high incidence.

PREVENTION
→The implementation of strict biosecurity measures.
→If purchase birds from sources of unknown health status, hold the birds way from the index flock for 30 days (and observe for the appearance of clinical signs) before introducing them into the home flock.
(as per Merck Veterinary Manual)

Mycoplasma Synoviae

(Infectious synovitis)
Extrapolated from information by David H. Ley, DVM, PhD, Professor, Department of Population Health and Pathobiology, College of Veterinary Medicine, North Carolina State University

M. Synoviae was first recognized as an acute to chronic infection of chickens and turkeys that produced an exudative tendinitis and synovitis (infectious synovitis); it now occurs most commonly as a subclinical infection of the upper respiratory tract, especially in multi-age layer flocks.

M. Synoviae infection is also a complication of air sacculitis in association with Newcastle disease or infectious bronchitis. It is distributed worldwide and is seen primarily in chickens and turkeys, but ducks, geese, guinea fowl, parrots, pheasants, and quail may also be susceptible. *M Synoviae* isolates vary widely in virulence, and suspected virulence factors include adhesins, sialidase, nitric oxide, cell invasion, and antigenic variation and immune evasion.

TRANSMISSION
M Synoviae is egg transmitted (transovarian), but the infection rate in breeder hens is low, and some hatches of progeny may be free of infection. Horizontal transmission is similar to that of *M Gallisepticum,* primarily via the respiratory tract, with direct and indirect routes.

The incidence of *M Synoviae* infection in commercial poultry in the USA has decreased because of the National Poultry Improvement Plan control programs implemented for chicken and turkey breeders.

The incidence in backyard/non-commercial flocks is endemic in some areas.

CLINCAL SIGNS
Although slight rales may be present in birds with *M. Synoviae* respiratory infection, usually no signs are noticed. Birds under stress or with concurrent infections are more likely to be clinically affected. The first signs of infectious synovitis include pale-bluish head parts and lameness in many birds with a tendency to sit.

The more severely affected birds are depressed and found resting around feeders and waterers. Hocks and footpads are swollen, and sternal bursitis (breast blisters) may be seen.

(*Courtesy of American Association of Avian Pathologists)*

Morbidity is usually low to moderate with mortality of 1%–10%. Effects on egg production are usually not apparent, but instances of transient egg production drops have occurred in layer flocks.

DIAGNOSIS
Skeletal abnormalities and trauma must be eliminated as the cause of lameness. Differential diagnoses include viral tenosynovitis as well as staphylococcal and other bacterial joint infections.

A presumptive diagnosis based on clinical signs and gross lesions should be confirmed by laboratory tests. Serum plate agglutination or ELISA are used to detect *M synoviae* antibodies, but cross-reactions with *M Gallisepticum* and other nonspecific reactions may occur. These reactors are confirmed as seropositive by hemagglutination-inhibition or by culture, isolation, and identification of the organism. PCR may be used to rapidly detect *M Synoviae* DNA from pre- or postmortem specimens.

TREATMENT
As with *M. Gallisepticum, M. Synoviae* infection responds to many types of antibiotics (macrolides, tetracyclines, fluoroquinalones) but not penicillins (for those act on cell walls, which Mycoplasma organisms lack). Antibiotics may alleviate the clinical signs and lesions, but not the infection. When those birds are stressed, the clinical signs may return. For this reason, in many states, affected flocks remain under quarantine or are depopulated to eliminate the infection.

CONTROL
The National Poultry Improvement Plan coordinates control and serology-based surveillance programs for *M. Synoviae* similar to those for *M. Gallisepticum*. These programs have resulted in eradication of the infection in most primary breeder flocks of chickens and turkeys in the USA. Chicks and poults should be obtained from *M Synoviae*–free breeders and raised with biosecurity to prevent introduction.

PREVENTION
→The implementation of strict biosecurity measures.
→If purchase birds from sources of unknown health status, hold the birds way from the index flock for 30 days (and observe for the appearance of clinical signs) before introducing them into the home flock.
→A live temperature-sensitive vaccine (MS-H) is commercially available and permitted in some areas
(as per Merck Veterinary Manual)

Duck Viral Hepatitis (DVH)

By Simone T. Stoute , DVM, PhD, DACPV, University of California, Davis

Duck viral hepatitis is an acute and fatal disease in ducklings caused by the Avihepatovirus DHV-1 and DHV-3. It causes opisthotonus and hepatitis. DHV-1 is found worldwide. It causes disease in young ducklings, usually <6 weeks of age and spreads rapidly within a flock. It is the most virulent of the DHV species. The viruses that cause DVH in ducklings should not be confused with duck hepatitis B virus, a hepadnavirus infection of older ducks.

ETIOLOGY
Family: Picornaviridae
Species: Duck Hepatitis Virus I, Ia, II, III
Representative species: DHV-3, DHV-1, DHV-2, DHV-1a

The originally described, most widespread, and most virulent subtype of duck viral hepatitis, traditionally referred to as DVH Type I, has been renamed duck hepatitis A virus type 1 (DHAV-1) and is now classified in the genus *Avihepatovirus* in the Picornaviridae family. Two antigenically distinct genotypes have been identified in Taiwan (DHAV-2) and identified in China and South Korea (DHAV-3).

INCIDENCE IN THE U.S.
Duck hepatitis A virus (DHAV) was first described in young White Pekin Ducks on Long Island, New York (Levine and Hofstad, 1945; Levine and Fabricant, 1950). Since then, DHAV has been reported in duck-raising areas worldwide (Woolcock, 2008), and most recently in China (Guo and Pan, 1984) and Korea (Park, 1985; Woo et al., 2000).

DHV type II (DAstV-1) was originally reported in Norfolk, England (Asplin, 1965). A duck astrovirus with very high sequence similarity to DAstV-1 (Todd et al., 2009) has been detected and sequenced in China (Fu et al., 2009), associated with very high mortality in 1- to 2-week-old commercial ducklings.

DHV type III (DAstV-2) is only known to have occurred in the USA.

(Courtesy of https://www.cabi.org/isc/datasheet/84184)

CLINICAL SIGNS

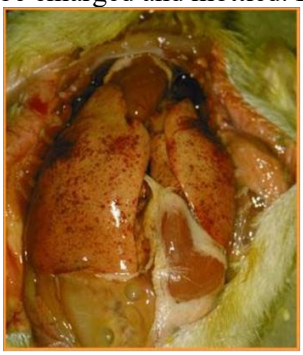
For Duck hepatitis A virus type 1, week-old duckling:
Affected ducklings become lethargic, lose balance, paddle spasmodically, and die within minutes, typically with opisthotonos. Although adults may become infected, clinical signs have not been seen in ducks >7 weeks old. Mortality may be as high as 95% in fully susceptible ducklings. Practically all deaths occur within 1 week after onset of signs.

(Courtesy of The Poultry Site)

The clinical course of DAstV-1 infection is similar to that of DHAV-1 and can be seen in ducklings immune to DHAV-1 infection. DAstV-2 infections are seen in ducklings despite immunity to DHAV-1. The clinical course of DAstV-2 infection is less severe, and mortality is rarely >30%.

LESIONS

The lesions caused by all three types of DVH are similar. The liver is enlarged and covered with hemorrhagic foci up to 1 cm in diameter. The spleen may be enlarged and mottled. Kidneys may be swollen, and renal blood vessels congested.



(Courtesy of Dr. Peter R. Woolcock)

DIAGNOSIS

- Presumptive diagnosis is based on history and lesions;
- Confirmation of diagnosis requires virus isolation or PCR.

TREATMENT

There is no specific treatment.

PREVENTION

Prevention and control is based on strict biosecurity and implementation of vaccination protocols. Strict isolation, particularly during the first 5 weeks of age, is recommended.

Contact with wild waterfowl should be avoided. Rats have been reported as a reservoir host of the virus; therefore, pest control is indicated.

Immunization of breeder ducks with modified-live virus vaccines, using DHAV, DAstV-1, and DAstV-2 , provides parenteral immunity that effectively prevents high losses in young ducklings. The DHAV-1 vaccine is administered SC in

the neck to breeder ducks at 16, 20, and 24 weeks of age and every 12 weeks thereafter throughout the laying period. Three immunizations are advisable for passive protection of ducklings.

An inactivated DHAV-1 vaccine for use in breeder ducks that have been previously primed with live DHAV-1 has been described. A single dose of the inactivated vaccine, given IM before the birds come into lay, provides passive immunity for a complete laying cycle to progeny ducklings.

(as per Merck Veterinary Manual)

Fowl Cholera

*By **Jean E. Sander**, DVM, MAM, DACPV, Zoetis*

ETIOLOGY

Pasteurella Multocida, the causal agent of fowl cholera, is a small, Gram-negative, nonmotile rod with a capsule that may exhibit pleomorphism after repeated subculture. *P. Multocida* is considered a single species although it includes three subspecies: M*ultocida*, *Septica*, and *Gallicida*. Subspecies *Multocida* is the most common cause of disease, but *Septica* and *Gallicida* may also cause cholera-like disease.

In freshly isolated cultures or in tissues, the bacteria have a bipolar appearance when stained with Wright's stain. Although *P. Multocida* may infect a wide variety of animals, strains isolated from non-avian hosts generally do not produce fowl cholera. Strains that cause fowl cholera represent a number of immunotypes (or serotypes). *P. Multocida* can be sub-grouped by capsule serogroup antigens into five capsular types (A, B, C, D, and F) and into 16 somatic serotypes. Turkeys and waterfowl are more susceptible than chickens, older chickens are more susceptible than young ones, and some breeds of chickens are more susceptible than others.

TRANSMISSION

Chronically infected birds and asymptomatic carriers are considered to be major sources of infection. Wild birds may introduce the organism into a poultry flock, but mammals (including rodents, pigs, dogs, and cats) may also carry the infection. However, the role of these as a reservoir has not been thoroughly investigated. Dissemination of *P. Multocida* within a flock and between houses is primarily by excretions from the mouth, nose, and conjunctiva of diseased birds that contaminate their environment. In addition, *P. Multocida* survives long enough to be spread by contaminated crates, feed bags, shoes, and other equipment. The infection does not seem to be egg-transmitted.

CLINICAL SIGNS

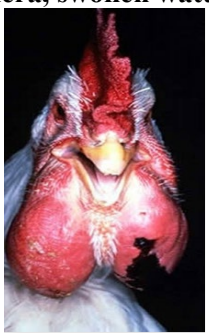
Clinical signs of fowl cholera vary greatly depending on the course of disease.

In acute fowl cholera, finding a large number of dead birds without previous signs is usually the first indication of disease. Mortality often increases rapidly.

In more protracted cases, depression, anorexia, mucoid discharge from the mouth, ruffled feathers, diarrhea, and increased respiratory rate are usually seen. Pneumonia is particularly common in turkeys.

In chronic fowl cholera, signs and lesions are generally related to localized infections of the sternal bursae, wattles, joints, tendon sheaths, and footpads, which often are swollen because of accumulated fibrinosuppurative exudate. There may be lameness, as well as exudative conjunctivitis and pharyngitis. Torticollis may result when the meninges, middle ear, or cranial bones are infected.

Fowl cholera, swollen wattles, broiler



(Courtesy of Dr. Jean Sander)

LESIONS

Lesions observed in peracute and acute forms of the disease are primarily vascular disturbances. These include general passive hyperemia and congestion throughout the carcass, accompanied by enlargement of the liver and spleen. Petechial and ecchymotic hemorrhages are common, particularly in subepicardial and subserosal locations

In chronic forms of fowl cholera, suppurative lesions may be widely distributed, often involving the respiratory tract, the conjunctiva, and adjacent tissues of the head. Caseous arthritis and productive inflammation of the peritoneal cavity and the oviduct are common in chronic infections.

DIAGNOSIS

- Confirmed by bacterial culture. Although the history, signs, and lesions may aid field diagnosis, *P. Multocida* should be isolated, characterized, and identified for confirmation. Primary isolation can be accomplished using media such as blood agar, dextrose starch agar, or trypticase soy agar.
- PCR has been used for the detection of *P. Multocida* in pure and mixed cultures and clinical samples. This method may help identify carrier animals within flocks. However, the specificity and sensitivity of the PCR must be improved. Conventional serotyping suffers from problems with reproducibility and reliability, and the methods are quite laborious.
- Serologic testing can be done by rapid whole blood agglutination, serum plate agglutination, agar diffusion tests, and ELISA. Serology may be used to evaluate vaccine responses but has very limited value for diagnostic purposes.

PREVENTION

- Good management practices, including a high level of biosecurity, are essential to prevention.Rodents, wild birds, pets, and other animals that may be carriers of *P multocida* must be excluded from poultry houses. The organism is susceptible to ordinary disinfectants, sunlight, drying, and heat.
- VACCINATION:
    - Adjuvant bacterins are widely used and generally effective. Because bacterins are only effective in preventing disease caused by the same serotypes included in the vaccine, somatic serotyping is important.
    - Attenuated live vaccines are available for administration in drinking water to turkeys and by wing-web inoculation to chickens. These live vaccines can effectively induce immunity against different serotypes of *P. Multocida*. They are recommended for use in healthy flocks only.

TREATMENT

- Antibiotics may reduce mortality but won't eliminate *P Multocida* from a flock. A number of drugs will lower mortality from fowl cholera; however, deaths may resume when treatment is discontinued, showing that treatment does not eliminate *P. Multocida* from a flock. Eradication of infection requires depopulation and cleaning and disinfection of buildings and equipment. The premise should then be kept free of poultry for a few weeks.
- When antibiotics are used, early treatment and adequate dosages are important. Sensitivity testing often aids in drug selection and is important because of the emergence of multi-resistant strains. Sulfamethazine or sulfadimethoxine in feed or water usually controls mortality.

(as per Merck Veterinary Manual)

Fowl Typhoid (Salmonella Gallinarum)

By Sherrill Davison Yeakel, VMD, MS, MBA, DACPV, Laboratory of Avian Medicine and Pathology, School of Veterinary Medicine, University of Pennsylvania

ETIOLOGY

The causal agent of fowl typhoid is *Salmonella enterica* Gallinarum. The incidence of fowl typhoid is low in the USA, Canada, and some European countries but is much higher in other countries. Although *S enterica* Gallinarum is egg-transmitted and produces lesions in chicks and poults similar to those produced by *S enterica* Pullorum, there is a much greater tendency to spread among growing or mature flocks. Mortality in young birds is similar to that seen in *S enterica* Pullorum infection but may be higher in older birds.

INCIDENCE

The incidence of fowl typhoid is low in the USA, Canada, and some European countries but is much higher in other countries. An eradication program sponsored by the National Poultry Improvement Plan (NPIP) has been instrumental in reducing the incidence.

CLINICAL SIGNS

Fowl typhoid may be acute or chronic.
Clinical signs in chicks and poults include **anorexia**, **diarrhea**, **dehydration**, **weakness** and high **mortality**.

Clinical signs and lesions in young birds are similar to those seen with *S enterica* Pullorum infection.

Older birds may be pale, dehydrated, and have diarrhea.

LESIONS

In older birds may include:

- a swollen, friable, and often bile-stained liver, with or without necrotic foci
- an enlarged spleen and kidneys
- anemia
- enteritis

DIAGNOSIS

- Clinical signs and lesions of fowl typhoid are similar to *S. enterica Pullorum* infection; therefore, diagnosis is confirmed by isolation and identification of the organism.
- Diagnosis should be confirmed by isolation, identification, and serotyping of *S. enterica Gallinarum* (National Poultry Improvement Plan testing procedure).
- The standard serologic tests for Pullorum disease also detect fowl typhoid.

TREATMENT

- Control measures, outlined in the National Poultry Improvement Plan, are based on elimination of the disease. Treatment is never recommended.

PREVENTION:

- There are no federally licensed vaccines in the USA. In other countries, vaccines (killed or modified live) made from a rough strain of *S enterica* Gallinarum (9R) had variable results in controlling mortality.
- More recently, vaccines derived from outer membrane proteins, mutant strains, and a virulence-plasmid-cured derivative of *S enterica* Gallinarum have shown promise in protecting birds against challenge.

(as per Merck Veterinary Manual)

Infectious Bursal Disease (IBD or Gumboro disease or Avian Infectious Nephrosis)

*By **Daral J. Jackwood**, PhD, The Ohio State University*

Infectious bursal disease (IBD) is seen in young domestic chickens worldwide and is caused by infectious bursal disease virus (IBDV). Symptoms of Depending on the IBDV strain and presence of maternal immunity, the disease can also present as a clinical or subclinical disease in young chicks.

IBD is a highly contagious disease of young chickens and turkeys caused by infectious bursal disease virus (IBDV),[1] characterized by immunosuppression and mortality generally at 3 to 6 weeks of age. The disease was first discovered in Gumboro, Delaware in 1962. It is economically important to the poultry industry worldwide due to increased susceptibility to other diseases and negative interference with effective vaccination.

ETIOLOGY
Infectious bursal disease is caused by a Birnavirus (infectious bursal disease virus; IBDV) that is most readily isolated from the bursa of Fabricius but may be isolated from other organs. Two serotypes of IBDV have been identified. The **serotype 1 viruses cause disease in chickens** and, within them, antigenic variation can exist between strains. Antigenic drift is largely responsible for this antigenic variation, but antigenic differences can also occur through genome homologous recombination. **Serotype 2 strains of the virus infect chickens and turkeys but have not caused clinical disease or immunosuppression** in these hosts.

TRANSMISSION
The virus is shed in the feces and transferred from house to house by fomites. It is very stable and difficult to eradicate from premises.

Infectious bursal disease is **highly contagious**; results of infection depend on age and breed of chicken and virulence of the virus.

CLINICAL SIGNS
Infections may be subclinical or clinical. Infections before 3 weeks of age are usually subclinical. **Chickens are most susceptible to clinical disease at 3–6 weeks of age** when immature B cells populate the bursa and maternal immunity has waned, but severe infections have occurred in Leghorn chickens up to 18 weeks of age.

Chickens may exhibit severe **prostration, incoordination, watery diarrhea, soiled vent feathers, vent picking, and inflammation of the cloaca.** Flock morbidity is typically 100%, and mortality can range from 5% to greater than 60% depending on the strain of virus and breed of chicken. Mortality is typically higher in layer breeds compared with broiler chickens. Recovery occurs in <1 week, and broiler weight gain is delayed by 3–5 days. The presence of maternal antibody will modify the clinical course of the disease.

Early subclinical infections are the most important form of the disease because of economic losses. They cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen. The humoral (B cell) immune response is most severely affected; the cell-mediated (T cell) immune response is affected to a lesser extent.

Some strains of IBDV can cause subclinical infections in older birds (3–6 weeks old), which leads to losses from poor feed efficiency and longer times to market. In these cases, the immunosuppression is usually transient, and convalescent birds may recover most or all of their humoral immune function.

LESIONS
The cloacal bursa can become enlarged, with a yellowish colored transudate on the surface. Hemorrhages on the serosal and mucosal services are sometimes observed. Atrophy of the bursa, which includes the loss of B-lymphocytes, occurs approximately 7-10 days after infection. Immunosuppression is directly related to this loss of B-lymphocytes, but immunosuppression and related secondary infections are typically seen in birds that recover from the disease. Severity of the immunosuppression depends on the virulence of the infecting virus and age of the host.

At necropsy, the lesions seen will depend on the strain of IBDV. For strains that cause a clinical disease, the cloacal bursa is swollen, edematous, yellowish, and occasionally hemorrhagic, especially in birds that died of the disease.

Enlarged, hemorrhagic bursa of Fabricius in a chicken infected with very virulent infectious bursal disease virus. This strain of the virus can also cause hemorrhages in skeletal muscles.

Strains of vvIBDV cause similar cloacal bursa lesions, and congestion and hemorrhage of the pectoral and leg muscles can also occur.

Enlarged bursa of Fabricius, infectious bursal disease virus, chicken

(Courtesy of Dr. Daral J. Jackwood)

DIAGNOSIS

- Initial diagnosis of infectious bursal disease is accomplished by the observation of gross lesions in the cloacal bursa. This is followed by microscopic analysis of the bursa for lymphocyte depletion in the follicles.
- Diagnosis can be accomplished by clinical evaluation of the cloacal bursa for macroscopic and microscopic lesions followed by molecular detection of the viral VP2 gene using RT-PCR
- Molecular diagnostic assays are most often used to identify IBDV in diagnostic samples. The reverse-transcriptase-PCR assay is used to identify the viral genome in bursa tissue. Sequence alignments and phylogenetic analysis of the VP2 coding region has been used to further characterize the viruses into genogroups. Samples for molecular diagnostic testing are typically collected after maternal antibodies have waned.

TREATMENT

There is no treatment. Rigorous disinfection of contaminated farms after depopulation has achieved limited success.

PREVENTION/VACCINATION

Live vaccines of chicken embryo or cell-culture origin and of varying low pathogenicity can be administered by eye drop, drinking water, or SC routes at 1–21 days of age. Replication of these vaccines and thus the immune response can be altered by maternal antibody, although the more virulent vaccine strains can override higher levels of maternal antibody.

The immune status of breeder flocks should be monitored periodically with a quantitative serologic test such as virus neutralization or ELISA. If antibody levels decrease, hens should be revaccinated to maintain adequate immunity in the progeny.

The goal of any vaccination program for IBD should be to use vaccines that most closely match the antigenic profile of the field viruses. Diagnostic testing for the genomic sequences of field strains can be used to select the most appropriate vaccination program.

(as per Merck Veterinary Manual)

Marek’s Disease

By **John Dunn, DVM, PhD, Veterinary Medical Officer, USDA-ARS**

Marek's disease is a highly contagious viral neoplastic disease in chickens. It is named after József Marek, a Hungarian veterinarian. Marek's disease is caused by an alphaherpesvirus known as 'Marek's disease virus' (MDV) or *Gallid alphaherpesvirus 2* (GaHV-2).[2] The disease is characterized by the presence of T cell lymphoma as well as infiltration of nerves and organs by lymphocytes.[3] Viruses *related* to MDV appear to be benign and can be used as vaccine strains to

prevent Marek's disease. For example, the related Herpesvirus of Turkeys (HVT), causes no apparent disease in turkeys and continues to be used as a vaccine strain for prevention of Marek's disease (see below). Birds infected with GaHV-2 can be carriers and shedders of the virus for life. Newborn chicks are protected by maternal antibodies for a few weeks. After infection, microscopic lesions are present after one to two weeks, and gross lesions are present after three to four weeks. The virus is spread in dander from feather follicles and transmitted by inhalation.

EIOLOGY.

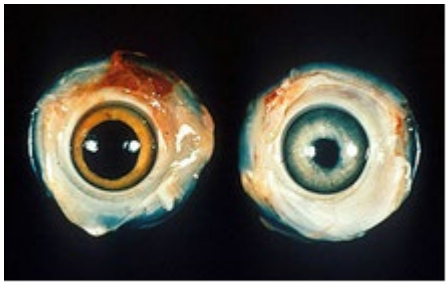
Marek's disease virus is a member of the genus *Mardivirus* within the subfamily Alphaherpesvirinae. Within the genus *Mardivirus* are three closely related species previously designated as three serotypes of Marek's disease virus. Gallid herpesvirus 2 (MDV-1) represents all virulent Marek's disease virus strains and is further divided into pathotypes, designated as mild (m), virulent (v), very virulent (vv), and very virulent plus (vv+). Gallid herpesvirus 3 (MDV-2) and Meleagrid herpesvirus 1 (turkey herpesvirus, MDV-3) represent avirulent virus strains isolated from chickens and turkeys, respectively, and are commonly used as vaccines against Marek's disease.

CLINICAL SIGNS

Six syndromes are known to occur after infection with Marek's disease. These syndromes may overlap.

- Classical Marek's disease or **neurolymphomatosis causes asymmetric paralysis** of one or more limbs. With vagus nerve involvement, difficulty breathing or dilation of the crop may occur.

  Besides lesions in the peripheral nerves, there are frequently lymphomatous infiltration/tumors in the skin, skeletal muscle, visceral organs. Organs that are commonly affected include the ovary, spleen, liver, kidneys, lungs, heart, proventriculus and adrenals.
- Acute Marek's disease is an epidemic in a previously uninfected or unvaccinated flock, causing **depression, paralysis, and mortality** in a large number of birds (up to 80%). The age of onset is much earlier than the classic form; birds are four to eight weeks old when affected. Infiltration into multiple organs/tissue is observed.
- Ocular lymphomatosis causes lymphocyte infiltration of the iris (making the iris turn grey), **unequal size of the pupils, and blindness.**



(Courtesy of Dr. Jean Sander)

- **Cutaneous** Marek's disease causes round, firm lesions at the feather follicles.
- Atherosclerosis is induced in experimentally infected chickens.
- Immunosuppression – Impairment of the T-lymphocytes prevents competent immunological response against pathogenic challenge and the affected birds become more susceptible to disease conditions such as coccidiosis and *Escherichia coli* infection .[6] Furthermore, without stimulation by cell-mediated immunity, the humoral immunity conferred by the B-cell lines from the Bursa of Fabricius also shuts down, thus resulting in birds that are totally immunocompromised.

(as per Fenner FJ, Gibbs EP, Murphy FA, Rott R, Studdert MJ, White DO (1993). *Veterinary Virology (2nd ed.)*. Academic Press, Inc.)

TRANSMISSION

The disease is highly contagious and readily transmitted among chickens. The virus matures into a fully infective, enveloped form in the epithelium of the feather follicle, from which it is released into the environment. It may survive for

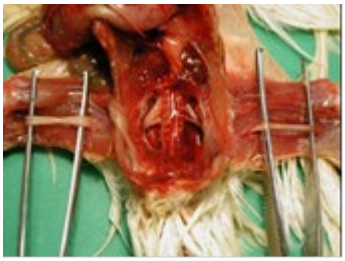
months in poultry house litter or dust. Dust or dander from infected chickens is particularly effective in transmission. Once the virus is introduced into a chicken flock, regardless of vaccination status, infection spreads quickly from bird to bird. Infected chickens continue to be carriers for long periods and act as sources of infectious virus. Shedding of infectious virus can be reduced, but not prevented, by prior vaccination. Unlike virulent strains of Marek's disease virus, which are highly contagious, turkey herpesvirus is not readily transmissible among chickens (although it is easily transmitted among turkeys, its natural host). Attenuated Marek's disease virus strains vary greatly in their transmissibility among chickens; the most highly attenuated are not transmitted. Marek's disease virus is not vertically transmitted.

PATHOGENESIS

Currently, four phases of infection in vivo are recognized: 1) early productive-restrictive virus infection causing primarily degenerative changes, 2) latent infection, 3) a second phase of cytolytic, productive-restrictive infection coincident with permanent immunosuppression, and 4) a proliferative phase involving nonproductively infected lymphoid cells that may or may not progress to the point of lymphoma formation. Productive infection may occur transiently in B lymphocytes within a few days after infection with virulent Marek's disease virus strains and is characterized by antigen production, which leads to cell death. Because few if any virions are produced, this has also been termed a restrictive-productive infection. Productive infection also occurs in the feather follicle epithelium, in which enveloped virions are produced. Latent infection of activated T cells is responsible for the longterm carrier state. No antigens are expressed, but virus can be recovered from such lymphocytes by co-cultivation with susceptible cells in tissue cultures. Some T cells, latently infected with oncogenic Marek's disease virus strains, undergo neoplastic transformation. These transformed cells, provided they escape the immune system of the host, may multiply to form characteristic lymphoid neoplasms. Cell-mediated and humoral immune responses are both directed against viral antigens, with cell-mediated immunity probably being the most important.

LESIONS

Enlarged nerves are one of the most consistent gross lesions in affected birds. Various peripheral nerves, but particularly the vagus, brachial, and sciatic, become enlarged and lose their striations. Diffuse or nodular lymphoid tumors may be seen in various organs, particularly the liver, spleen, gonads, heart, lung, kidney, muscle, and proventriculus. Enlarged feather follicles (commonly termed skin leukosis) may be noted in broilers after defeathering during processing and are a cause for condemnation. The bursa is only rarely tumorous and more frequently is atrophic. Histologically, the lesions consist of a mixed population of small, medium, and large lymphoid cells plus plasma cells and large anaplastic lymphoblasts. These cell populations undoubtedly include tumor cells and reactive inflammatory cells. When the bursa is involved, the tumor cells typically appear in interfollicular areas.



peripheral nerve enlargement, chicken

(Courtesy of Dr. John Dunn)

DIAGNOSIS

For the diagnosis of Marek's disease, it is critical to diagnose the tumors and not the infection because Marek's disease is considered ubiquitous within commercial poultry flocks. Usually, diagnosis is based on enlarged nerves and lymphoid tumors in various viscera.

A diagnosis based on typical gross lesions may be confirmed histologically, or preferably by demonstration of predominant T-cell populations and Marek's viral DNA in lymphomas by histochemistry and PCR, respectively. There is a quantitative association between viral load and Marek's disease tumors; most tumor-bearing chickens have high viremia titers and are usually PCR positive.

PREVENTION

Vaccination is the central strategy for the prevention and control of Marek's disease. The efficacy of vaccines can be improved, however, by strict sanitation to reduce or delay exposure and by breeding for genetic resistance. Probably the most widely used vaccine consists of turkey herpesvirus (HVT), which has seen rapidly increased use as a backbone in recombinant vaccines featuring the insertion of genes from other poultry viruses, such as administered at hatching and require 1–2 wk to produce an effective immunity, exposure of chickens to virus should be minimized during the first few days after hatching.

Vaccines are also effective when administered to embryos at the 18th day of incubation. In ovo vaccination is now performed by automated technology and is widely used for vaccination of commercial broiler chickens, mainly because of reduced labor costs and greater precision of vaccine administration.

Proper handling of vaccine during thawing and reconstitution is crucial to ensure that adequate doses are administered. Cell-associated vaccines are generally more effective than cell-free vaccines, because they are neutralized less by maternal antibodies. Under typical conditions, vaccine efficacy is usually >90%. Since the advent of vaccination, losses from Marek's disease have been reduced dramatically in broiler and layer flocks. However, disease may become a serious problem in individual flocks or in selected geographic areas (eg, the Delmarva broiler industry). Of the many causes proposed for these excessive losses, early exposure to very virulent virus strains appears to be among the most important.

(as per Merck Veterinary Manual)

Pullorum Disease

By Sherrill Davison Yeakel, VMD, MS, MBA, DACPV, Laboratory of Avian Medicine and Pathology, School of Veterinary Medicine, University of Pennsylvania

Pullorum disease in poultry is caused by the bacterium *Salmonella enterica Pullorum.* The disease affects mainly young chicks, but can also affect older chickens, turkey poults and other avian species. The historical name for this disease is **bacillary white diarrhea (BWD)** so named that it causes a pasty white diarrhea in chicks. In the late 1920s, early '30s due to the high (80-100%) mortality of chicks the testing (the whole blood agglutination test which is still in use today) program was launched in and effort to control the disease. There was no one program overseeing this initiative which resulted in the Congressional appropriation in 1935 funding the National Poultry Improvement Plan (NPIP).

Pullorum disease **was once common but has been eradicated from most commercial chicken stock in the USA**, although it may be seen in other avian species (eg, guinea fowl, quail, pheasants, sparrows, parrots, canaries, and bullfinches) and in small backyard or hobby flocks. In adult chickens, mortality may be high, but frequently there are no clinical signs.

TRANSMISSION

Transmission can be vertical (transovarian) but also occurs via direct or indirect contact with infected birds (respiratory or fecal) or contaminated feed, water, or litter. Infection transmitted via egg or hatchery contamination usually results in death during the first few days of life up to 2–3 weeks of age. Transmission between farms is due to poor biosecurity.

CLINICAL SIGNS

The disease may be seen in all age groups, but **birds <4 weeks old are most commonly affected**. Birds may die in the hatchery shortly after hatching.

Affected birds:

- huddle near a heat source
- are anorectic
- appear weak

- have whitish fecal pasting around the vent (diarrhea)

Survivors are small in size and frequently become asymptomatic carriers with localized infection of the ovary. Some of the eggs laid by such hens hatch and produce infected progeny.

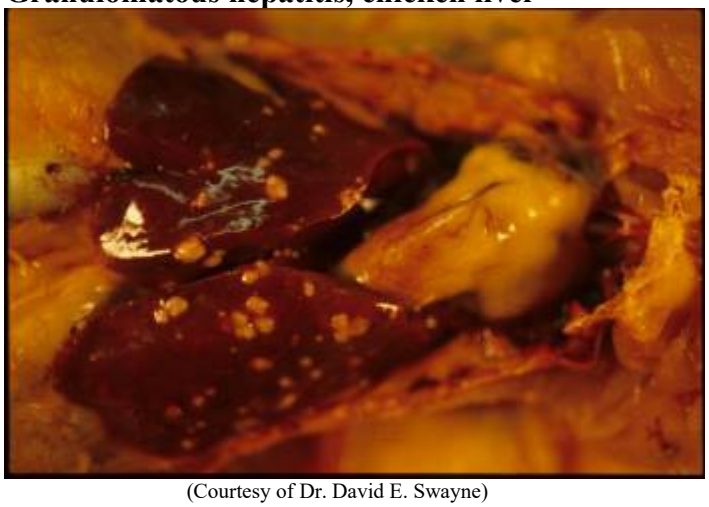
LESIONS
There may be no lesions due to an acute septicemia and death.

Lesions in young birds usually include unabsorbed yolk sacs and classic gray nodules in the liver, spleen, lungs, heart, gizzard, and intestine. Firm, cheesy material in the ceca (cecal cores) and raised plaques in the mucosa of the lower intestine are sometimes seen. Adult carriers usually have no gross lesions but may have nodular pericarditis, fibrinous peritonitis, or hemorrhagic, atrophic, regressing ovarian follicles with caseous contents. In mature chickens, chronic infections produce lesions indistinguishable from those of fowl typhoid.

Granulomatous splenitis, adult chicken

(Courtesy of Dr. David E. Swayne)

Granulomatous hepatitis, chicken liver



(Courtesy of Dr. David E. Swayne)

DIAGNOSIS

- Serologic testing to detect potentially positive birds, but isolation, identification, and serotyping is essential to confirm infection.
- Lesions may be highly suggestive, but diagnosis should be confirmed by isolation, identification, and serotyping of *S enterica Pullorum.* Infections in mature birds can be identified by serologic tests,

followed by necropsy evaluation complemented by microbiologic culture and typing for confirmation. Official testing recommendations for flocks in the U.S.A. are outlined in the National Poultry Improvement Plan.

TREATMENT

- Treatment of infected flocks will not alleviate the perpetuation of the carrier state and is never recommended.
- Freedom from infection and elimination of positive birds and flocks is key to control. Treatment will not eliminate the carrier state and is never recommended.

CONTROL

Control is based on routine serologic testing of breeding stock to assure freedom from infection. In addition, management and biosecurity measures should be taken to reduce the introduction of *S enterica Pullorum* from feed, water, wild birds, rodents, insects, or people. Birds should be purchased from sources free of *S enterica Pullorum.* The National Poultry Improvement Plan outlines the essential components for eradication of *S enterica Pullorum*. (as per Merck Veterinary Manual)

Turkey Rhinotracheitis

(aka: Avian Metapneumovirus, Avian Pneumovirus, Swollen Head Syndrome)
By Silke Rautenschlein , DVM, PhD, University of Veterinary Medicine-Hannover

Avian metapneumovirus (AMPV) causes turkey rhinotracheitis (or avian pneumovirus infection of turkeys), an acute respiratory tract infection of turkeys. It is also associated with swollen head syndrome (or avian rhinotracheitis) in broilers and broiler breeders, as well as reproductive disorders, with a significant drop in egg production in chickens and ducks. AMPV has been detected not only in chickens and turkeys but also in pheasants, Muscovy ducks, and guinea fowl, geese, most other duck species.

Infection with AMPV is often complicated by secondary bacterial infections, leading to high economic losses.

ETIOLOGY

Avian metapneumovirus is a member of the family Paramyxoviridae, genus *Metapneumovirus*, sub-family Pneumovirinae of the family Paramyxoviridae, which currently comprises the species AMPV and HMPV. Isolates of AMPV are currently grouped in subtypes A to D.

Rhinotracheitis in turkeys is perhaps the most economically important disease caused by avian metapneumovirus (aMPV) amongst poultry species, although avian metapneumovirus represents a number of related viruses that can replicate to a greater or lesser extent in other Galliformes, including the domestic fowl, and also in wild birds (Gough and Jones, 2008).

TRANSMISSION

Wild birds are considered natural reservoirs for avian metapneumovirus, and migratory birds may contribute to the distribution of the virus. A high apparent prevalence was recently determined particularly in mallards and American black ducks. The spread of AMPV appears to depend on the poultry population density, standard of hygiene, and biosecurity. Within or between poultry flocks, AMPV may spread rapidly horizontally by direct contact or by contact with contaminated material (morbidity rate up to 100%).

AMPV is assumed to be highly contagious. The enveloped virus is rapidly destroyed after release from the host to the environment. Because AMPV affects mainly ciliated epithelial cells of the upper respiratory tract, transmission is most likely to be airborne, especially by aerosol.

CLINICAL SIGNS

The incubation period is 3–7 days. The mortality may be 1%–50% depending on age and constitution of the flock as well as secondary infections. Birds without secondary infections with good constitution may recover within 7–10 days.

Typical respiratory signs of avian metapneumovirus in young turkeys include:

- serous ocular and nasal discharge
- frothy eyes
- conjunctivitis

At later stages, signs include mucopurulent, turbid nasal discharge; plugged nostrils; swollen infraorbital sinuses; and snicking, sneezing, coughing, or tracheal rales. These respiratory signs are accompanied by depression, anorexia, and ruffled feathers.

turkey poult, swollen infra-orbital sinuses.

(Courtesy of Dr. Rebecca Lindenwald)

Turkey with mucopurulent ocular discharge

(Courtesy of Dr. Rebecca Lindenwald)

LESIONS

Macroscopic lesions depend on the course of infection, especially on secondary bacterial infections, and are most prominent on days 4–10 after infection. Gross lesions induced after experimental infection are due to rhinitis, tracheitis, sinusitis, and air sacculitis. Infected birds may be free of gross lesions. Serous, turbid mucus, mucopurulent discharge may be observed in the nasal cavity, nasal turbinates, trachea, and in infraorbital sinuses.

(Courtesy of Dr. Arne Jung)

DIAGNOSIS

- Virus detection and serology is necessary to diagnose avian metapneumovirus infection.Obtaining samples from the upper respiratory tract of birds in the early stages of the disease is extremely important when attempting avian metapneumovirus isolation.
- Reverse transcriptase PCR (RT-PCR), as well as real-time RT-PCR, tests targeting the F, N, or G gene of AMPV have been developed. Some systems are commercially available, and these techniques are widely used to detect virus in clinical material, particularly respiratory swabs.

PREVENTION

- Vaccination (live or inactivated) and improved biosecurity.
- Live vaccines, which may be applied by spray or drinking water in the field, stimulate both local respiratory and systemic immunity, and cross-protection between subtypes may occur. But live vaccines may induce only short-lived protection, especially for grow-out of toms, because of the fast decline of local immunity.
- Inactivated AMPV vaccines are often used for booster immunization of layer and breeder flocks after priming with live vaccines.
- Good management practices can significantly reduce the severity of avian metapneumovirus infection, especially in turkeys; in particular, optimal ventilation, stocking densities, temperature control, litter quality, and biosecurity have a positive influence on the outcome of the disease.

TREATMENT

No specific treatment. Disinfectants such as quaternary ammonia, ethanol, iodophors, phenol derivatives, as well as bleach may be used to reduce the viability of AMPV. Some success in reducing disease severity by controlling secondary bacterial infections with antibiotics has also been reported.

(as per Merck Veterinary Manual and https://www.cabi.org/isc/datasheet/60956)

Infectious Bronchitis

By Mark W. Jackwood , MS, PhD, Department of Population Health, College of Veterinary Medicine, University of Georgia

Avian infectious bronchitis (**IB**) is an acute and highly contagious respiratory disease of chickens.

ETIOLOGY

The disease is caused by avian infectious bronchitis virus (IBV), a coronavirus. IBV was the first Coronavirus described and varies greatly genetically and phenotypically, with hundreds of serotypes and strains described. Coronaviruses contain the largest known viral RNA genome in number of nucleotides, of approximately 30,000 bases.

CLINICAL SIGNS

Respiratory signs predominate including gasping, coughing, sneezing, tracheal rales, and nasal discharge. In young chickens, severe respiratory distress may occur. Coughing and rattling are common, most severe in young, such as broilers, and rapidly spreading in chickens confined or at proximity. Morbidity is 100% in non-vaccinated flocks. Mortality varies according to the virus strain (up to 60% in non-vaccinated flocks).

In layers, respiratory distress, nephritis, decrease in egg production, and loss of internal (watery egg white) and external (fragile, soft, irregular or rough shells, shell-less) egg quality are reported.

wrinkled egg shells

(Courtesy of Dr. Jean Sander)

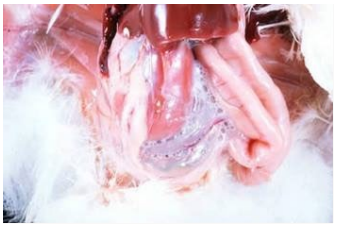
Respiratory signs will subdue within two weeks. However, for some strains, a kidney infection may follow, causing mortality by toxemia. Younger chickens may die of tracheal occlusion by mucus (lower end) or by kidney failure. The infection may prolong in the cecal tonsils.

In laying hens, there can be transient respiratory signs, but mortality may be negligible. However, egg production drops sharply. A great percentage of produced eggs are misshapen and discolored. Many laid eggs have a thin or soft shell and poor albumen (watery) and are not marketable or proper for incubation. Normally-colored eggs, indicative of normal shells for instance in brown chickens, have a normal hatchability.

LESIONS

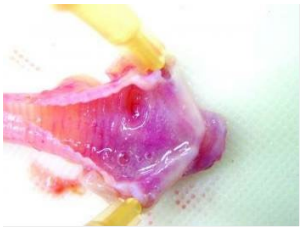
In the respiratory tract, the trachea, sinuses, and nasal passages may contain serous, catarrhal, or caseous exudates, and the air sacs a foamy exudate initially, progressing to cloudy thickening.

air sacculitis



(Courtesy of Dr. Jean Sander)

trachea with excessive amount of mucus



(Courtesy of Dr. Pedro Villegas)

If complicated by infection with *E coli*, there may be caseous air sacculitis, perihepatitis, and pericarditis. Birds infected when very young may have cystic oviducts, whereas those infected while in lay have an oviduct of reduced weight and length and regression of the ovaries. Infection with nephropathogenic strains results in swollen, pale kidneys

DIAGNOSIS

Chicken and turkey respiratory diseases are difficult to differentiate and cannot be diagnosed based on respiratory signs and lesions.

- Detection of rising antibody titers by ELISA or HI testing and virus detection and typing using RT-PCR and sequencing.
- Laboratory confirmation is required for diagnosis of respiratory forms of infectious bronchitis because of similarities to mild forms of disease caused by agents such as Newcastle disease virus, avian metapneumovirus, infectious laryngotracheitis virus, mycoplasmas, *A. paragallinarum*, and *Ornithobacterium rhinotracheale*.
- Demonstration of seroconversion or a rise in antibody titer against IBV by ELISA, or hemagglutination inhibition or virus neutralization tests can be used for diagnosis when there is a history of respiratory disease or reduced egg production.
- Presumptive diagnosis is commonly achieved using reverse transcriptase PCR assays to detect viral RNA in nucleic acid extracts of tracheal, cecal tonsil, or kidney tissue.
- Definitive diagnosis is generally based on virus detection and identification. Virus can be isolated by inoculation of homogenates of tracheal, cecal tonsil, and/or kidney tissue into 9- to 11-day-old SPF chicken embryos, with growth of IBV indicated by embryo stunting and curling and by deposition of urates in the mesonephros, with variable mortality.

TREATMENT

No medication alters the course of IBV infection, although antimicrobial therapy may reduce mortalities caused by complicating bacterial infections. In cold weather, increasing the ambient temperature may reduce mortalities, and reducing the protein concentrations in feed and providing electrolytes in drinking water may assist in outbreaks caused by nephropathogenic strains.

PREVENTION

- Attenuated live and killed vaccines are used to control the disease, but little or no cross reactivity between types requires the correct vaccine type be applied.
- The live-attenuated vaccines used for immunization may produce mild respiratory signs. These vaccines are initially given to 1- to 14-day-old chicks by spray, drinking water, or eye drop, and birds are commonly revaccinated approximately 2 weeks after the initial vaccination. Revaccination with a different serotype can induce broader protection.
- As with most infectious diseases of poultry, the application of rigid biosecurity is the best preventive measure.

(as per Merck Veterinary Manual)

Virulent Newcastle Disease

(aka: Exotic Newcastle Disease, END, vND, VVND, Neurotropic and Viscerotropic strains)
*By **Patti J. Miller**, DVM, PhD, USDA/Agricultural Research Service*

Virulent Newcastle disease (**vND**), formerly known as **exotic Newcastle disease** is a contagious viral avian disease affecting many domestic and wild bird species; it is transmissible to humans. Though there are rare cases where the disease gives a mild fever and/or conjunctivitis. Its effects are most notable in domestic poultry

due to their high susceptibility and the potential for severe impacts of an epizootic on the poultry industries. It is endemic to many countries.

GEOGRAPHIC DISTRIBUTION

- Endemic Areas
  - Asia, the Middle East, Africa, Mexico, Central and South America
- Vaccine use makes assessment of true geographical distribution difficult worldwide
- International monitoring by:
  - FAO
  - OIE
  - USDA APHIS Veterinary Services

ETIOLOGY

The Newcastle diseases are cause by members of Family Paramyxoviridae, Genus *Avulavirus*, which includes 9 serotypes [Avian Paramyxovirus-1 thru 9, (APMV-1 to APMV-9)]. Virulent Newcastle disease virus is APMV-1.

There are three pathotypic strains **(lentogenic, mesogenic, and velogenic)** of Newcastle disease virus:

Lentogenic	Mesogenic	Velogenic
Common in non-commercial backyard flocks	Uncommon	**Most recently vND outbreak in Southern California (2018-20)**
Subclinical	Intermediate virulence: Occasionally neurological signs	**Most serious poultry disease in world**
Mild respiratory disease, decreased egg production & quality, weight loss		**Death without clinical signs**
Negligible mortality	Low mortality	**95-100% mortality**
No trade restrictions		**Shut down trade**

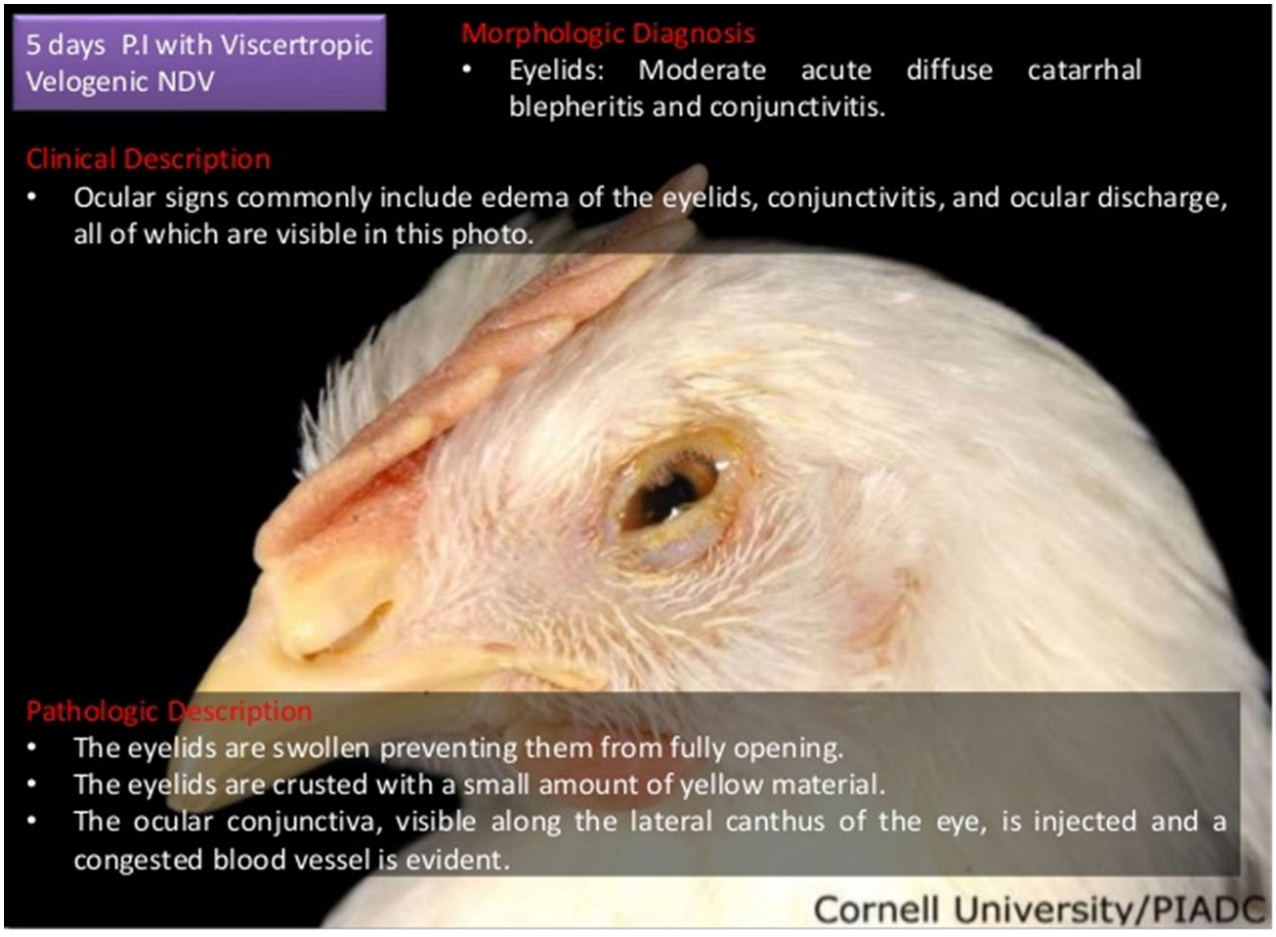
Virulent Newcastle disease (vND) is reputed to be the most severe of all diseases affecting chickens. There have been three devastating incursions of into the United States since 1971 in the same geographical area, and

same demographic in Southern California. The first of those occurred during 1971-1974, was traced to the importation and release of infected exotic pet birds, and cost over $56 million in Federal funds to eradicate. The second occurred during 2002-2003, started in back yard holdings of illegally imported game fowl, and eventually spread to commercial poultry. Over $180 million in Federal funds was expended to eradicate that outbreak. The third incursion occurred in May 2018, in chickens examined at a veterinary clinic in Los Angeles County. California Department of Food and Agriculture staff and the United States Department of Agriculture have been working on eliminating VND in South California and more than 400 birds have been confirmed to have VND.[6][7] On February 27, 2019, California State Veterinarian, Dr. Annette Jones, increased the quarantine area in Southern California and on March 15, 2019 and April 5, 2019, cases of VND in Northern California and Arizona respectively. (as per *"Virulent Newcastle Disease". California Department of Food and Agriculture. Retrieved 6 April 2019.)*

CLINICAL SIGNS

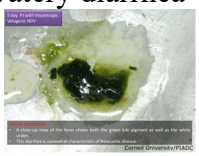
- High mortality within 24 to 48 hours (deaths continue for 7 to 10 days)
- Drop in egg production

- Upper Respiratory Signs



- Edema of head, especially around eyes

- Greenish, dark watery diarrhea



- Neurological signs:

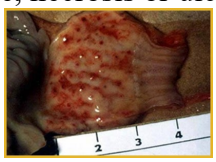
- Signs vary with species and virulence
- Surviving birds may have neurological or reproductive damage

Morbidity and Mortality:

- Morbidity: up to 100%
- Mortality: 90-99%
- Varies greatly depending on:
  - Virulence and strain
  - Avian species and susceptibility of host
  - Environmental conditions
  - Vaccination history
- Some species show few or no signs
  Carrier state may exist

LESIONS

- Edema, hemorrhage, necrosis or ulceration of lymphoid tissue

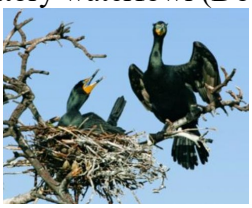


- Hemorrhagic lesions
  - Tracheal mucosa
  - Proventriculus
  - Intestinal mucosa

POSSIBLE ROUTES OF ENTRY INTO THE UNITED STATES

- Smuggled (fighting) cocks

- Exposed psittacine birds entering the U.S.
- Exposed migratory waterfowl (Double Crested Cormorants & Northern Pintail dabbling ducks)

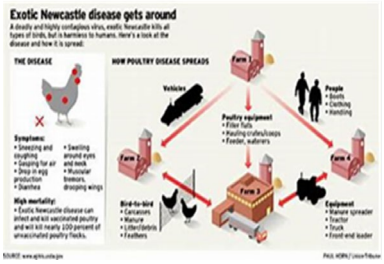


SOURCES OF vND Virus:

- Respiratory secretions/discharges and feces of infected birds
- All parts of an infected carcass
- Virus is shed during the incubation period, during clinical stages and for a limited period during convalescence
- Cormorants, pigeons, doves, and imported psittacine species are more commonly infected with vNDV and have also been sources of vNDV infections of poultry
- Some psittacine birds have been demonstrated to shed ND virus intermittently for over 1 year

TRANSMISSION

- Direct contact with feces, respiratory secretions
- Indirect contact:
  - Contaminated feed or water
  - Equipment
  - Human clothing
- Contaminated or incompletely inactivated vaccines
- Migratory birds (Double Crested Cormorants & Northern Pintail dabbling ducks)
- Doves, Feral Pigeons, or Psittacines
- Contaminate Fomites (equipment, vehicles, people)



DIFFERENTIAL DIAGNOSIS

- Fowl cholera
- Highly pathogenic avian influenza
- Infectious Laryngotracheitis
- Fowl Pox (diphtheritic form)
- Psittacosis (psittacine birds)
- Mycoplasmoses
- Infectious Bronchitis

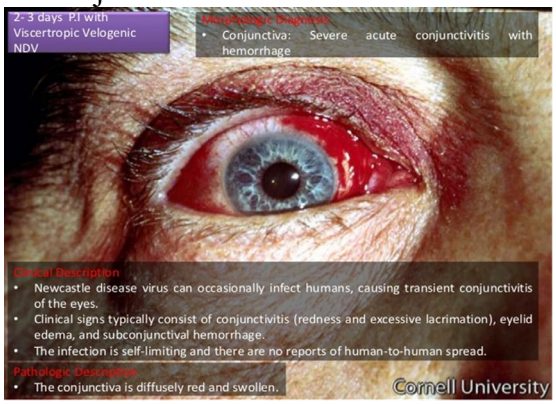
- Aspergillosis
- Management errors such as deprivation of water, lack of or nutritionally deficient feed and poor ventilation
- In pet birds: Pacheco's parrot disease (psittacine birds), salmonellosis, adenovirus, and other paramyxoviruses
- In cormorants and other wild waterfowl: botulism, fowl cholera and conformational abnormalities

DIAGNOSIS

- **Hemagglutination and hemagglutination inhibition tests:** most widely used and detects antibody response to virus glycoprotein (predictor of protection against disease)
- **Enzyme-linked immunosorbent assay (ELISA):** as whole virus is used as antigen, detects antibody to all of the virus proteins
- **Polymerase Chain Reaction (PCR)** on swab samples
- **Virus isolation** (the prescribed test for international trade): inoculation of embryonated specified pathogen free (SPF) eggs and tested for haemagglutination (HA) activity and/or by use of validated specific molecular methods

ZOONOTIC POTENTIAL

Can cause severe conjunctivitis'



ECONOMIC IMPACT

- **Global Economic Impact**
  - vND more costly than any other animal virus?
    - Control measures expensive
    - Repeated testing for trade purposes
- **Developing Countries**
  - Endemic vND affects quality and quantity of dietary protein
  - Human health

History of Exotic Newcastle Disease

Newcastle disease was first identified in Java, Indonesia, in 1926, and in Newcastle-upon-Tyne, England, in 1927. However, it may have been prevalent as early as 1898, when a disease wiped out all the domestic fowl in northwest Scotland (as per *Macpherson, LW (May 1956). "Some Observations On The Epizootiology Of Newcastle Disease". Canadian Journal of Comparative Medicine and Veterinary Science.* ***20*** *(5): 155–68)*

The policy of slaughter ceased in England and Wales on March 31, 1963, except for the peracute form of Newcastle disease and for fowl plague. In Scotland the slaughter policy continued for all types of fowl pest.[5] Interest in the use of NDV as an anticancer agent has arisen from the ability of NDV to selectively kill human tumor cells with limited toxicity to normal cells. (as per *"Newcastle disease: Newcastle disease outbreaks in Great Britain". DEFRA. Archived from the original on 2007-06-27)*

1950: First Reported vND case in the United States

- The first case of vND occurred in 1950 in partridges and pheasants imported from Hong Kong.
- In November, 1971, a major outbreak occurred in commercial flocks in southern California after the arrival of a shipment of infected pet birds from Latin America (i.e., the probable source was an imported parrot)/
    - In March of 1972, a national animal health emergency was declared and a major eradication campaign began.
    - During the 2 year effort, 1,321 infected and exposed flocks were located, and almost 12 million birds were destroyed. The operation cost taxpayers approximately $56 million.
    - In July 1974 the U.S. succeeded in eradicating vND. Frequent outbreaks of vND have occurred in the U.S. since due to illegal importation of exotic birds and poultry.

- In December, 2002, vND was again disclosed on southern California in backyard "exhibition" flocks which spread to commercial operations.
    - The introduction of the disease was attributed to smuggled fighting cocks out of Mexico.
    - The disease spread to southern Nevada and western Arizona.
    - The cost of eradicating the disease was ~$160 M.
    - The impact of trade restrictions resulting from the disease incursion was ~$395 M.

Quarantine Zone in 2002-03:

- **In April, 2003, vND was also disclosed in Texas**
    - The difference was that the infected game fowl flock was immediately diagnosed and depopulated BEFORE the disease spread to poultry with minimal cost.
    - The END virus had originated out of Chihuahua, Mexico, and different than the virus in California...
    - One infected Game Fowl disclosed east of El Paso
    - Samples were couriered to NVSL.
    - Flock depopulated before VI at NVSL
    - ~2,000 flocks tested with no evidence of spread.
    - Quarantine released without any further infection found.

The 2018-20 OUTBREAK

- On May 16, 2018, California Department of Food and Agriculture (CDFA) officials reported a presumptive positive vND detection from a sample collected on May 15 by an accredited veterinarian from a chicken from a backyard premises in Los Angeles County, CA; the National Veterinary Services

Laboratories (NVSL) later confirmed the sample as positive for vND. CDFA personnel immediately placed a quarantine on the premises and began an epidemiological investigation. District 6, in cooperation with CDFA, activated the CA Blended IMT, which included representation from both VS and CDFA.

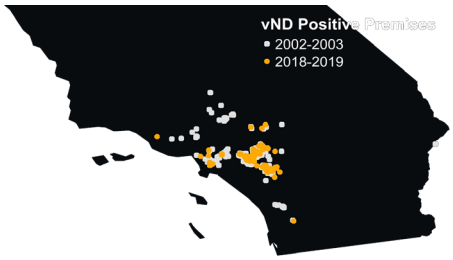
- Genetics of the 2018 vND virus included:
  - Analysis of the CA 2018 viruses isolates supports a single, recent introduction followed by secondary spread.
  - No change in the amino acid profile of the cleavage site has been observed for sequenced viruses (PGGRRQKR/FVGAII).
  - Chickens remain predominantly affected, other species identified include quail, peafowl (peacock) and turkey, as well as duck, goose, parakeet, and pigeon.

(as per Dimitrov KM, et al. Virology, 2019 Vol 531:203- 218, https://doi.org/10.1016/j.virol.2019.03.010)

- Map of quarantined areas:

  - Spread of the disease to Alameda Co. and counties in Utah and Nevada resulted from movement of exposed birds out of the quarantine zone to those areas.

- Comparison of affected flocks in 2002-03 with those in 2018-20:



Many of the same flocks were repeatedly affected.

- Trade embargos were delayed because of OIE's definition of "poultry":
  - Domesticated birds, including backyard poultry, used for the production of meat or eggs for consumption, for the production of other commercial products, for restocking supplies of game, or for breeding these categories of birds, as well as fighting cocks used for any purpose.

- Birds that are kept in captivity for any reason other than those reasons referred to in the preceding paragraph, including those that are kept for shows, races, EXHIBITION, competitions or for breeding or selling these categories of birds as well as pet birds, are not considered to be poultry.
- Once the infection entered into commercial laying operations in the area, trade restrictions were levied.

- Example of Response Complexity:
  - Ventura Co. Infected Flock:
    - Over 80 miles from nearest affected flock in LA C0.
    - Disclosed on sick bird call (2 pos. out of 6 samples)
    - Rooster Boarding Facility (w/ pigeons)
      (actually a "training camp" for exhibition birds)
    - >4,400 birds depopulated
    - 57 owners (38 VS Form 1-23's processed)
    - No adjacent flocks; >400 contacts

- Affected premises by production type:

Production Type	Infected Premises
Backyard Exhibition	476
Backyard Non-Commercial Laying Chickens	6
Backyard Non-Commercial Turkeys	1
Backyard Non-Commercial Poultry	1
Backyard Non-Commercial Chickens	1
Commercial Table Egg Layer	3
Commercial Table Egg Pullets	1
Live Bird Sales / Non-Slaughter	1
Live Bird Sales / Slaughter	1
Antibody/Research Facility	1
Retail Feed Store	4
Retail Pet Store	1
Veterinary Clinic	1
Total	**498**

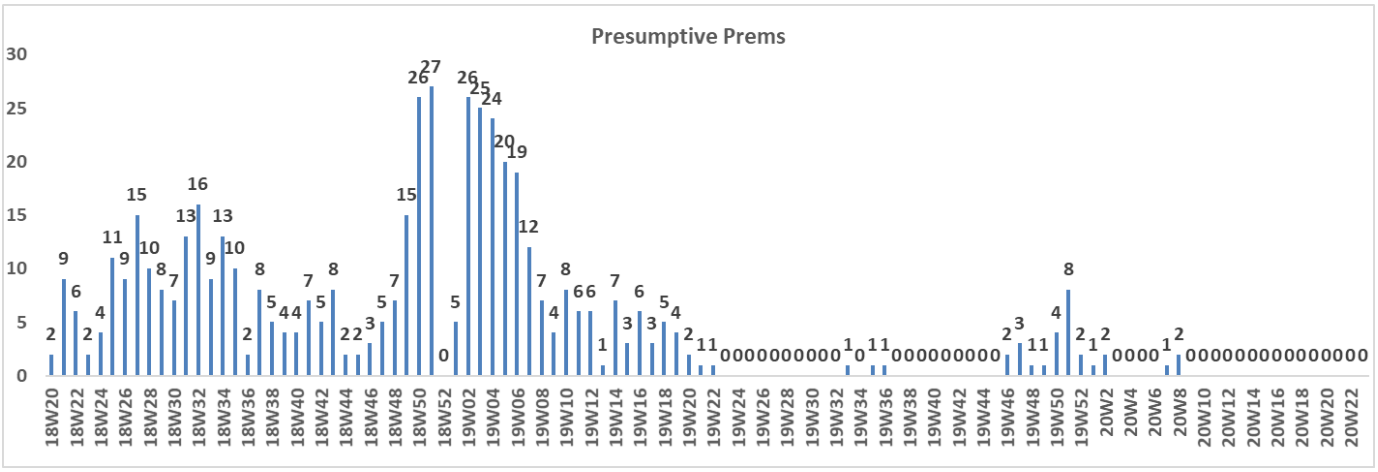
- Number of birds euthanized by production type:

Production Type	Total
Confirmed Positive	**1,169,418**
Backyard Exhibition	53,615
Commercial Table Egg Layer	719,321
Commercial Table Egg Pullets	102,574
Backyard Non-Commercial Laying Chickens	292,772
Live Bird Sales / Non-Slaughter	493
Live Bird Sales / Slaughter	170
Research Facility	54
Retail Feed Store	419
Presumptive Positive	**1,549**
Backyard Exhibition	1,549
Dangerous Contact	**73,709**
Backyard Exhibition	70,758
Commercial Rendering	119
Retail Feed Store	2,702
Retail Pet Store	130
Grand Total	**1,244,676**

- Number of premises depopulated in each affected county:

County	Most Recent Depopulation	# Premises Depopulated
ALAMEDA	04/12/2019	1
LOS ANGELES	01/24/2020	142
RIVERSIDE	02/24/2020	1,505
SAN BERNARDINO	02/25/2020	692
SAN DIEGO	09/8/2019	8
SAN JOAQUIN	04/12/2019	1
VENTURA	08/16/2018	1
Grand Total		**2,350**

- Epidemiological Summary (affected premises disclosed per week from initial to quarantine release):



(Data as per CA vND 2018 SitRep #393 6-03-2020)

- Predicting Epidemiological Risk (Case Control Study):
  - Premises with more than 20 poultry (OR = 6.4)
  - Poultry used for exhibition or commingled for any purpose (OR = 4.8)
  - Premises with over 50% roosters in the flock (OR = 2.4)
  - Premises with poultry in contact with wild birds (OR = 2.6)
  - Close geographic proximity to other premises containing outdoor poultry (OR = 2.2)
    (as per Dr. Jennifer Siembieda, CEAH)
- Surveillance Testing To Prove No Infection:
  - Testing to identify new cases:
  - Commercial – twice weekly
  - Independent – twice weekly
  - Backyard – once and then during control area release
  - LBM – monthly with down days every 120 days
  - Sick bird calls
    (as per Dr. Jennifer Siembieda, CEAH)
- Virus Elimination Practices:
  - 120-day fallow period on backyard exhibition bird premises
    - 20, 40, 60, 80, 100, and 120 days
    - 10,994 fallow checks
  - C&D commercial and non-commercial table egg layer in accordance with flock plans and include:
  - Vector control, dry/wet cleaning, flushing water lines, removal of feed, a Wildlife Services assessment, disinfection, and environmental sampling requirements

- Conclusions:
  - In all three outbreaks ((1971-74, 2002-03, and 2018-20) virtually identical geographic area and primary demographics involved:
    - Primarily backyard exhibition birds
    - Biosecurity non-existent
  - A potential “wildlife reservoir” in the area:
    - In Southern California there are at least 11 species of feral (free-ranging) parrots inhabiting at least 35 cities.
    - Many came to Southern California via the imported pet trade or were released by people when they got tired of confining them.
  - Confounding factor of vaccination is ever apparent.

- CDFA has a staff in place to constantly encourage optimum biosecurity among the poultry producers in the area.

- **WE ARE HOPEFUL TO PREVENT ANOTHER Virulent Newcastle Disease Incursion.**

(as per Merck Veterinary Manual)

Highly Pathogenic Avian Influenza (HPAI, H5 & H7 LPAI)

*By **David E. Swayne**, DVM, PhD, DACVP, DACPV, USDA-ARS, Southeast Poultry Research Laboratory*

Avian influenza (AI) is a viral infection of domestic poultry, and pet, zoo, and wild birds. In domestic poultry, AI viruses are typically of low pathogenicity (LP), causing subclinical infections, respiratory disease, or drops in egg production, but a few AI viruses are highly pathogenic (HPAI), causing severe systemic disease with multiple organ failure and high mortality. The H5 and H7 strains low pathogenic avian influenza of influenza A viruses are of particular interest because of their propensity to mutate into HPAI, so they are managed as HPAI.

ETIOLOGY

Influenza viruses belong to the family *Orthomyxoviridae*. They are classified into three main types:

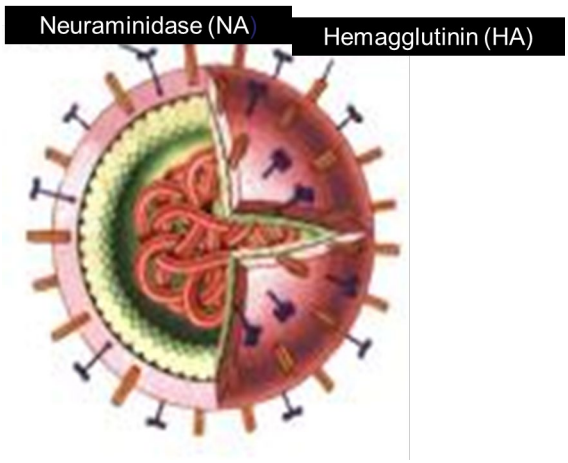
- Influenza type A viruses affect multiple species including ALL avian species.
- Influenza types B and C both infect humans, but type C is also known to infect swine.

Comparison of Three Antigenic Types:

COMPARISON OF INFLUENZA VIRUS TYPES			
Antigenic type	**A**	**B**	**C**
RNA segments	8	8	7
Surface glycoproteins	Hemagglutinin (1 to 15) Neuraminidase (1 to 9)	Hemagglutinin Neuraminidase	HEF: hemagglutinin esterase fusion protein
Genetic variability	Drifts and shifts	Drifts	Drifts
Human disease	Epidemic, pandemic, sporadic	Epidemic, sporadic	Sporadic
Known hosts	Humans, swine, horses, poultry, sea mammals several avian species	Humans	Humans, swine
Target for current antivirals	M2 protein (amantadine, rimantadine) Neuraminidase (zanamivir, oseltamivir)	Neuraminidase (zanamivir, oseltamivir)	Not available

- Comparison of Influenza A Antigenic Types"
  - Influenza A viruses can be divided into 16 Hemagglutinin (H) antigen and 9 Neuraminidase (N) antigen groups.
  - Extreme antigenic variability brought about by genetic reassortment in host cells.

- Therefore, there are 144 possible different serotypes of the virus, based on 16 H types and 9 N types



  - H5 and H7 serotypes cause the most severe disease in birds
  - Human AI diseases are caused by H1, H2, H3, H5, H7, H9, N1, N2.

(as per N.G. Darwaza)

- The AI Genome contains 8 segments of single strand RNA. Can code for up to 11 proteins.
- Important proteins:
  - Hemagglutinin: attachment to cell receptors –contains viral neutralizing antigens-16 antigenic types H1-H16
  - Neuraminidase: contains viral neutralizing antigens –9 antigenic types N1-N9
  - Nucleoprotein: major structural protein
  - Matrix: major structural protein

Standard nomenclature: A/chicken/Pennsylvania/1370/83 (H5N2)

(as per Dr. Mia Torchetti, NVSL)

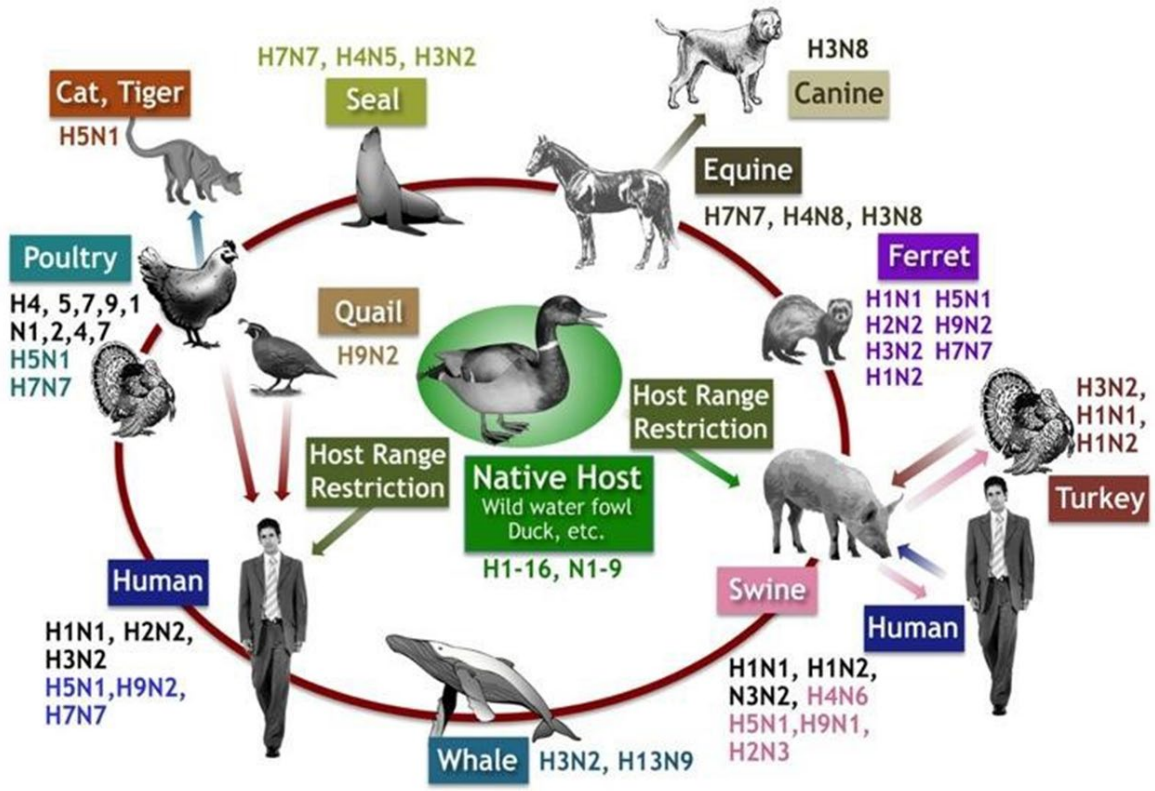
- Influenza A Viruses are now classified in three pathogenic groups (instead of the formerly two, HPAI & LPAI):
  1. HPAI, which are mainly H5 & H7, but can be any strain of Influenza A viruses
  2. LPAI, which are now only H5 & H7 subtypes.
     These two subtypes can mutate, or reassert, to become HPAI.
  3. Influenza A Viruses of low pathogenicity are in a separate class.

  Note: Only the first two groups are reportable. (as per Dr. Mia Torchetti, NVSL)
- HPAI viruses (particularly H5) are created by mutation in poultry (chickens and turkeys), and not in waterfowl. Transmission of these HPAI viruses is from poultry to waterfowl. Once HPAI is in the waterfowl population it can be perpetuated in that group and be transmitted to susceptible poultry. Highly Pathogenic Avian Influenza (HPAI) can now be any subtype of avian influenza virus (no longer restricted to just H5 or H7 subtypes). (as per Dr. Mia Torchetti, NVSL)
- **All Influenza Viruses are constantly evolving, by:**
  →Mutation
  →Reassortment: when two Influenza virus come together in the same host to cause exchange of genetic material.
  →Antigenic Drift: mutations in the H gene casing minor antigenic changes in the H protein
  It is an ongoing process which limits efficacy of protective and vaccine induced immunity
  →Antigenic Shift: Replacement of H or N
  →All of the aforementioned processes can result in new strains of novel (pandemic) strains.

(as per Dr. Mia Torchetti, NVSL)

EPIDEMIOLOGY

- Host Range:

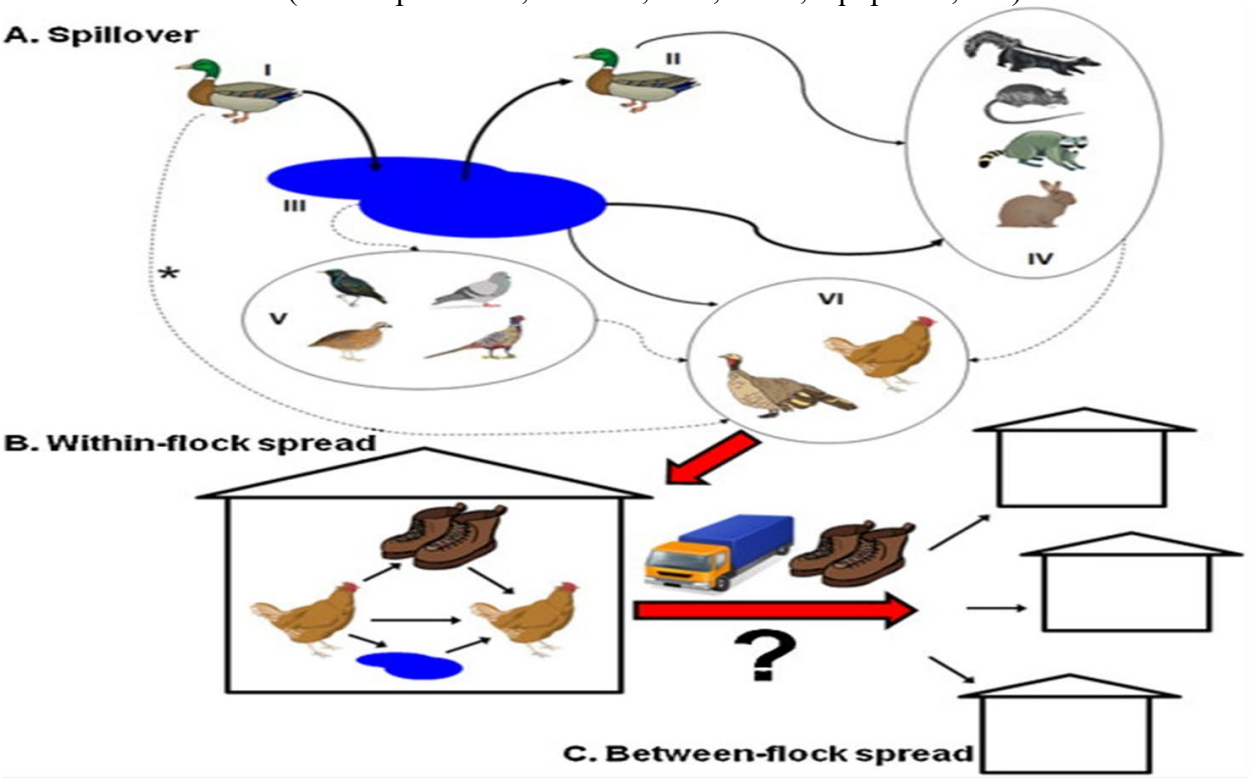


(as per http://go.usa.gov/KpGP)

- Low pathogenicity avian influenza viruses are distributed worldwide and are recovered frequently from clinically normal shorebirds (Chadriiformes) and migrating waterfowl (Anseriformes). Occasionally, LP viruses are recovered from imported pet birds and ratites. The viruses may be present in village or backyard flocks and other birds sold through live-poultry markets, but most commercially raised poultry in developed countries are free of AI viruses. (as per David Swayne, ARS)
- In certain geographic areas, dogs (H3N8 and H3N2) and cats (H7N2) may be commonly infected by specific influenza A viruses that are adapted to each specific species. (as per David Swayne, ARS)
- Avian influenza virus (AIV) is endemic in wild birds
  - Transmission of AIV from wild birds to poultry species (ducks, chickens, turkeys) commonly occurs
  - AIV on rare occasions may become adapted to and become endemic in poultry species (chickens and turkeys)
  - AIV once adapted to chickens and turkeys can be difficult to eradicate
  - Has been demonstrated to jump species barriers (as per Kapczynski, SEPRI)

TRANSMISSION

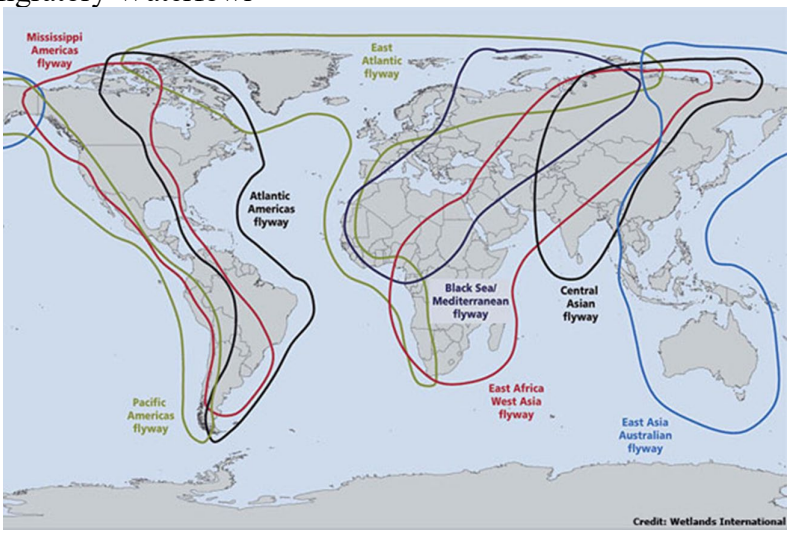
- Potential Sources of AIV:
  - Migratory waterfowl or other wild birds
  - Domestic poultry
  - Domestic pigs
  - Companion or pet birds (least likely source) (as per Kapczynski, SEPRI)

- Most likely transmission routes are direct or indirect exposure to affected migratory waterfowl or by contaminated fomites (service personnel, vehicles, feed, water, equipment, etc.)
- 

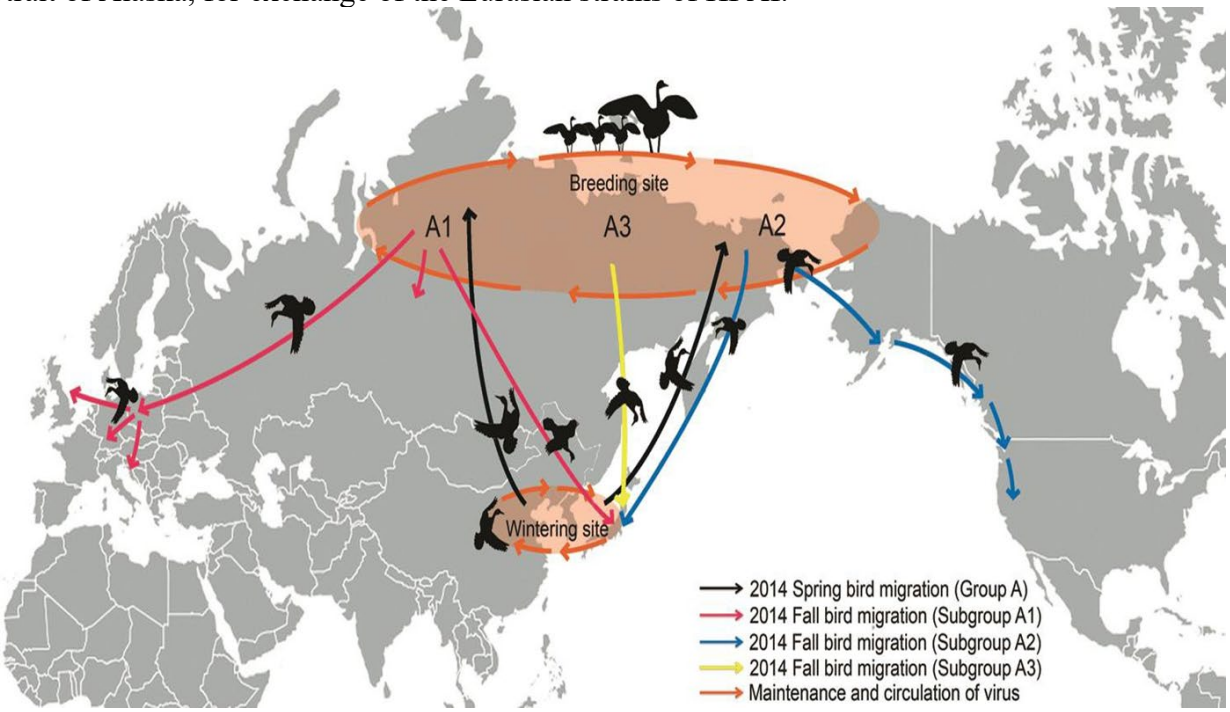
(as per Pepina, et.al)

- Possible Routes of Introduction into an Area or Flock:
  - Contact with Exposed Migratory Waterfowl

  - Imported (smuggled?) Exposed/Infected Birds
  - Movement of contaminated equipment or people onto premises
  - Mutation of LPAI into HPAI
- Flyways of Migratory Waterfowl



- The three flyway that cross North America are:
  - Pacific Flyway which is parallels the Pacific Coast
  - The Central Flyway which courses down the midwestern United States
  - The Mississippi Flyway which roughly follows the course of the Mississippi River
  - The Atlantic Flyway which parallels the Atlantic Coast
  - Exposed migratory waterfowl might traverse any of these four flyways
- There is potential transmission between Asian migratory (from China, Taiwan, South Korea, etc. where HPAI is more or less endemic) waterfowl with North American migratory waterfowl over the Bering Strait of Alaska, for exchange of the Eurasian strains of HPAI:

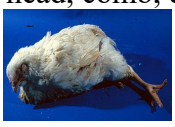


(Lee DH, Torchetti MK, Winker K, Song CS, Swayne D. *Intercontinental Spread of Asian-origin H5N8 to North America through Beringia by Migratory BirdsJ. Virol. JVI.00728-15; epub ahead of print 8 April 2015, doi:10.1128/JVI.00728-15)*

CLINICAL SIGNS:

- Most wild waterfowl are carriers with subclinical signs
- Infections in poultry:
  - Dyspnea and oral/nasal discharges
  - Cyanosis of head, comb, & waddle

  - Ataxia



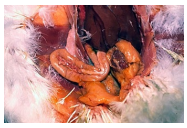
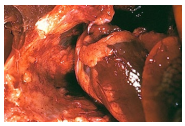
- Hemorrhagic Skin of Head: 

- Blanched shanks and paws 

- **High mortality**
  - Approaches 100% in commercial poultry flocks
  - Deaths within 2 to 12 days after first signs of illness
  - Survivors in poor condition

(Courtesy of Dr. David E. Swayne)

LESIONS

- Petechial hemorrhages in many organ systems.
- Tracheitis: 
- Pancreatis: 
- Epicardial hemorrhages: 

.(Swayne)

(Courtesy of Dr. David E. Swayne)

DIAGNOSIS

- Direct Detection:
  - Antigen detection: antigen capture immunoassays detect influenza A nucleoprotein. Not as sensitive as RT-PCR or virus isolation. Very useful as a screening test.
  - RT-PCR and rRT-PCR: have become the standard routine detection methods in many cases. rRT-PCR for detection of the influenza A matrix gene often used as a screening test followed by rRT-PCR for H5 and/or H7 hemagglutiningenes.
  - Virus isolation and identification: performed in embryos. Still the gold standard.

- Antibody Detection:
  - Agar gel immunodiffusion (AGID): detects antibody to viral nucleoprotein which is common to all influenza A viruses. Used as a screening assay primarily.
  - ELISA: widely used as a screening test. Some level of false positives which must be confirmed with AGID or direct detection.
  - Hemagglutination Inhibition (HI): not typically used as a screening test. Used primarily to detect hemagglutinin type specific antibody in samples that were found to be positive by ELISA or AGID.

TREATMENT

No specific treatment for HPAI or LPAI

- Quarantine and Depopulate all HPAI and H5/H5 Flocks
- Supportive care for LPAI flocks.

PREVENTION

- Practice of exclusion biosecurity strategies to prevent introduction of AI into poultry is the best preventive measure. Suspected outbreaks should be reported to appropriate regulatory authorities.
- Should a HPAI incursion occur in a commercial flock, in order to qualify for indemnity, they are required to apply NPIP's 14 Biosecurity Principles in place.
- Antigenically matched and properly administered vaccines can prevent clinical signs and death and greatly reduce virus replication and shedding from the respiratory and GI tracts.
- Specific protection is achieved through autogenous virus vaccines or from vaccines prepared from AI virus of the same hemagglutinin subtype.

ZOONOTIC RISK

- Avian influenza viruses exhibit host adaptation to birds. Human infections have occurred, usually as isolated, rare, individual cases.
- Most human cases have originated from infection with Eurasian H5 HPAI virus (A/Goose/Guangdong lineage), and, most recently, H7N9 LPAI virus (Chinese lineage). This lineage of H5N1 HPAI virus has total accumulated human cases in Asia and Africa from 2003–June 2019 of 861, of which 455 were fatal.
- The primary risk factor for human infection has been direct contact with live or dead infected poultry, but a few cases have resulted from consumption of uncooked poultry products, defeathering of infected wild swans, or close contact with human cases.

* * * * * * * *

OVERVIEW of HISTORY OF HPAI in the United States:

- 1890's-1950's, numerous fowl plague outbreaks, subtypes not known 1924-25 Northeast US?
- 1927, New Jersey?
- 1983-84, Pennsylvania, H5N2 (mutated from LPAI to HPAI)
- 2004, Southeastern Texas, one independent broiler flock, H5N2 – HPAI per cleavage site
- 2015, H5N2, H5N8, multiple states
- 2016, H7N8, (NA Lineage), southern Indiana, one Control Zone
- 2017, H7N9 US (NA Lineage), Tennessee, 2 affected flocks (one Control Zone) mutated from LPAI to HPAI.
- 2020, H7N3, South Carolina (one house, mutated from LPAI to HPAI)

* * * * * * * *

1983-84 H5N2 HPAI Incursion:

- Introduced into backyard flocks by exposure to migratory waterfowl as a LPAI H5N2
- Mutated into HPAI and spread into commercial layer and broiler flocks

- Began in Lancaster Co., Pennsylvania, with spread into Virginia
- State of Emergency Declared and "VS Task Force Deployed)
    - $63 million spent
    - 443 affected flocks disclosed
    - 17 million birds depopulated
- No human infection was disclosed.

* * * * * * * *

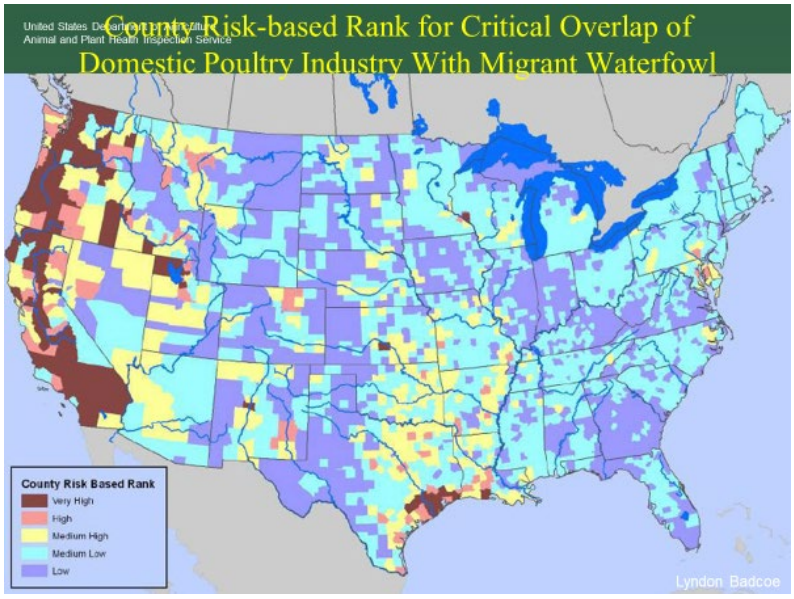
2004, H5N2 HPAI disclosed in Gonzales, Texas

- In a small independent broiler flock
- The rapid response was the key to limiting the infection.
- Feb. 17 - report of positive HPAI H5N2
- Feb 21 – flock depopulated (6,600 chicken broilers) on index farm & no additional infection disclosed (limited to one flock).
- Owner had procured replacements from a live bird market in Houston which (by circumstantial evidence) was the source of the infection.
- No human infection disclosed.

* * * * * * * *

2014-15 H5N2 HPAI Incursion

- In December, 2014 in British Columbia, Canada, several commercial flocks were disclosed with H5N2 HPAI.
- In early January, 2015, several falcons in a Raptor Rehabilitation Center located just south of the Canadian Border and passed away. They had fed hunter-harvested ducks to the falcons. H5N2 HPAI was disclosed.
- In rapid succession in January and early February, H5N2 HPAI was disclosed in:
    - Washington: 4 backyard flocks, and 1 game bird flock.
    - Oregon: 2 backyard flock
    - California: 2 commercial poultry flocks
- Map portraying risk of domestic poultry interacting with migratory waterfowl:

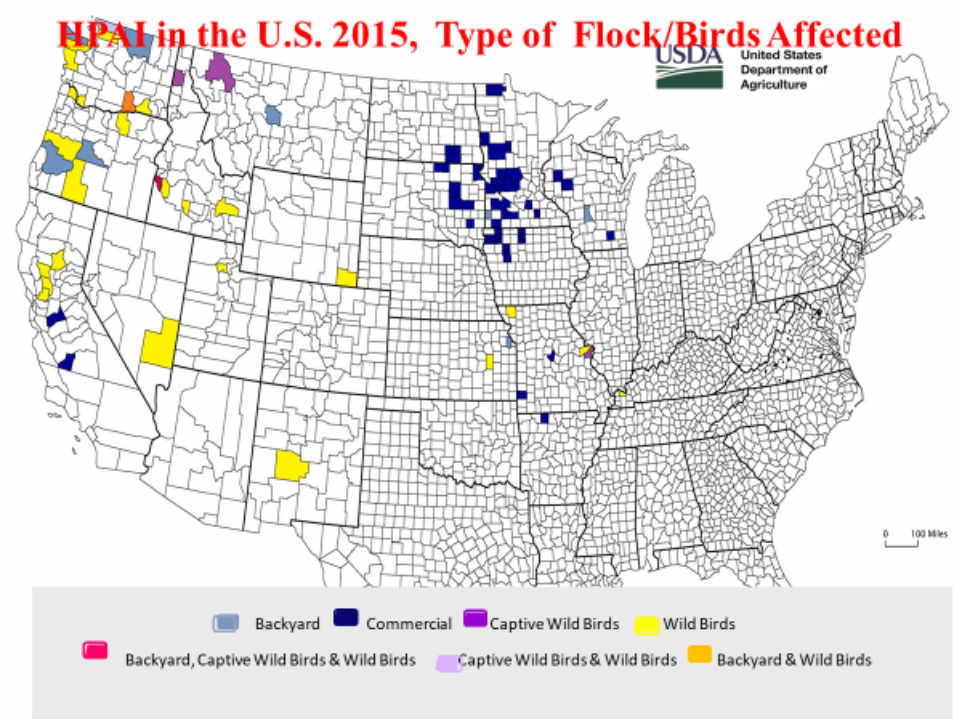


For this reason poultry flocks location on the Pacific Coast are at high risk of exposure to migratory waterfowl. It was the hope that this H5N2 HPAI incursion would be contained in the Pacific Flyway.

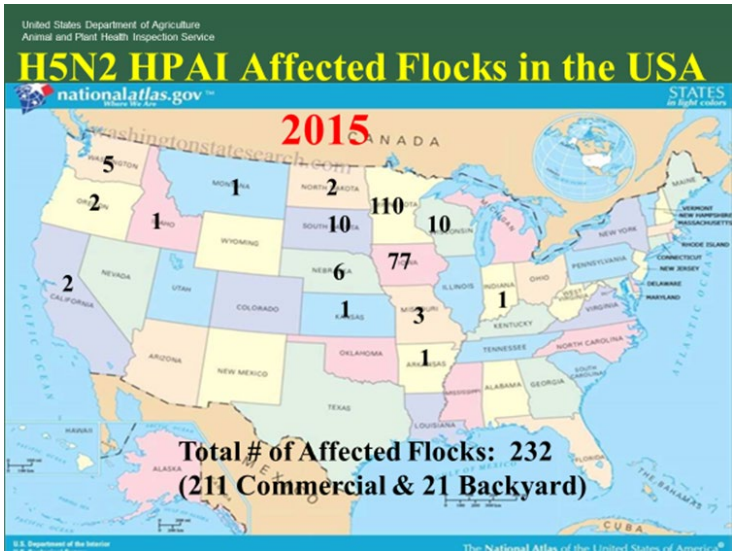
- Those hopes were to no avail because in the first week of March, the first commercial turkey flock was disclosed with the infection in Minnesota.
- In rapid succession 2 commercial turkey flocks in Missouri and one in Arkansas, were disclosed.

HPAI EA/AM H5N2 positive Partial HA/NA sequence is >99% similar to A/northern pintail/Washington/ 40964/20
March 4, NVSL confirmation in Pope Co., MN
March 8, NVSL confirmation in Jasper Co., MO
March 9, NVSL confirmation in Moniteau Co., MO
March 10, NVSL confirmation in Boone Co., AR

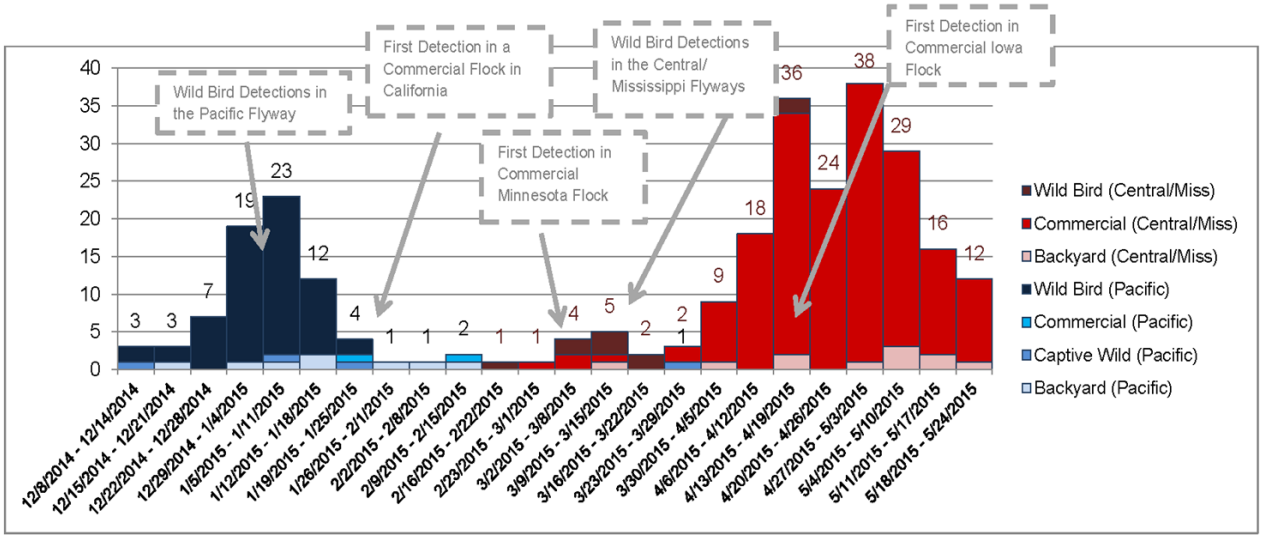
- The infection rapidly spread to (mostly) commercial flocks located in the northern Central Flyway.
- The disease continued to spread until the middle of June when it was declared to be eradicated:
  - 21 states were affected (with positive domestic poultry flocks and positive migratory waterfowl.



  - There were 232 premises positive for HPAI (211 commercial & 21 backyard).
  - ~49.6 M commercial poultry were affected
  - 7.5 M Turkeys
  - 42.1 M chickens
  - ~$1 B was spent by APHIS & the U.S. Congress for the response.
- Map of affected flocks in each state:



- Epidemiological Curve Tracking the Spread of Infection



- Once the infection got into commercial turkey and laying flocks in Minnesota and Iowa (in particular) it spread like wildfire due to human and fomite trafficking between flocks.
- The Four Primary Risk Factors that were identified in an epidemiological study in Minnesota orchestrated by Dr. Brian McCluskey included:
  - Sharing farm equipment between premises and poultry barns is a leading culprit in spreading avian influenza in the outbreaks that have plagued the U.S. poultry industry in 2015.
  - An audited biosecurity plan should be in place.
  - Wild birds were observed in ~38% all the houses.
  - Vehicles moving on and off the poultry farm. investment in equipment/facilities for the washing/spraying of vehicles moving on and off every farm will be worthy of consideration.
- Throughout the outbreak, Wildlife Services continued its surveillance testing of migratory waterfowl. After the final quarantines were released on domestic poultry in the middle of June, between July 1, 2015, and March 18, 2016, that number of affected waterfowl was greatly diminished:
  - Total birds sampled: 45,459

        - Pacific: 11,823
        - Central: 9,202
        - Mississippi: 13,747
        - Atlantic: 10,663
    - American Oceania : 24
- Cases: 2 PCR positives om Mallard ducks:
    - Great Salt Lake, UT
    - Oregon
- This marked the end of the HPAI nightmare of 2015. Since then the funding in Wildlife Services for surveillance testing of migratory waterfowl has dwindled to a trickle of what it was in 2015.

CONCLUSION

- The 2014-15 H5H2 HPAI incursion proved to be the most severe and costly foreign animal disease response in the history of the United States. As the result of this FAD Incursion, ~2 hundred term AHTs and VMOs were hired to combat the anticipated return of HPAI in 2016. This abrupt increase in the roster changed the image of Veterinary Services.
- It is the hope that through the adoption of stringent biosecurity in both the non-commercial (backyard) and commercial poultry industry will prevent a similar disaster.
- Effective February 9, 2016, interim rule went into effect.
    - Provide a formula that will allow APHIS to split such payments between company & egg growers based on the proportion of the production cycle completed.
    - Requiring owners and contractors, unless specifically exempted, to provide a statement that, **at the time of detection of HPAI in their facilities, they had in place and were following a biosecurity plan aimed at keeping HPAI from spreading to commercial premises.**
    - The biosecurity plan installed included NPIP's 14 Biosecurity Principles.

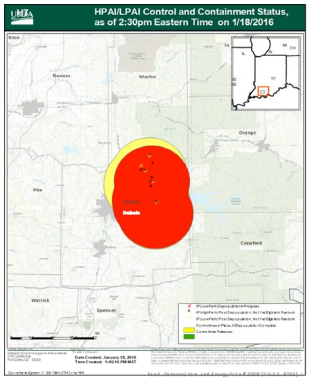
* * * * * * * *

2016 H7N8 HPAI Incursion in South Indiana

- On January 15th, NVSL-confirmed H7N8 HPAI positive results from a commercial turkey flock located in Dubois County, IN.
    - Virus was characterized as high pathogenicity avian influenza (HPAI) per cleavage site analysis).
    - Both the H_7N_8 HPAI and LPAI are highly similar across 6 of 8 gene segments to a recent wild bird detection H_7N_8 LPAI in Kentucky at the end of November (lesser scaup collected 11/28/15).
    - This was the first detection of H7N8 HPAI in domestic species in the United States.

(as per Paul Brennan, IN Dept. of Ag)

- Control Area:



- Of the additional samples collected from premises within the 10 km Control Zone, eight additional commercial turkey flocks were confirmed at NVSL for H7N8 LPAI. (One suspect positive result from a ninth premises could not be confirmed by partial sequencing. Virus isolation results on samples from the ninth premise are negative, but the flock was handled the same as the other H7N8 LPAI flocks.)
- On one farm H7N8 LPAI was converted to HPAI by genetic insertion.
    - January 10, reduced water consumption was observed which continued
    - January 13, mortality in the house jumped to 100
    - When first reported on the morning of January 14, mortality was 500, by the end of the day it had reached 800
- In the Control Zone, surveillance testing included:
    - 65 commercial poultry premises;
    - 1 egg breaking plant;
    - 2 feed mills; 105 small backyard flocks;
    - with a total of 414 K birds were affected.
- In the Surveillance Zone (outside of the 10 km Control Zone, 60 additional commercial flocks
- A total of 233 poultry premises were placed under a regimen of surveillance testing.
- Epidemiological Conclusion:
    - The initial H7N8 Infection introduced onto one (of the 10) farms by a point source (direct or indirect exposure to affected waterfowl).
    - H7N8 was spread to other 8 or 9 farms by contaminated personnel or vehicles.

(as per Paul Brennan, IN Dept. of Ag)

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2017 H7N9 HPAI Incursion in Tennessee

- On March 4, 2017, APHIS confirmed highly pathogenic avian influenza (HPAI) H7N9 in a commercial broiler breeder flock in Lincoln County, Tennessee; this virus is of North American wild bird lineage and is not related to Asian H7N9 HPAI viruses.
- On March 14, 2017, a second commercial broiler breeder flock was confirmed to have HPAI H7N9 in Lincoln County, Tennessee.
- · This second HPAI detection was within the Control Zone (10 km) of the first HPAI detection.
- Pathogen Characterization:
    - NVSL partial sequence attempted directly from swab specimen confirmed H7 HPAI of North American wild bird lineage confirmed 03/04/2017; NA-type pending;
    - The TN H7N9 HPAI is of North American wild bird origin based upon full genome sequencing direct from samples collected from migratory waterfowl.
    - The virus is not related to the China H7N9 viruses - all gene segments are North American wild bird lineage.

- Summary of Avian Influenza Incursions in 2017:

State	Backyard	Commercial	Serotype	Total
Wisconsin	0	1 (turkey)	H5N2 LPAI	1
Tennessee	2	1 (broiler)	H7N9 LPAI	3
	0	2 (broiler)	H7N9 HPAI	2
Alabama	3	3 (broiler)	H7N9 LPAI	6
Kentucky	1	1 (broiler)	H7N9 LPAI	2
Georgia	0	1 (broiler)	H7N9 LPAI	1
Idaho	1 (gamebird)	0	H5N2 LPAI	1
Total	7	9		16

- Epidemiology:
  - LPAI H7N9 was introduced into one or more commercial and or backyard flocks by indirect or direct exposure to migratory waterfowl carrying LPAI H7N9.
  - The LPAI H7N9 mutated to H7N9 in one commercial flock, and it spread to another commercial flock in the same Control Zone in TN, through a breakdown in biosecurity.
  - It spread to other commercial and/or backyard flocks though the movement of contaminated personnel, vehicles, and/or equipment.
- Conclusions:
  - The original incursion occurred through an indirect or direct exposure to migratory waterfowl carrying H7N9 LPAI and it spread to other flocks in adjoining states through a breakdown in biosecurity.
  - THE APPLICATION OF STRINGENT BIOSECURITY is the most important preventive measure that protects against an incursion of LPAI/HPAI, and all infections diseases.

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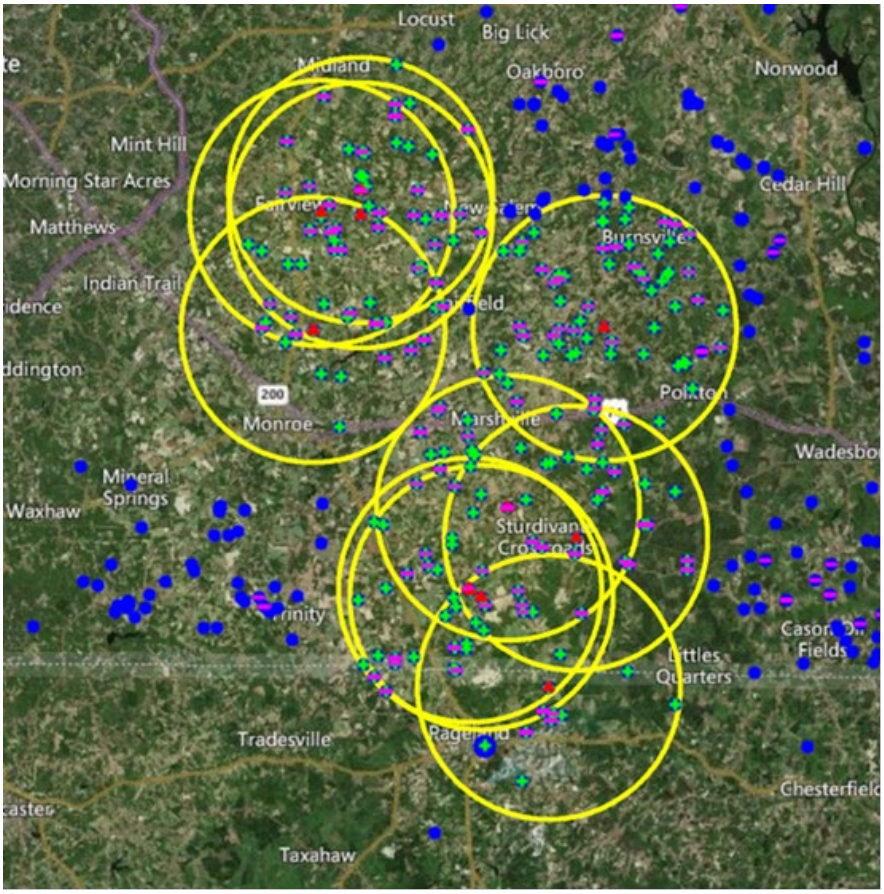
2020 H7N3 HPAI Incursion in South Carolina

- Beginning in March, there were a total of 11 farms affected with LPAI H7N3 involving 305,250 turkeys. Two premises had breeding flocks, and meat birds were at the other 9. Ten of the outbreaks were in North Carolina (eight in Union County, two in Anson County, and one was in Chesterfield County, South Carolina).
- There is one owner of 9 of the 11 affected farms (the 2 turkey breeders are not under his ownership). He owns or contracts with 50 turkey farms in the area. He sells his birds to multiple companies.
- In early April, the presence of HPAI of the H7N3 serotype was found in a flock of 34,160 commercial turkeys in Chesterfield County, South Carolina.
- Dr. Mia Torchetti, NVSL, DNA fingerprinted the N7H3 virus from each to determine the index premise from which there was lateral spread.
- Control Zones:
  All affected flocks were located within a 50 mile radius.



- Surveillance testing was completed and the quarantine was released without further spread.

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EPIDEMIOLOGY OF POULTRY DISEASES

As VS Epidemiologists, one of our primary endeavors is the conduction of epidemiological investigations. After you have orchestrated the definitive diagnosis of one of the aforementioned diseases (especially HPAI/H5/H7 or vND, but you may also have the opportunity to investigate, and determine the source of Infectious Laryngotracheitis and the Mycoplasmoses, which are quarantinable and economically important in sone jurisdictions). The manifestation of infection can be subclinical, but most of the diseases of importance my involve significant clinical signs and high mortality. Epidemiology tries to find answers to specific questions:

1. Why is a specific disease on a specific farm, or in a specific chicken/turkey house, community, or even region?
2. What was the source of the infection?
3. Was its manifestation the result in a deficiency of the biosecurity measures that have been in place?
4. What can be done to control/eradicate the current disease problem, and to prevent a future incursion of the disease?

Credible veterinary epidemiological studies require knowledge in a variety of fields beyond "veterinary science." They require knowledge of diseases, infectious agents non-infectious factors, interactions of potential causes, diagnostic techniques husbandry, feed, housing, economics, mathematics, statistics…the list is continuous. In this treatise, I have tried to provide the rudimentary elements (from history, epidemiology,

diagnostics, clinical signs, etc.) to give you a fundamental knowledge. The study of epidemiology is a "collegiate course" in itself. It's a matter of determining the "who," "what," "where," "why," "when," and "how," answers that we seek in an epidemiological investigation.

I have attached Appendices A thru C, which are epidemiological questionnaires been used in the last two incursions of HPAI and vND. They will provide guidance in conducting and epidemiological investigation. In the recent H7N3 HPAI incursion in South Carolina. Due to the COVID-19 Pandemic restrictions the questionnaires were presented to the producers over the internet, which was probably adequate in this instance. However, an on-site visit to the affected premise is usually required to complete a credible investigation.

* * * * * * * *

CONCLUSIONS

It is important that you become familiar with USDA's Reportable Disease List as well as that of the local jurisdiction in which you work.

As VS Epidemiologists we may be asked to consult on disease problem in a backyard or commercial poultry operation. Ten of the 15 diseases on USDA's Reportable Disease list have a primary or secondary respiratory component in their clinical signs. Our primary concern are the two foreign animal diseases (vND and HPAI/H5/H7) but all of them are clinically indistinguishable and requiring a laboratory diagnosis in order to arrive at a definitive diagnosis. It is essential that a FAD be diagnosed immediately in order to minimize the spread of the infection which results in severe economic repercussions in international trade.

It is important to promote the application of **stringent biosecurity** measures in any production system as the primary preventive measure of most infectious diseases.

APPENDIX A:

Animal and Plant Health Inspection Service
Veterinary Services

HPAI/H5/H7 Investigation - Questionnaire

INSTRUCTIONS

The purposes of these investigations are to assess potential pathways of initial introduction of HPAI viruses onto commercial poultry operations and potential lateral transmission routes of HPAI viruses from infected premises to noninfected premises.

Following confirmation of an HPAI virus introduction into a commercial flock, an investigation should be initiated as soon as possible, no later than 1 week following detection. The investigator(s) assigned should be integrated into other response activities but their primary focus is on completion of the introduction investigation.

The investigation form provided is a guide for conducting a systematic and standardized assessment of potential pathways of initial virus movement onto the farm and potential movement of the virus off the farm. All sections of the form should be completed through direct conversation with the individual(s) most familiar with the farm's management and operations and questions are to be answered for the period 2 weeks prior to the detection of HPAI. Where applicable, direct observation of the biosecurity or management practice asked about should be conducted. This is not a box-checking exercise but an in-depth review of the current biosecurity and management practices and exposure risks on an affected farm. For example, direct observation of the farm employee donning and doffing procedures and compliance with company biosecurity practices is more important than checking the box on the form that indicates workers wear coveralls into the poultry houses. Investigators are encouraged to take notes and include them with the investigation form when completed. An investigation form should be completed for the infected house or farm and **at least one** noninfected house or farm within the same complex as near as possible to the index infected flock.

Date: _____

Interviewer name/organization: _____

Interviewee name/organization: _____

A. PREMISES INFORMATION

Farm name: _____

Farm address: _____

Farm (premises) ID: _____ County: _____

Township: _____ Range: _____ Section: _____

Is facility enrolled in NPIP? ☐$_1$ Yes ☐$_3$ No

B. PREMISES CONTACT INFORMATION

1.Contact name: _____

Phone: _____ Cell phone: _____ Email:_____

2.Contact name: _____

Phone: _____ Cell phone: _____ Email: _____

Contact name: _____

Phone: _____ Cell phone: _____ Email: _____

3.Flock Veterinarian: _____

Phone: _____ Cell phone: _____ Email: _____

C. PREMISES DESCRIPTION

1. Poultry type: ☐$_1$ Broiler ☐$_2$ Layer ☐$_3$ Turkey ☐$_4$ Other (specify: _____)

2. Production type: ☐$_1$ Meat ☐$_2$ Egg ☐$_3$ Breeding ☐$_4$ Other (specify: _____)

3. Age: ☐$_1$ Multiple age ☐$_2$ Single age

4. Sex: ☐$_1$ Hen ☐$_2$ Tom ☐$_3$ Both

5. Flock size: _____ # birds

6. Facility type: *[Check all that apply]*
   ☐ Brood
   ☐ Grow
   ☐ Other (specify: _____)
   ☐ Both brooder & grower houses are present on the same premises
   ☐ Breeder
   ☐ Commercial

7. If brooder and grower houses are present on the same premises, are there multiple stages of management (brooding and growing), in the same house?
   ☐$_1$ Yes ☐$_3$ No

8. Farm capacity _____ # birds
   Number of barns _____ # barns
   Barn capacity _____ # birds

9. What is the **primary** barn type/ventilation: *[Check one only.]*
   ☐$_1$ Curtain sided
   ☐$_2$ Environmental control
   ☐$_3$ Side doors
   ☐$_4$ Other (specify: _____)

10. Are cool cell pads used? ☐$_1$ Yes ☐$_3$ No

    If Yes, what is the source of water for these pads? _____

11. Distance in yards of closest body of water near farm: _____ yards

12. Water body type: *[Check all that apply.]*
    ☐ Pond
    ☐ Lake
    ☐ Stream
    ☐ River
    ☐ Other (specify: _____)

13. What other types of animals are present on the farm?
    a. Beef cattle ☐$_1$ Yes ☐$_3$ No

b. Dairy cattle ☐1 Yes ☐3 No
c. Horses ☐1 Yes ☐3 No
d. Sheep ☐1 Yes ☐3 No
e. Goats ☐1 Yes ☐3 No
f. Pigs ☐1 Yes ☐3 No
g. Dogs ☐1 Yes ☐3 No
h. Cats ☐1 Yes ☐3 No
i. Poultry or domesticated waterfowl ☐1 Yes ☐3 No
j. Other (specify: _____) ☐1 Yes ☐3 No

14. What is the **primary** water source for poultry? *[check only one]*
☐1 Municipal
☐2 Well
☐3 Surface water (e.g., pond)
☐4 Other (specify: _____)

15. Is water treated prior to delivery to poultry? ☐1 Yes ☐3 No
If Yes, how is it treated and with what? _____

D. FARM BIOSECURITY

1. Is there a house with a family living in it on the property? ☐1 Yes ☐3 No

2. Is there a common drive entrance to farm and residence? ☐1 Yes ☐3 No

3. Do you have signage of "no admittance" or "biosecure area" on this property? ☐1 Yes ☐3 No

4. Is there a gate to this farm entrance? ☐1 Yes ☐3 No

5. Is the gate secured/locked? ☐1 Yes ☐3 No
If Yes, what hours is it secured? _____

6. Is the farm area fenced in? ☐1 Yes ☐3 No

7. How frequently is vegetation mowed/bush hogged on the premises? _____ times/month

8. Is facility free of debris/clutter/trash piles? ☐1 Yes ☐3 No

9. Is there a wash station/spray area available for vehicles? ☐1 Yes ☐3 No
If Yes, what disinfectant is used? _____

10. Is there a designated parking area for workers and visitors
away from the barns/pens? ☐1 Yes ☐3 No

11. Is there a changing area for workers? ☐1 Yes ☐3 No
Do they shower before entering? ☐1 Yes ☐3 No

12. Do workers don dedicated laundered coveralls before entering
each house on the premises? ☐1 Yes ☐3 No

13. Do worker wear rubber boots or boot covers in poultry houses? ☐1 Yes ☐3 No

14. Are the barn/pen doors lockable? ☐1 Yes ☐3 No
Are they routinely locked? ☐1 Yes ☐3 No

15. Are foot pans available at barn/pen entrances? ☐1 Yes ☐3 No
Are they in use? ☐1 Yes ☐3 No

16. Are foot baths dry (powdered or particulate disinfectant)? ☐1 Yes ☐3 No

17. Are foot baths liquid disinfectant? ☐1 Yes ☐3 No

18. Frequency foot pan solutions are changed? _____ times/month

What disinfectant is used? _____

19. Is there an entry area in the barns/pens before entering the bird area? ☐1 Yes ☐3 No

20. What pest and wildlife control measures are used on this farm?
    a. Rat and mouse bait stations ☐1 Yes ☐3 No
    b. Bait stations checked at least every 6 weeks ☐1 Yes ☐3 No
    c. Fly control used ☐1 Yes ☐3 No
    If Yes, type and frequency: _____
    d. Houses are bird proof ☐1 Yes ☐3 No
    e. Wild birds seen in house ☐1 Yes ☐3 No
    If Yes, type, number and frequency: _____
    f. Raccoons, possums, foxes seen in or around poultry houses ☐1 Yes ☐3 No
    g. Wild turkeys, pheasants, quail seen around poultry ☐1 Yes ☐3 No

21. Are biosecurity audits or assessments (company or third party) conducted on this farm? ☐1 Yes ☐3 No
If Yes, when was the last audit or assessment conducted? _____
(Obtain a copy of the result of the audit or assessment if available.)

22. Has this farm been confirmed positive for HPAI? ☐1 Yes ☐3 No

E. FARM HELP/WORKERS

1. Total number of persons working on farm _____ #

2. Number of workers living on the farm premises who are:
    a. Family _____ #
    b. Nonfamily _____ #

3. Workers are assigned to: *[check only one]*
    ☐1 Entire farm
    ☐2 Specific barns/areas

4. Do the workers have a common break area? ☐1 Yes ☐3 No
If Yes, location: _____

5. Are workers employed by other poultry operations? ☐1 Yes ☐3 No

6. How often are training sessions held on biosecurity for workers? _____ times/year

7. Are family members employed by other poultry operations or processing plants? ☐1 Yes ☐3 No

   If Yes, poultry operation or processing plant: _____

8. Do part-time/weekend help and other extended family members on holidays and vacations? ☐1 Yes ☐3 No

9. Are workers (full & part-time) restricted from being in contact with backyard poultry? ☐1 Yes ☐3 No

   How is this verified?_____

10. Are workers (full & part-time) restricted form hunting migratory waterfowl? ☐1 Yes ☐3 No
    If "No," are they counselled on the prudent biosecurity precautions that must be taken? ☐1 Yes ☐3 No

    How is this communicated? _____

F. FARM EQUIPMENT

Is the equipment used on this premises farm specific, under joint ownership that remains on this premises, or under joint ownership and used on other farm premises? A list of equipment follows.

1. Company vehicles/trailers:
   Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

2. Feed trucks (excess feed):
   Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
   Dates: _____

3. Gates/panels:
   Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____

   Dates: _____

4. Lawn mowers:
   Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____

   Dates: _____

5. Live haul loaders:

Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

6. Poult trailers: Farm specific?
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

7. Pre-loaders:
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____

Dates: _____
Describe pre-loader cleaning and disinfection procedures:

_____

_____

8. Pressure sprayers/washers:
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

9. Skid-steer loaders:
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

10. Tillers:
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

11. Trucks:
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

12. Other equipment: _____
Farm specific? ☐1 Yes ☐3 No
If No, by whom is equipment jointly used: _____
Dates: _____

G. LITTER HANDLING

1. Litter type: _____

2. Supplier/source: _____

3. Is a litter shed present? ☐1 Yes ☐3 No
4. Do you do partial cleanouts? ☐1 Yes ☐3 No
   If Yes, give dates of last partial cleanout: _____

5. Date of last cleanout: _____ date
   Frequency of cleanout: _____ times/month

6. Who does the cleanout?
   ☐1 Grower
   ☐2 Contractor
   If contractor, name and location_____

7. Litter is disposed of:
   ☐1 On farm
   ☐2 Taken off site
   If taken offsite, name and location: _____

H. DEAD BIRD DISPOSAL

1. Approximate normal daily mortality _____ # birds

2. How is daily mortality handled?
   a. On-farm: Burial pit/incinerator/composted/other (specify: _____)

   b. Off-farm: Landfill/rendering/other (specify: _____)

   c. Off-farm disposal performed by: Owner/employee/other (specify: _____)
   d. If burial or compost pits are used, are carcasses covered with soil on a daily basis? ☐1 Yes ☐3 No

3. Contact name of company or individual responsible for disposal:_____

   _____
   If rendering is used, include location of carcass bin on the farm map.

4. What is the pickup schedule? _____

5. Does the carcass bin have a cover? ☐1 Yes ☐3 No
   Is it routinely kept closed? ☐1 Yes ☐3 No

I. FARM VISITORS

1. How many visitors do you have on a daily basis? _____ #

2. Is there a visitor log to sign in? ☐1 Yes ☐3 No
   Is it current? ☐1 Yes ☐3 No

3. Do you provide any outer clothing to visitors entering the farm? ☐1 Yes ☐3 No
If Yes, identify items of clothing provided: _____

4. Mark the following services that were on the farm when this flock was on the farm. List date of service and name of person (or contract company) and if they had contact with the birds.

Service		**Dates**	**Name Contact?**	
Service person	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No
Vaccination crew	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No
Moving crew (moving from brood to grow, or pullet house to layer house)	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No
Processing plant load out	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No

Load-out crew (positive flock) ☐1 Yes ☐3 No ☐Yes ☐No
If load-out took more than one night, was returning crew the same crew? ☐1 Yes ☐3 No

Truck #/#'s _____

Trailer #/#'s _____

What plant did flock go to? _____

Load-out crew (flock previous to positive flock)
☐Yes ☐No _____ _____ ☐1 Yes ☐3 No

If load-out took more than one night, was returning crew the same crew? ☐1 Yes ☐3 No

Truck #/#'s _____

Trailer #/#'s _____

What plant did flock go to? _____

Poult delivery	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No
Rendering pickup	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No
Litter services	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No
Cleanout services	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No

Equipment shared/rented/loaned/borrowed (each of the categories of visitor is likely to be accompanied by equipment of some sort or another)
☐Yes ☐No _____ _____ ☐1 Yes ☐3 No

Feed delivery	☐Yes ☐No	_____	_____	☐1 Yes ☐3 No

5. Who makes sure covers are closed after delivery? _____

6. Are feed covers kept closed? ☐$_1$ Yes ☐$_3$ No

J. WILD BIRDS

1. Do you see wild birds around your farm? ☐$_1$ Yes ☐$_3$ No
   If Yes, what type of birds? *[Check all that apply.]*
   ☐ Waterfowl
   ☐ Gulls
   ☐ Small perching birds (sparrows, starlings, swallows)
   ☐ Other water birds (egrets, cormorants)
   ☐ Other _____

2. Do you see birds all year round? ☐$_1$ Yes ☐$_3$ No
   If Yes, what type of birds? _____

3. Is there seasonality to the presence of some types of birds? ☐$_1$ Yes ☐$_3$ No
   If Yes, what type of birds and what seasons do you see them? _____
   _____

4. Where are wild birds seen in relation to the farm?
   ☐$_1$ On adjacent habitats away from facilities and equipment (identify location of habitat on photos)
   ☐$_2$ On the farm but not in the barns (identify facilities or equipment birds have contact with)
   ☐$_3$ On the farm and sometimes in the barns (identify facilities or equipment birds have contact with)

K. NARRATIVE/COMMENTS

<u>FARM DIAGRAM -</u>Attach a download from satellite imagery if possible. In addition, draw a simple schematic map of the farm site centering with the poultry houses/pens. Identify where the HPAI positive flocks were housed. Also include: fan banks on houses, residence, driveways, public roads, bodies of water, feed tanks, gas tanks, out buildings, waster dumpsters, electric meters, dead bird disposal, parking areas, other poultry sites. Digital photographs, if allowed, are excellent supporting documentation.

North

APPENDIX B

Animal and Plant Health Inspection Service

Veterinary Services

HPAI/LPAI Epidemiological Investigation Form (Backyard, Exposed, or Trace-Out Non-Commercial Recipient Flock)

BACKGROUND:
The purpose of this investigation is/are:

- To assess potential pathways of initial exposure of HPAI/LPAI viruses into backyard/non-commercial poultry operations;
- Reduce the potential for lateral transmission routes of HPAI/LPAI viruses from the exposed/infected premises to non-infected premises; and,
- Enhance the backyard/non-commercial flock owner's awareness of the importance of biosecurity in preventing the introduction of infectious diseases into his/her flock.

Following notification of introduction of exposed birds, or contaminated equipment, of HPAI/LPAI virus into a flock, an investigation should be initiated as soon as possible, no later than 1 week following detection. The investigator(s) assigned should be integrated into other response activities but their primary focus is on completion of the introduction investigation.

The investigation form provided is a guide for conducting a systematic and standardized assessment of potential pathways of initial virus movement onto the farm and potential movement of the virus off the farm. All sections of the form should be completed through direct conversation with the individual(s) most familiar with the farm's management and operations and questions are to be answered for the period 2 weeks prior to the detection of HPAI. Where applicable, the direct observation of the biosecurity or management practices asked about should be conducted. This is not just a box-checking exercise but an in-depth review of the current biosecurity and management practices and exposure risks on an affected farm. Investigators are encouraged to take notes and include them with the investigation form when completed.

Date: _____

Interviewer name/organization: _____

Interviewee name/organization (if applicable): _____

A. PREMISES INFORMATION

Farm/Backyard Premises name: _____

Farm/Backyard address: _____

GPS Coordinates of affected flock: Lat: _____ Long: _____

Farm (premises) ID: _____ County: _____

Township: _____ Range: _____ Section: _____

Is facility enrolled in NPIP? ☐ Yes ☐ No

If so, in what in what Subpart of NPIP is flock enrolled? _____ Number of years of enrollment: ____

B. PREMISES CONTACT INFORMATION

1.Contact name: _____

Phone: _____ Cell phone: _____ Email: _____

2.Contact name: _____

Phone: _____ Cell phone: _____ Email: _____

3. Contact name: _____

Phone: _____ Cell phone: _____ Email: _____

4.Flock Veterinarian: _____

Phone: _____ Cell phone: _____ Email: _____

5.Probable source of Exposure: _____(See Item #6)

6. Indicate the nature of presumed exposure:
   a. Trace out from an affected flock……☐ Yes ☐ No
   b. Exposure to a neighboring affected flock…… ☐ Yes ☐ No
   c. Potentially contaminated visitors……☐ Yes ☐ No
   d. Contaminated visitors who own poultry……☐ Yes ☐ No

e. Untreated water source…… ……. ☐ Yes ☐ No
f. Goats…… ☐ Yes ☐ No
f. Pigs …… ☐ Yes ☐ No
g. Dogs……☐ Yes ☐ No
h. Cats…… ☐ Yes ☐ No
i. Rodents……. ☐ Yes ☐ No
j. Vehicles or equipment…… ☐ Yes ☐ No
k. Wind-driven (aerosol) from infected neighboring flock…… ☐ Yes ☐ No
l. Wild birds (migratory waterfowl or others)…… ☐ Yes ☐ No
l. Unknown……... ☐ Yes ☐ No
m. Not Applicable (N/A)…… ☐ Yes ☐ No

7. Is/are owner(s) aware of infectious diseases that can be transmitted to:
a. To their backyard flock (such as mycoplasmosis, A.I., or I.L.T.)?.... ☐ Yes ☐ No
b. To humans (such as salmonellosis, colibacellosis, influenza? …….. ☐ Yes ☐ No

C. FARM/BACKYARD EQUIPMENT

Is the equipment used on this premises farm/backyard specific, under joint ownership that remains on this premises, or under joint ownership and used on other farm/backyard premises? A list of equipment follows.

1. Farmyard vehicles/trailers:
Farm/backyard specific?..... ☐ Yes ☐ No

If no, with whom is equipment shared: _____

Dates: _____

2. Feed/hauling/delivery trucks:
Farm/backyard specific?..... ☐ Yes ☐ No

If no, by whom are trucks shared: _____

Dates: _____

3. Lawn mowers/bush hogs/tillers are used: ……☐ Yes ☐ No
Farm/backyard specific?..... ☐ Yes ☐ No

If no, with whom is equipment shared: _____

Dates: _____

C. PREMISES DESCRIPTION

1. Poultry type: ☐ Broiler ☐ Layer ☐ Turkey ☐ Mixed ☐ Guinea Fowl
☐ Waterfowl (Ducks & Geese) ☐ Pigeons ☐ Game Birds

2. Production type: ☐ Meat only ☐ Table Egg ☐ Both Egg & Meat ☐ Hobby
☐ Exhibition ☐ 4-H or FFA Project

3. Age: ☐ Multiple age ☐ Single age

4. Sex: ☐ Hen ☐ Tom/gander/drake/rooster ☐ Both

5. Flock size: …… # birds _____

6. Distance in yards of closest body of water near farm: _____ (yards or miles)

7. Water body type: *[Check all that apply.]*
   - ☐ Pond
   - ☐ Lake
   - ☐ Stream
   - ☐ River
   - ☐ Marsh
   - ☐ Other (specify: _____)

8. What other types of animals are present on premises or on adjacent premises?
   - a. Beef cattle ☐ Yes ☐ No
   - b. Dairy cattle ☐ Yes ☐ No
   - c. Horses ☐ Yes ☐ No
   - d. Sheep ☐ Yes ☐ No
   - e. Goats ☐ Yes ☐ No
   - f. Pigs ☐ Yes ☐ No
   - g. Dogs ☐ Yes ☐ No
   - h. Cats ☐ Yes ☐ No
   - i. Poultry or domesticated waterfowl ☐ Yes ☐ No
   - j. Other (specify: _____) ☐ Yes ☐ No

9. What is the primary water source for poultry? *[Check one only.]*
   - ☐ Municipal
   - ☐ Well
   - ☐ Surface water (e.g., stream or pond)
   - ☐ Surface water (e.g., irrigation)
   - ☐ Other (specify: _____)

10. Is water treated prior to delivery to poultry? ☐ Yes ☐ No
    If yes, how is it treated and with what? _____

11. Source of feed:
    - ☐ Livestock feed store or mill
    - ☐ Home Produced (table scraps, etc,)
    - ☐ Other (specify: _____)

12. Feed storage:
    - ☐ Closed (rain and rodent-proof)
    - ☐ Uncovered (open)

D. FARM/BACKYARD BIOSECURITY

1. Is the farm/backyard area fenced in? ☐ Yes ☐ No

2. How frequently is vegetation mowed/bush hogged on the premises? _____ times/month

3. Is facility free of debris/clutter/trash piles? ☐ Yes ☐ No

4. Has this premises ever been confirmed positive for HPAI or LPAI? ☐ Yes ☐ No

5. Are poultry free roaming?……☐ Yes ☐ No

6. Are poultry confined at night?…….
☐ Yes ☐ No

7. Is there a house with a family living in it on the property?.....….. ☐ Yes ☐ No

8. Is there a gate to the chicken pen(s)?..... ……☐ Yes ☐ No

9. Is the gate secured/locked?..... ☐ Yes ☐ No

If yes, what hours is it secured? _____

10. Is there a designated parking area for visitors?..... ……. ☐ Yes ☐ No

11. Is there rubber boots or boot covers for visitors/owners for poultry pen/houses?. ☐ Yes ☐ No

12. Is there designated laundered clothing (cover-alls) for entry into the poultry pen?.☐ Yes ☐ No

13. Are foot pans available at barn/pen entrances?..... ☐ Yes ☐ No

Are they in use? ☐ Yes ☐ No

14. Are foot baths dry (powdered or particulate disinfectant)?..... ☐ Yes ☐ No

Type of disinfectant:_____

15. Are foot baths liquid disinfectant?..... ☐ Yes ☐ No
Type of disinfectant:_____

16. Frequency foot pan disinfectants are changed? _____ times/week/month

17. Do owners wear rubber/latex/nitrile gloves into the poultry pen/houses?..... ☐ Yes ☐ No

18. Do owners/visitors wash their hands immediately before entering pen?..... ☐ Yes ☐ No

19. Do owners/visitors wash their hands immediately after departing pen?..... ☐ Yes ☐ No

20. Owners/visitors are prohibited from eating food while in poultry pen? ……. ☐ Yes ☐ No

21. What pest and wildlife control measures are used on this farm?

a. Rat and mouse traps or bait stations…… ☐ Yes ☐ No

b. Bait stations checked at least every 6 weeks…… .. ☐ Yes ☐ No

c. Fly control used?..... ☐ Yes ☐ No

If yes, type and frequency: _____

d. Houses are bird proof?..... ☐ Yes ☐ No

e. Wild birds seen in house?..... ☐ Yes ☐ No
If yes, type, number and frequency _____

f. Raccoons, possums, foxes seen in or around poultry houses?..... ☐ Yes ☐ No

g. Wild turkeys, pheasants, quail seen around poultry? ……☐ Yes ☐ No

E. LITTER HANDLING

1. Litter type: _____

2. Supplier/source: _____

3. Date of last clean-out of house (if applicable) _____

3. Litter is disposed of:
☐ On farm (in garden or field)
☐ Taken off site
If taken offsite, name and location: _____

F. DEAD BIRD DISPOSAL

1. Approximate normal daily mortality _____ # birds

2. How is daily mortality handled?
a. On-farm: burial pit/incinerator/composted/other (specify: _____)
b. Off-farm: landfill/rendering/other (specify: _____)
c. Off-farm disposal performed by: Owner/employee/other (specify: _____)
d. If burial or compost pits are used, are carcasses covered with soil on a daily basis? ☐ Yes ☐ No

e. Carcasses are double bagged and designated for public land fill……☐ Yes ☐ No

f. Diagnoses are sought for unexplained mortalities?..... ☐ Yes ☐ No

If so, from whom:
1. Private Veterinarian ……☐ Yes ☐ No
2. County Extension Agent……..☐ Yes ☐ No
3. Neighbor……...☐ Yes ☐ No
4. Local feedstore…….☐ Yes ☐ No
5. Internet……☐ Yes ☐ No
6. Poultry Health Specialist (at college or gov't))…….☐ Yes ☐ No
7. Submission of specimens to diagnostic lab……☐ Yes ☐ No
8. If none of the above, from whom_____

G. WILD BIRDS

1. Do you see wild birds around your farm/backyard? ☐ Yes ☐ No
   If yes, what type of birds? *[Check all that apply.]*
   ☐ Waterfowl
   ☐ Gulls
   ☐ Small perching birds (sparrows, starlings, swallows)
   ☐ Other water birds (egrets, cormorants)
   ☐ Other _____

2. Do you see birds all year round? ☐ Yes ☐ No
   If yes, what type of birds? _____

3. Is there a seasonality to the presence of some types of birds? ……..☐ Yes ☐ No
   If yes, what type of birds and what seasons do you see them? _____
   _____

4. Where are wild birds seen in relation to the farm/backyard?
   ☐ On adjacent habitats away from facilities and equipment (identify location of habitat on photos)
   ☐ On the farm but not in the barns (identify facilities or equipment birds have contact with)
   ☐ On the farm and sometimes in the barns (identify facilities or equipment birds have contact with)

Investigator's Comments/Observations:_____

_____.

_____

Owner's Comments:

_____

Investigating Veterinarian's Signature:_____ Date:_____

Owner's Signature: _____ Date: _____

FARM/BACKYARD DIAGRAM - Draw a simple schematic map of the backyard premises in relation to the neighbors, lakes, ponds, etc., site centering with the poultry houses/pens. Identify where the traced birds or eggs were housed.

NORTH

APPENDIX C:

QUESTIONNAIRE California Department of Food and Agriculture CA VND 2018

Non-Commercial Premises Virulent Newcastle Disease Epidemiology Questionnaire

Investigator name: _____Date of Investigation: _____/_____/_____

Investigator name: _____

Quarantine # _____ Date Quarantine Issued: _____/_____/_____

1.Name of Premises Owner:

_____
(First) (MI) (Last)

2. Premises Address (location of birds): _____

_____

Latitude: _____ Longitude: _____

3.Premises Owner Telephone #:

a. Mobile: _____

b. Home: _____

c. Other: _____

If Premises Owner is the Bird Owner skip to Question 7

Name of Bird Owner:

_____
(First) (MI) (Last)

5. Bird Owner Address: _____

_____

6. Bird Owner Telephone #: _____

7. Other than the interviewee, how many other owners with birds are on this premises: #_____

8. How many birds do you have on the premises today? # _____

9. What percent of the adult chickens are:

a) Roosters %_____

b) Hens %_____

10. Which of the following birds are on the premises? Complete table below.

Type of Bird:
Adults _____

Young birds _____

Total # _____

Backyard Poultry _____

Exhibition Birds _____

Gamefowl _____

Ducks/Geese _____

Other Specify _____

11. Which of the following animals are on the premises (potential fomites)?
a) Livestock (Horses, Cattle, Swine, Sheep, Goats) □1 Yes □3 No
b) Dogs/Cats □1 Yes □3 No
c) Other (specify _____) □1 Yes □3 No

12. Which of the following housing types are used to house birds?
a) Inside the home □1 Yes □3 No
b) Outdoor open top poultry pen or enclosure □1 Yes □3 No
c) Outdoor cages or coops - fully enclosed □1 Yes □3 No
d) Individually tethered □1 Yes □3 No
e) Free range □1 Yes □3 No
f) Other (Specify_____) □1 Yes □3 No

13. Has there been an increase in illness in your birds on your premises? □1 Yes □3 No
a) If yes, how many days ago did the birds first show signs of illness: _____days

Which of the following clinical signs of illness have you observed? Check all that apply.
a) Not eating □1 Yes □3 No
b) Coughing/gasping □1 Yes □3 No
c) Depressed □1 Yes □3 No
d) Twisting of the neck □1 Yes □3 No

e) Paralysis □1 Yes □3 No
f) Diarrhea □1 Yes □3 No
g) Swellings around the eyes and neck □1 Yes □3 No
h) Sudden death □1 Yes □3 No
i) Other (specify_____) □1 Yes □3 No

14. Have there been any deaths in your birds on this premises during the past 30 days? □1 Yes □3 No

a) If yes, when did the first bird die? _____/___/_____

b) If yes, how many birds died in the first 7 days? # _____

c) If yes, how many birds have died in the past 7 days? # _____

15. Do you keep any birds at another premises? □1 Yes □3 No

a) If yes, where are the birds housed?

_____

_____

16. Have you brought new birds onto this premises during the past 30 days? □1 Yes □3 No If Yes, list date and name the source and location of the new birds:

Date Source/Location ___/___/___a _____

b ___/___/___c _____

d ___/___/___e _____f

17. Have any of the following had contact with your birds, feed or water sources on your property in the last 30 days?
a) Wild birds (e.g., pigeons, doves, sparrows) □1 Yes □3 No
b) Neighborhood/community chickens □1 Yes □3 No
c) Wild animals □1 Yes □3 No

18. Have any of your birds left these premises during the last 30 days? □1 Yes □3 No

If Yes, for what purposes listed below were the birds moved?

Purpose:
Sale ____
Show ____
Competition ____
Veterinary care ____
Gift/trade ____
Other: _____

Date Destination (City/State): _____

# of birds

Other Specify:

If Yes, did any birds leave and then return to these premises? □1 Yes □3 No

19. Do you give away or sell eggs from this premises? □1 Yes □3 No

20. Do your neighbors have birds? □1 Yes □3 No

If No, skip to Question 23.

If Yes, please note location(s) on the map at the end of the questionnaire.

21. When not cooped, do your birds ever visit the neighbor's property? □1 Yes □3 No

22. Do your neighbor's birds ever come onto your property? □1 Yes □3 No

a) If Yes, do the neighbors birds have contact with your birds? □1 Yes □3 No

23. Do you have family members or close friends who own/keep birds? □1 Yes □3 No

If Yes, do any of the following situations occur (evaluating direction of exposure):

a) Your family or friends handle birds when they visit. □1 Yes □3 No

b) When visiting family/friends do you handle their birds. □1 Yes □3 No

24. What is the name and location of the store(s) where you get feed and supplies for your birds?

Name Location (City) _____

a _____

b _____

c _____

d _____

e _____

25. Have the birds on your premises today been vaccinated for Newcastle vaccine? □1 Yes □2 Unsure □3 No

Vaccine does not protect against disease!

a) If Yes, at what age(s) were your birds vaccinated with Newcastle vaccine?

26. Have you seen any dead wild birds on your premises □1 Yes □3 No in the last 30 days?

If Yes, what type of wild bird(s)?

a _____

b _____

c_ _____

Additional comments, observations and leads:

Insert Google Maps Image of the premises or draw a map and specify bird locations. Please indicate which neighbors, if any, have birds.

I _____certify that I have _____birds on / / @

_____

(owner signature) (number) (date and time)

_____

Investigator's Name Signature Date