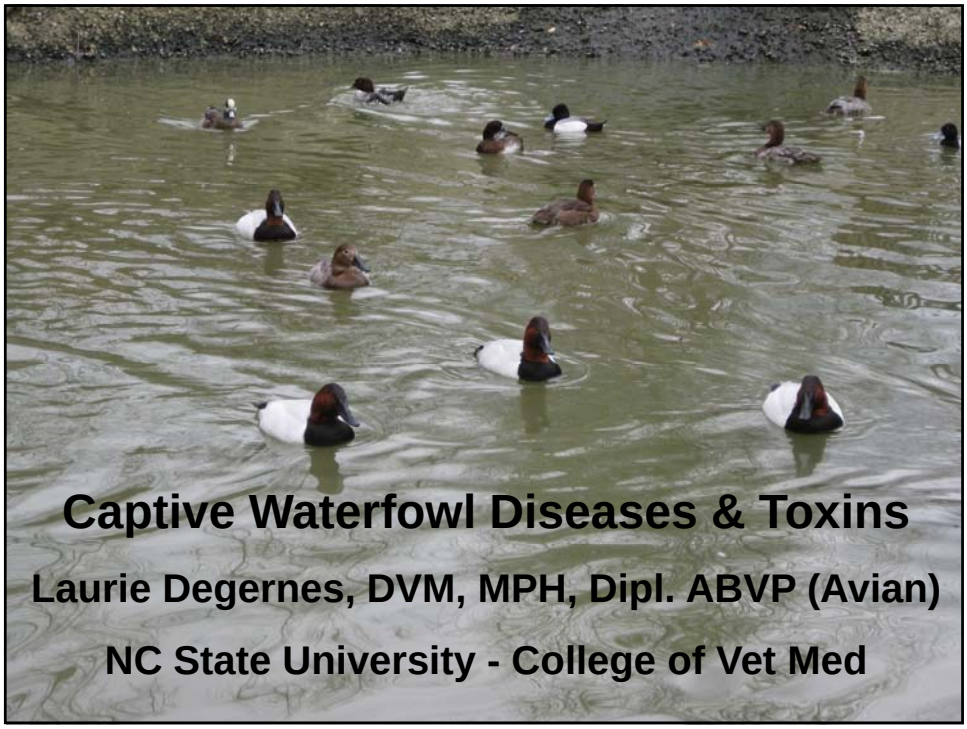

Captive Waterfowl Diseases & Toxins

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Overview

- Bacterial Dz (cholera)
- Viral Dz (duck viral enteritis)
- Fungal Dz (aspergillosis)
- Biotoxins (botulism) & Heavy Metal Toxins (lead, zinc)

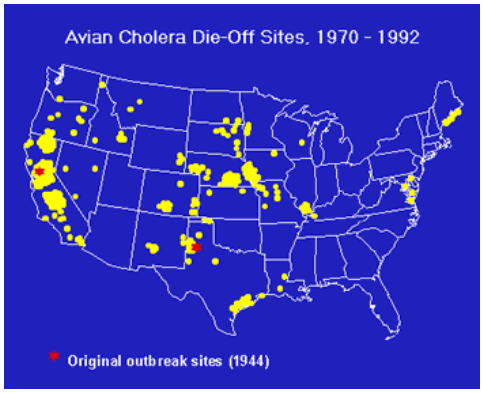
Bacterial Diseases

- **Avian Cholera** (*Pasteurella multocida*)

- *Pasteurella anatipestifer*
- *Mycobacteria* spp.
- *Salmonella* spp.
- *E. coli* (Colibacillosis)
- *Mycoplasma* spp.
- Erysipelas
- Chlamydiosis

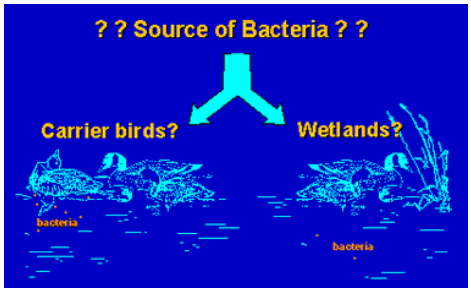
Avian Cholera

- *Pasteurella multocida*
- Bacteria is readily disinfected and destroyed by sun, drying, heat
- Annual waterfowl outbreaks during periods of stress – migration, crowding, breeding, etc.



Avian Cholera - transmission

- Chronically infected birds likely carriers. Wild birds possible source for captive waterfowl
- Contaminated food and water from eyes, oral, nasal, and fecal shedding of bacteria
- Route of infection via ingestion, inhalation or inoculation into feet through contaminated substrate (corn stubble)



Avian Cholera – clinical signs

- Few or none if acute
- Anorexia, fluffed feathers, diarrhea, mucous oral discharge
- Rapid death, incubation (<24 hrs)
- Classic – high mortality, few symptoms
- Chronic – variable symptoms - swollen joints & sinuses, respiratory disease

Avian Cholera - diagnosis

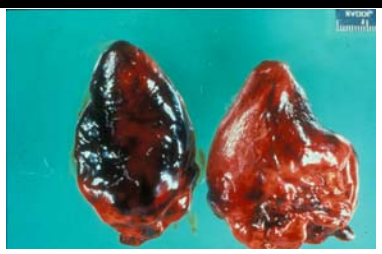
Tentative diagnosis based on history, clinical signs, gross lesions, and observance of Gram negative bipolar rod bacteria in heart blood or from liver cytology

Confirm diagnosis with bacterial culture / identification

Consider other bacterial diseases too, as well as DVE (discussed later)

Avian Cholera - pathology

- If acute death - good body condition
- Lots of hemorrhage - heart, lungs, gut, abdominal fat
- Swollen, dark liver with focal white spots (necrosis)
- GI tract - thick yellow or bloody fluid, loaded with *P. multocida* bacteria
- Chronic form – localized infection, caseous abscesses, pneumonia



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Avian Cholera - control

Captive flocks – eliminate carriers, maintain closed flocks, good hygiene & biosecurity from wild birds

In the face of an outbreak, the disease may spread very rapidly

- Carefully collect & remove carcasses to minimize further site contamination
- Implement strict biosecurity between groups

Avian Cholera - treatment

- Treatment options on an individual basis with broad spectrum antibiotics
- Killed bacterin or MLV vaccines available for flocks with persistent problems

Viral Diseases

- **Duck Viral Enteritis** (herpesvirus)

- Avian Pox
- Duck Viral Hepatitis
- Avian Influenza
- Newcastle's disease
- Goose Parvovirus

Duck Virus Enteritis ("DVE" or "Duck Plague")

- Herpesvirus, sensitive to heat & chemical disinfectants
- Only waterfowl; variable species susceptibility; adults > juveniles
- Worldwide; first US outbreak in 1967 in Pekin duck industry; now widespread, but sporadic
- Latent ("silent") carriers likely; outbreaks primarily in captive populations (rare in wild waterfowl)
- Most outbreaks in April-June during breeding season

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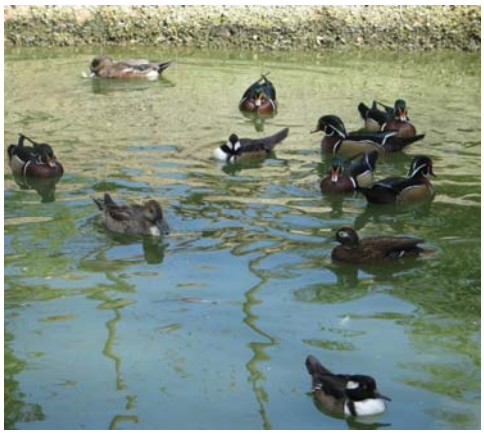
DVE - transmission

- Direct & indirect contact
- Fecal - contaminated water likely
- Oral secretions
- Blood-sucking insect vectors possible
- Carrier states in asymptomatic birds likely (typical of most herpesviruses)
- Vertical transmission from female to egg possible



DVE – clinical signs

- Incubation period 3-7 days; death in 1-5 days after acute onset of signs
- Signs – usually in good body condition; lethargy, ataxia, photophobia, drooping wings, fluffed feathers, unable to fly, bloody discharge from nares, vent; neurologic signs & convulsions just before death

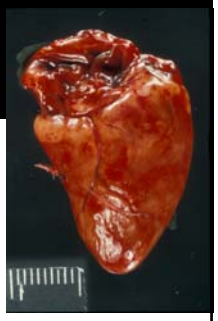
Field Manual of Wildlife Diseases

DVE – diagnosis

- Presumptive – Clinical signs & gross necropsy lesions (must differentiate from cholera!)
- Definitive – cytology is helpful (inclusion bodies), but virus isolation is best (liver, spleen samples)
- Virus neutralization titers – use to monitor progression in flock

DVE – pathology

- Gross - hemorrhage on surface of heart, liver, kidney, pancreas, lung, & GI tract; necrotic bands or patches in GI, under tongue, esophagus (some of these changes are nearly diagnostic for DVE)



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DVE – Treatment & Control

- No specific treatment available (Acyclovir?)
- Supportive care for valuable birds
- Euthanasia of sick birds to prevent spread & environmental contamination
- Captive waterfowl collections – implement strict biosecurity; prevent flight movement of birds between ponds; consider drain and chlorinate ponds; +/- depopulation; prevent contact with wild waterfowl; commercial attenuated live virus vaccine may be available

Fungal Diseases

- **Aspergillosis**

- Gastrointestinal yeast (*Candida* spp.) uncommon except in young birds

Aspergillosis

- *Aspergillus fumigatus* & *A. flavus* most common
- Most birds susceptible, but young birds, stressed birds, and certain species more susceptible (northern swans, eiders)
- This disease is often secondary to other stressors or disease

Aspergillosis - transmission

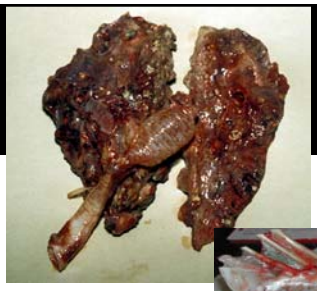
- Ubiquitous, world-wide distribution
- Infection by inhalation of spores (no bird-to-bird transmission)
- Warm temperatures & decomposing organic matter common source (i.e., moldy litter or food)

Aspergillosis – clinical signs

- Variable, and may not be apparent until advanced disease
- Young birds – may die very quickly with overwhelming infection
- Signs can include difficulty breathing, open-mouth or raspy breathing, weight loss, anorexia, weakness, and occasional neurologic signs

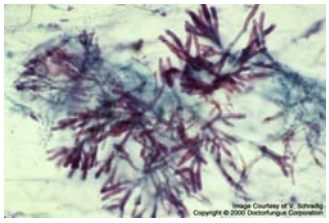
Aspergillosis - pathology

- Gross lesions usually in lungs and/or airsacs; occasionally in trachea, eyes, or brain
- Peracute – lung congestion & multiple granulomas throughout lungs
- Chronic – lung nodules, and small to coalescing airsac granulomas



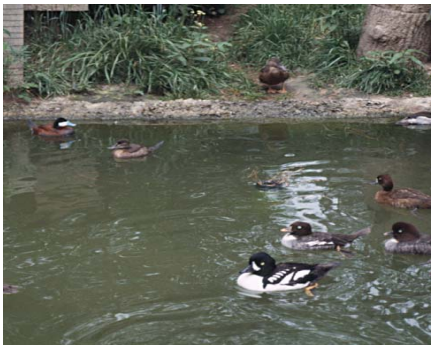
Aspergillosis - diagnosis

- Antemortem diagnosis is difficult – includes blood work, radiographs, CT scan (not often practical!), cultures, endoscopy, serology
- Most commonly, postmortem diagnosis based on gross lesions, histopathology, and culture of organisms



Aspergillosis - control

- Strict sanitation to minimize conditions that favor spore growth – remove wet food, bedding and other organic matter promptly
- Consider preventive antifungals when treating susceptible species (i.e., certain waterfowl species such as eiders, swans, etc.)



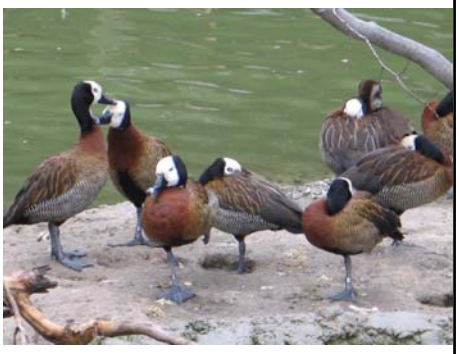
Aspergillosis – treatment

- On an individual basis, treatments can be done, but are costly, and may take weeks to months
- Treatment options – supportive care, plus Amphotericin B, and/or itraconazole, voriconazole, or terbinafine
- Prognosis for success is usually poor
- Killed bacterins used in some collections

Biotoxins

- **Botulism** (toxins from *Clostridium botulinum*)

- Mycotoxins
- Algal toxins



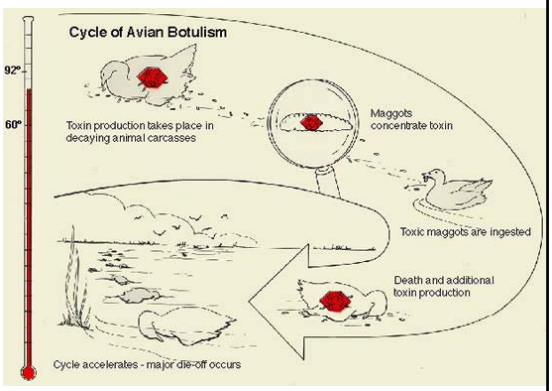
Botulism (AKA "Limberneck")

- Pre-formed toxins from *Clostridium botulinum* (usually type C)
- All waterfowl species are susceptible (plus others – shorebirds, loons, gulls, etc.)
- Worldwide

Potomacwildlife.org

Botulism

- Spores stable for years in environment (heat & drought resistant)
- Favorable environmental conditions - hot months (July-Sept.), rotting plant & animal material, anaerobic conditions, fluctuating water levels
- Fly maggots concentrate toxins



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Botulism - clinical signs & pathology

- Signs related to amount of toxin ingested
- Ascending neurologic signs – first leg paralysis, then wings, neck, nictitans (3rd eyelid)
- Death by drowning or respiratory failure
- No typical gross necropsy lesions & usually in good body condition

Field Manual of Wildlife Diseases

Botulism - diagnosis

- Presumptive – history of past problems at location, time of year, symptoms, and absence of gross lesions at necropsy
- Definitive – mouse bioassay, ELISA, or PCR

Field Manual of Wildlife Diseases

Botulism – control & Rx

- Prompt removal / disposal of carcasses during fly season
- Raise water levels if possible
- Fly control if possible
- Treatment – if possible, provide supportive care (gavage, fluids, shade, +/- antitoxin if available)

Heavy Metal Toxins

- **Lead poisoning**
- **Zinc poisoning**

Lead Poisoning

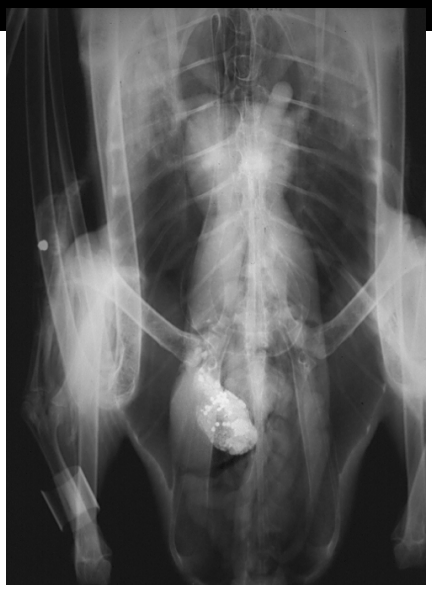
Sources – lead shot, fishing sinkers

Clinical signs -

- Weakness, lethargy
- Anemia
- Anorexia, weight loss
- Green stained vent & green-stained watery feces
- Neurologic signs
- Lose ability to fly first, then unable to walk or stand

Lead poisoning - diagnosis

- Presumptive – history, clinical signs, radiographs, gross pathology lesions
- Definitive – elevated blood lead (> 0.2 ppm)



Lead poisoning – treatment

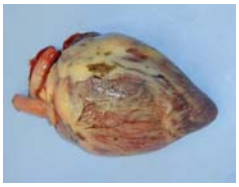
1. Supportive (fluids, nutrition, warmth)
2. Chelation therapy (i.e., CaEDTA, Succimer)
3. Removal of lead from GI tract (gastric lavage +/- endoscopy works well).

Lead poisoning – Lead Removal

- Gastric lavage
- Endoscopy
- Oral "lubricants"
  - Bulk laxatives (psyllium)
- Surgery usually last resort (very risky!)

Lead poisoning – gross pathology

- Wt. loss, emaciation
- Green stained liver
- Green, sloughed koilin lining (+/- lead pieces)
- Proventricular impaction*
- Pale, streaked heart muscle



Zinc Toxicosis

- Sources
  - various galvanized hardware items
  - US pennies (minted >1982, 97% Zn)

Zinc Poisoning

- Clinical signs & treatment similar to lead poisoning
- Except can discontinue treatment after zinc removal from GI tract
- Diagnosis - >> 3+ ppm plasma zinc