I. **PASTEURELLA** and **MANNHEIMIA**

   **Morphology, culture**

   Gram negative small coco-bacillary rods. May show bipolar staining. Grows best on blood agar (BA) **Main species:** *P. multocida* (non-hemolytic on BA), *Mannheimia haemolytica* (formerly, *Pasteurella haemolytica*) (hemolytic on BA). Facultative anaerobes.

   **Habitat/Epidemiology:** Commensals of upper respiratory tract of animals. *P. multocida* is a commensal of the mouths of dogs and cats, and sometimes vagina of dogs. Opportunistic respiratory pathogens. Also, wound infections (*P. multocida*) in dogs and cats. Horizontal transmission. No free existence outside body under natural conditions.

A. **Pasteurella multocida**

   Heterogeneous in nature, comprises several serological types (A,B,D,E and F) **based on capsular antigens.** Capsular types may be subdivided further into somatic types based on “O” antigens (lipopolysaccharides), e.g., A1, B2 etc.

   **Disease conditions**

   - Disease may be predisposed by stress and viral infections.
1. **SEPTICEMIC DISEASE**

- Can be a primary cause of disease causing *septicemia* (causal agent found in blood and internal organs).

(a) **Fowl cholera** (common in North America) wild ducks, geese → domestic flocks
- Transmission via oral, respiratory route, eye
- Sudden deaths in acute cases. Hemorrhages in internal organs and necrotic foci in liver may be seen.
- Epizootic avian cholera due to *P. multocida* has been reported in Canada.
- Type A is the common cause of fowl cholera.

(b) **Hemorrhagic septicemia** (H.S) of cattle, American Bison, water buffalo, wild/exotic Cervids
- Found mainly in tropics and subtropics. Elephants can also be affected.
- Clinical signs: high fever, hemorrhages, laryngeal edema, recumbancy, death
- Types B (Asia) and E (Africa) are responsible for H.S.
- An outbreak of H.S (Type B) occurred in a closed buffalo herd in Ontario, signs varied from sudden death to depression, anorexia, and recumbency. In 2001, Agriculture Canada warned about the possible entry of H.S causing strains into Canada.

2. **RESPIRATORY DISEASE** and OTHER

- Septicemia may occur in rabbits but not in epidemic proportions. It occurs rarely in other small animals.

a. **Disease in Rabbits:**
*P. multocida* is the most common cause of respiratory disease in rabbits.

- Most rabbits carry *P. multocida* (type A).

- Stress or concurrent disease → disease

- **Clinical syndromes**: snuffles, abscesses, pneumonia, otitis, metritis, septicemia

- **Snuffles (rhinosinusitis)**: Nasal discharge (staining of forelimbs may be seen due to attempts to clean the muzzle), epiphora (excessive tears), conjunctivitis, sneezing and coughing.

- If left untreated, may progress to **bronchopneumonia** with marked dyspnea, weight loss and death.

- Peracute cases: septicemia → death

- **Abscesses** may result from entry via a skin wound or, more rarely, secondary to bacteremia.

**b. Pneumonic disease in cattle, pigs and sheep:**

**Note:** *M. haemolytica* is a more common cause of pneumonic disease in cattle, sheep.

- *P. multocida* is typically a secondary invader with type A (cattle), and types D + A in pigs and sheep.

**c. Atrophic Rhinitis (A.R) in pigs:**

- *P. multocida* (mainly, type D) +/- *Bordetella bronchiseptica* typically associated with Progressive Atrophic Rhinitis (PAR)

- Pathogenic strains + overcrowding, poor ventilation, elevated ammonia levels, lack of immunity in piglets → Disease
- **Clinical signs**: nose bleeds, twisted snouts, reduced weight gain

- Snout sections may reveal atrophy of turbinates and deviation of nasal septum.

d. **Disease in cats and dogs**

- *P. multocida* (type C) is found in the mouths of healthy cats and dogs.

- Most infections occur following wounds (important agent in wounds and abscesses in cats). Typically have polymicrobial etiology (*Pasteurella*, G+ve cocci, anaerobes).

- *P. multocida* is **NOT** a cause of pneumonia and septicemia (rare exceptions) in cats and dogs. It can sometimes complicate viral pneumonia by proliferating in the lower airways and cause influx of inflammatory cells.

- Entry via bites, scratches, injury or following excessive licking (*P. multocida* was isolated from an abscess at an injection site in a cat in a clinic in Atlantic Canada).

- Cutaneous *Pasteurella* infections are best treated by incision and drainage, and thorough topical cleansing. Systemic therapy with antibiotics (e.g. tetracycline) may be required as an adjunct to topical treatment [see general treatment section].

- *P. multocida* can cause secondary infection following viral respiratory infection, and often isolated from pleural exudates in cats with pyothorax.
3. **Virulence factors of *P. multocida***:

- **Capsule**: virulent strains with capsule resist phagocytosis.
- **Endotoxin**: Responsible for fever, intravascular coagulation, hemorrhages
- Strains causing PAR in pigs produce a *dermonecrototoxin* responsible for turbinate atrophy.

4. **Transmission**: By contact, inhalation, ingestion (spread from cattle to cattle, birds to birds and so on). Rarely, animals such as pigs may serve as carriers of strains affecting poultry. Bites and scratch wounds in dogs and cats.

5. **Pathogenesis**: In cattle, sheep, and pigs, stress and primary viral and mycoplasmal infections of the respiratory tract predispose to *P. multocida* by impairing alveolar macrophage function and by damaging the mucociliary clearance mechanism in the trachea and bronchi. The basic lesion is *fibrinous pneumonia*. Other organisms may be present concurrently (ie. *B. bronchiseptica* in PAR contributes to the pathogenesis of *P. multocida*). Stress, including poor management predisposes to chronic endemic pasteurellosis in rabbits. Approximately forty percent of cat bites are infected with *P. multocida*. Strains that cause fowl cholera and H.S are very virulent and can cause disease in the absence of stress or other infections.

6. **Diagnosis**: Examination of a blood smear is of value in the case of septicemia (H.S, fowl cholera). All specimens should be cultured. *P. multocida* grows well on blood agar,
not on MacConkey agar. It is non-hemolytic, non-motile and indole + ve. Serotyping may be of value for control measures.

7. **Treatment and Control**: *P. multocida* strains are generally susceptible to several agents including penicillin, tetracyclines and sulfonamides. Susceptibility testing is important since resistant strains are not uncommon.

**H.S:** Annual vaccination of cattle (rather than treatment after outbreak) is important in endemic areas. Good vaccines are available. The vaccines are serotype (type B or E) specific, and protection is mainly due to specific antibodies. Oxytetracycline and sulfonamides are used for treatment of cattle in affected areas. Wild animals such as elephants may be vaccinated.

**Pneumonia in cattle and pigs:** For types involved in pneumonia, vaccines are only partially effective. Most vaccines contain antigens from other respiratory pathogens (e.g. *Mannheimia haemolytica*) as well. For treatment, antibiotics (e.g. Ampicillin-sulbactam, florfenicol, sulfa-trimethoprim) are used.

**Fowl cholera:** Live avirulent, and killed vaccines (bacterins) are available for fowl cholera (chickens, turkeys). Antibiotics (e.g. sulfonamides, tetracyclines) may be used.

**Disease conditions in rabbits:** For rabbits, penicillin group of drugs are not recommended because of possible overgrowth of *Clostridium spiroforme*. Chloramphenicol, tetracyclines or Enrofloxacin are suggested. Preventative steps for pasteurellosis in rabbits include: routine therapy, culling by regular nasal sampling, and the use of caesarian derived stock. Avoid stress such as overcrowding.
**Atrophic rhinitis in pigs:** In the event of an outbreak antibiotics (oxytetracycline, sulfonamide-trimethoprim, penicillin/streptomycin) may be used. Sulfonamides in feed or water has been used to prevent AR in weaners and growers. In disease free pig herds, nasal swabs should be examined to ensure freedom from toxigenic *P. multocida* strains using PCR or ELISA. Introduction of carrier pigs into such herds can result in outbreaks of AR.

Commercial vaccines containing *P. multocida* + *Bordetella bronchiseptica* are available and help in reducing severity of lesions.

**Wound infections and abscesses in dogs and cats:** Drugs such as penicillin or potentiated penicillin (e.g. amoxycillin-clavulanic acid) are used for treatment. Culture and antibiotic sensitivity tests should be done to select appropriate antibiotic (there are several).

**Public health significance of *P. multocida***

Human infection may result from animal bites or scratches (wound infections) and other unidentified sources (respiratory and septicemic cases).

[Note: *P. multocida* is NOT the cause of “cat-scratch fever”].

B. **Other *Pasteurella spp.*** (cause occasional disease)

- *P. pneumotropica* : opportunistic infections (pneumonia, abscesses) in lab animals
- *P. trehalosi* : ovine septicemia, pneumonia
- *P. canis* (part of normal flora in dogs), *P. dagmatis* (normal flora in dogs and cats): bite wound infections in humans
C. **Mannheimia (Pasteurella) haemolytica**

Can be distinguished from *P. multocida* in the following ways:

- Shows a tiny zone of hemolysis on BA and is indole negative
- There are many (1-16) serotypes (based on somatic antigens)

1. **Pathogenesis: (includes virulence factors)**

- *M. haemolytica* is an **important cause** (much more than *P. multocida*) of pneumonia in cattle, sheep and goats. It is **NOT** associated with dogs and cats.

- **Shipping fever** in cattle can be a fatal disease. The condition normally occurs within 1-2 weeks of transportation. Predisposing stress factors include overcrowding, poor ventilation, fatigue and dipping (for ectoparasites). Viruses (Bovine parainfluenza virus 3 and bovine respiratory syncytial virus) and mycoplasma may be contributory agents in pathogenesis.

  Stress reduces mucociliary clearance mechanism. *M. haemolytica* multiplies in nasopharynx, enter trachea and lung and releases toxin (see below).

- All serotypes of *M. haemolytica* produce a cytotoxin which is toxic to neutrophils and macrophages. This can impair lung defenses. The organism produces **fibrinous pleuropneumonia in the anteroventral areas of the lungs** due to downward drainage of initial exudates.

2. **Signs**: dyspnea (difficulty in breathing), fever, soft cough, nasal discharge, anorexia.

- **Septicemia** and death may follow.

- In the case of **septicemia** groups of organisms may enter the blood stream as emboli from
the upper respiratory tract, and can lodge in the capillary beds of lung, liver and spleen. Rapid multiplication of *M. haemolytica* in these tissues leads to death, presumably from the release of endotoxin. *M. haemolytica* can also cause acute **mastitis** in **sheep** (“blue bag”) which may be fatal due to endotoxemia.

3. **Virulence factors**: capsule, endotoxin, leukotoxin (impairs lung defense)

4. **Diagnosis**: 
   - Culture material from tracheal aspirates or lung lesions at necropsy in cases of pneumonia. Culture milk in cases of mastitis.
   - Examination of smears from the specimens (blood, spleen) in cases of septicemia may be helpful before culture.

5. **Vaccines/Treatment/Control**:
   - PRESPONSE ® HM contains leukotoxin toxoid + killed *P. multocida*.
   - Treat with antibiotics for several days. Oxytetracycline long-acting form, trimethoprim-sulphadoxine, ampicillin - sulbactam, ceftiofur and florfenicol are the commonly used drugs. Antibiotics cannot be used if live vaccines such as “Ship-Guard” are used.
   - Reduce stress factors such as overcrowding.

II. **HAEMOPHILUS, HISTOPHILUS** and **TAYLORELLA**
- *Histophilus somni* (formerly, *Haemophilus somnus*), an important pathogen of cattle

- *Taylorella equigenitalis* (formerly, *Haemophilus equigenitalis*), is an equine pathogen exotic to North America

**Morphology, culture:**

- Small Gram negative rods or coco-bacilli. Pleomorphic, and may form filaments.

- Enriched media may be required for initial isolation. Generally form small colonies.

- Incubation in an atmosphere of 10% CO\textsubscript{2} enhances growth of many species.

- *Haemophilus spp.* have a requirement for growth factors “X “(hemin) and/or “V” (nicotinamide adenine dinucleotide: NAD). Hemin can be provided from RBCs in blood agar. NAD is usually provided by a co-cultured *Staphylococcus* streak. NAD requiring bacteria form colonies only in proximity to the Staph streak (satellite growth).

**Habitat/Epidemiology:**

Upper respiratory and/or genital tract mucosa. Strict parasites. Don't have saprophytic existence. Carriers ➔ clinical disease (transmission via aerosol, contact). Tend to be host specific.

**Main species and disease conditions:**


**B. *Haemophilus parasuis***

- Less serious respiratory pathogen in swine than *A. pleuropneumoniae*. Part of flora
of the nasopharyngeal mucosa of normal pigs. Secondary invader in respiratory disease

**Pathogenesis:**
- Stress may predispose systemic invasion.
  - Severe outbreaks can occur in specific pathogen-free (SPF) herds.
  - Primary agent of **Glasser's disease**, a disease of young pigs characterized by polyserositis arthritis and meningitis.

**Control:**
- Bacterins stimulate serotype specific protective antibodies, and can be used in problem herds. Pigs from SPF or minimal disease - herds should not be mixed with conventional pigs. Drugs used for treatment (prompt treatment is essential) are penicillin, tetracyclines and trimethoprim-sulfa.

C. **Haemophilus paragallinarum**
Causes **Fowl Coryza** /Infectious Coryza, a widespread disease of economic significance.
- Similar to **H. influenzae** in humans in pathogenesis.
- Frequently associated with viral infections.
- Symptoms: Acute rhinitis, sinusitis with edema, facial swelling, and nasal discharge.

**Control:**
Serological testing to detect infected flocks, use of bacterin type vaccines, and medication with penicillin, tetracycline or spectinomycin in water.

D. **Histophilus somni** (previously **Haemophilus somnus**)
- Does **NOT** require X or V factor, but blood agar and a CO₂ incubator are needed for isolation.

- **Habitat, and Epidemiology:**
  
  Male and female bovine genital tract. Transient colonization of upper respiratory tract, including nasal cavity in some animals. Spread by contact, and possibly aerosol via infected urine (splashing), infected semen from carriers.

- **Disease:**
  
  1. **Infectious thromboembolic meningoencephalitis (ITEME)** in cattle, the neural form of *H. somni* infection, particularly in feedlot calves.

  - Endogenous infection + stress, exhaustion + other factors including unknown properties of the bacterium triggers disease. During outbreaks the virulence of the organism increases by passage from one animal to another (shown experimentally in calves).

    Peracute: death

    Acute: fever, staggering, knuckling of fetlock, somnolence, blindness, coma, death

  **Pathogenesis and lesions:** The organism may migrate from the genital tract, or respiratory infection may follow inhalation via aerosol from the urine splash from infected animals. Septicemia is followed by localization in the central nervous system. *H. somni* attaches to vascular endothelial cells. Platelet aggregation, thrombosis, meningitis, necrotic and hemorrhagic areas in the brain are the possible consequences.

  Virulent strains *H. somni* can multiply within phagocytes. Lesions in retina →blindness.

  2. **Other conditions in cattle:**
VPM 201 Hariharan/Lewis

- Respiratory disease (bronchopneumonia)
- Endometritis and abortion
- Myocarditis and arthritis following septicemia

- **Diagnosis:**
  - In ITEME *H. somni* can be demonstrated in brain lesions. Blood and visceral organs should also be cultured. The organism grows best on blood agar under 10% CO₂.

- **Vaccines, Treatment:**
  - Commercial vaccines are available since it has been shown that presence of serum antibody correlates well with resistance to *H. somni* infection.
  - Treatment with oxytetracycline is effective in early stages. Trimethoprim-sulfadoxine and florfenicol are alternative drugs.
  - Test (semen/preputial washing cultures, Ab ELISA) and treat infected bull.

**E. Taylorella equigenitalis (Haemophilus equigenitalis)**

Causes **contagious equine metritis** (CEM), a sexually transmissible disease in mares.

Evidence of endometritis, cervicitis or vaginitis 2-12 days after being bred to a carrier stallion. Abortion is rare, but has been reported. Symptoms are uncommon in stallions. [In one report from Germany, *T. equigenitalis* was isolated from an infected stallion at slaughter from the prepuce, urethra, testis, epididymis, and seminal vesicles]. Copious vaginal discharge lasting for 10 days or so, and uterine edema are characteristics. CEM
Occurs in Europe, and it was diagnosed in 1999 in Germany and Switzerland. Canada and USA are free of this reportable disease. Strict import regulations exist. Repeated culturing (3 times in 2 weeks) is required to establish freedom from the disease. Regulations may include test-mating of culture negative stallion.

Very slow growing (culture for 7 to 10 days), PCR is used, serology is not reliable. Treatment of carriers (stallions) and antibiotic treatment of semen are useful measures in disease endemic countries. Penicillin, ampicillin, gentamicin, and cefotaxime are examples of drugs used. Vaccines are not used.

III. **ACTINOBACILLUS**

- Pleomorphic G-ve rods (bacillary, occasional cocci: “Morse code” morphology.
- Growth on MacConkey’s (except for A. pleuropneumoniae). Lactose fermenters

**A. Actinobacillus pleuropneumoniae** (previously *Haemophilus pleuropneumoniae*)

It causes contagious pleuropneumonia of swine. There are several serotypes; 1,3,4,5 are more common in North America. Cases commonly occur in late fall and winter. There may be a history of ventilation failure or environmental temperature decrease prior to an outbreak. Introduction of pigs carrying this organism into “clean herds” (infection free herds) usually results in disease. Mortality is usually high in outbreaks in “clean herds”. *(Symptoms: fever, shivering, often bleeding from nose and mouth).*

Sero fibrinous pleuritis and fibrinous pneumonia in pigs around 3 months of age. Nursing pigs may develop septicemia and meningitis. Survivors of acute infection often develop
chronic lung lesions (sequestra, abscesses, and pleuritic adhesions) and become carriers. Chronically infected animals have reduced feed conversion, higher medication costs, lower weaning rates, and reduced market value.

**Virulence factors/ Pathogenesis:**
Capsule, endotoxin, cytotoxins (including a hemolytic toxin). The endotoxin and cytotoxins are responsible for damage to blood vessels and the lesions. The cytotoxins produce pores in cell membranes. Neutrophils attracted to lung tissue are damaged and release lytic enzymes. This inflammatory response is a major factor in rapid tissue necrosis.

**Diagnosis:**
Culture specimens on blood agar and inoculate a *Staphylococcus* culture across the center of the plate. Most strains form tiny hemolytic colonies and show *satellite growth* on *Staph* streak (Factor V/NAD requirement) and are urease positive. To identify carrier pigs serological tests such as ELISA are used.

**Immunity:**
There are 12 serotypes based on capsular antigens; immunity is serotype specific. Protective antibodies are passively transferred in colostrum to baby pigs.

**Control:**
- Bacterins (polyvalent) reduce mortality, but do not prevent pulmonary necrosis in all
situations. Experimental work in Canada using avirulent strains appear promising. Also, subunit vaccines containing toxoids and capsular antigens have been developed. Though the organism is susceptible to several antimicrobials, therapy is of no use to pigs with chronic disease.

- Tiamulin (a macrolide antibiotic like erythromycin and tylosin) has been reported to significantly improve the performance of pigs with pleuropneumonia.

- Introduce only serologically negative animals in clean herds.

- Avoid predisposing factors such as poor ventilation and chilling.

**B. A. lignieresii** - Wooden or timber tongue.

- Commensal of the alimentary tract, particularly the mouth of cattle and sheep.

- **Opportunistic pathogen** that gains access to deeper tissues through wounds.

- Slowly developing pyogranulomatous lesions in the soft tissues of the lower jaw and neck, also the tongue with proliferation of fibrous tissue. Occasionally lesions in lungs.

- Granulomatous lesions on jaw can be difficult to distinguish clinically from “Lumpy jaw” (*Actinomyces bovis*). Table 1 (next page) summarizes differences.

- Treatment: **Surgical drainage** + Abx (Tetracycline, Sulfonamides) or K iodide

**C. A. equuli** : Sleepy Foal Disease

- Opportunistic pathogen. Commonly in the tonsils and intestines of healthy horses.

- Septicemia in neonatal foals following *in utero* or umbilical infection. If foal survives acute infection →then purulent nephritis, pneumonia and arthritis are frequent sequella.
- Can see arthritis, enteritis, meningitis in older foals.

- Mares - occasional abortion and/or septicemia.

- TxT: Ampicillin, Trimethoprim-sulfadiazine, Gentamicin, 4th generation Cephalosporin are choices. No vaccine

**D. A. suis**

- hemolytic (*A. pleuropneumoniae* as well), hydrolyzes esculin (only one of the four main *Actinobacillus spp.*)

- likely upper resp. and tonsil commensal but not easily cultured from these sites

- disease primarily in high-health status herds, increased incidence in 90's in N.A.

- fatal septicemia in pigs under 3 months of age

- Older pigs - pneumonia, arthritis, endocarditis

- Note: historically some strains of *A. suis* believed to cause disease in foals that mimics the clinical picture presented by *A. equuli* infection and vice versa.

- easily isolated from lesions, blood etc.

- *A. pleuropneumoniae* vaccines may offer some cross-protection, autogenous vaccines can offer some protection however there are different strains

- TxT: Ceftiofur, Gentamicin, TMS

**IV. BORDETELLA**

**Morphology/Habitat:**

Gram negative rods and coccobacilli. Grows on Blood agar and MacConkey agar.
Natural habitat is upper respiratory tract of mammals and birds. Typically secondary pathogens that do not survive long off of host.

A. **B. pertussis**: Causes whooping cough in children.. Not of concern in animals.

B. **Bordetella bronchiseptica**

**Habitat and host spectrum**

Found in the nasopharynx of many species of animals: dogs, cats, wild carnivores, rodents, swine, occasionally horses. Can cause respiratory disease in several species of animals, but more commonly in dogs, cats and pigs. Occasionally it affects humans.

**Transmission:**

Mainly by inhalation. Infection may be endogenous or exogenous.

**Pathogenesis and Disease**

1. **Dogs:**

Secondary invader in the pneumonia of distemper. Involved in **infectious tracheobronchitis (ITB) (kennel cough)** along with viruses (canine parainfluenza virus, canine adenovirus and canine distemper virus). The disease is highly contagious, but self-limiting (av. 3 weeks duration), except in neonatal and immunocompromised animals.

Most commonly occurs and spreads rapidly in places such as kennels, pet stores and veterinary hospitals.

**Pathogenesis and symptoms:**

Viral infection damages respiratory epithelial cells and predispose to secondary infection with **Bordetella** and/or **Mycoplasma. B. bronchiseptica** adheres to tracheal cilia. It
produces ciliotoxic substances including an extracellular bacterial enzyme, adenylate cyclase (ECAC) which plays an important role in infection. ECAC diminishes phagocytic and bactericidal capacities of phagocytic cells. Paralysis of cilia, inflammation and mucous accumulation lead to coughing and retching.

Fever is not a sign of uncomplicated ITB. Complicated ITB is characterized by chronic bronchopneumonia, dyspnea, weight loss, and fever. Miniature breeds with congenital tracheal collapse, and older immunocompromised animals are more likely to develop complicated ITB. The cough lasts for 1-3 wks; dogs can shed the organism for 2-3 months. The disease is self-limiting, and clinical recovery coincides with development of mucosal immunity (IgA).

**Diagnosis:** Symptoms and confirmation by culture of tracheal aspirate.

*Bordetella bronchiseptica* is motile and hemolytic (not consistent) on blood agar. It is urease positive, and does not ferment carbohydrates. It grows on MacConkey agar.

**Prevention, Treatment, Control:** Vaccines are available for prevention. Examples include a live intranasal vaccine (containing *B. bronchiseptica*, canine parainfluenza virus and canine adenovirus -2), for annual immunization of puppies and adult dogs. If coughing persists, or if bronchopneumonia is present, antibiotic therapy may be required. Amoxicillin + clavulanic acid, trimethoprim-sulfa, enrofloxacin or doxycycline are among the effective drugs. Additionally, glucocorticoids, bronchodilators, and antitussives may be used.

2. **Cats:***

*Bordetella bronchiseptica* alone can cause severe respiratory disease in cats.
Bronchopneumonia due to this bacterium in younger cats can lead to systemic manifestations (cyanosis, deaths). A recent study in UK (1999) indicates the possibility of cats acquiring infection from dogs.

A live intranasal vaccine for cats is available in USA. Recommended drugs are similar to those described for dogs. Antimicrobial sensitivity testing is recommended.

3. **Pigs:**

Causes nonprogressive atrophic rhinitis (**NPAR**) alone or contributes to pathology of progressive atrophic rhinitis (**PAR**) in association with *P. multocida* (type D). Aerosol spread.

*B. bronchiseptica* provides nasal irritation through certain exotoxins (similar to canine strains), rendering the turbinates susceptible to the action of the dermonecrototoxin of *P. multocida*. Pathological changes in osteoblasts and osteocytes ultimately lead to cell lysis and turbinate atrophy.

**Signs:** Coughing, sneezing, nosebleeds, twisted snouts. Cross section of snouts at abattoir will reveal turbinate atrophy

**Diagnosis of AR:** Signs and culture of nasal swabs. The swabs may be positive for *Pasteurella multocida* and *B. bronchiseptica* or *P. multocida* alone. The *P. multocida* isolates should be positive for dermonecrototoxin (ELISA tests are available commercially) or the gene for the toxin (detected via PCR on culture or specimens/swabs). *Bordetella* is identified by morphological and biochemical properties.

**Control of AR:** Vaccines are available. Overstocking of animals should be avoided. Good ventilation (ammonia levels). Sulfonamides can be used prophylactically. On disease-free farms, nasal swabs are examined for toxigenic strains of *Pasteurella multocida*
4. Other species:

Infection in horses, poultry, and rarely in humans (immunocompromised) and captive wild animals have been reported. (JAVMA 1993, reported a fatal pneumonia case in a Koala in Chicago zoo)

C. **B. avium**

Agent of turkey coryza. Found in Canada and USA. Organism survives well in dust.

Spread and pathogenesis - same principles for B. bronchiseptica apply for B. avium.

Infection with B. avium can predispose secondary infections due to bacteria such as *Escherichia coli*.

*B. avium* is susceptible to several antimicrobials including tetracycline. Live vaccines are available for immunization of poultry via spray, eyedrops, or drinking water.

Environmental factors such as temperature, humidity and air quality should be optimized.

Serological tests are available to detect infected flocks.

V. **MORAXELLA**

**Morphology:**

Gram negative short rods. Often occur in pairs as diplobacilli or even as cocci.

Several species (at least 10). *M. bovis* and *M. ovis* are of primary importance.

**Moraxella bovis**

Non-motile, virulent strains are hemolytic on BA (do not grow on MacConkey’s) and have pili. Many pilus antigenic types. Causes infectious keratoconjunctivitis (IBKC, also called
"Pink eye" and “New Forest disease”) in cattle all over the world. According to one estimate, losses in USA due to this condition (leads to reduced weight gain) in beef calves and feed lot cattle are around $150 million annually.

**Habitat:**

Some cattle carry the organism on conjunctiva and upper respiratory mucosa with no signs.

**Disease/pathogenesis:**

Several predisposing factors. Young cattle of < 2 years of age are more susceptible. Other factors include dust, tall grass, u.v. light, infection with bovine herpesvirus, and fly activity. Transmission by direct contact and spread by flies.

Strains with pili adhere to cornea → pitting, edema, lymphocytic infiltration, sloughing of tissue → ulcer.

**Symptoms:** include photophobia, conjunctivitis, copious serous discharge from the eye (approx. 2-6 weeks), and later corneal edema, opacity, ulceration and sometimes even rupture of anterior chamber. Loss of weight and decline in milk production may occur.

**Virulence factors:**

Pili, hemolysins and toxins (dermonecrotic factors, an exotoxic factor which causes ocular and local edema), hyaluronidase, collagenase

**Diagnosis:**

Signs + isolation of *M. bovis* from eye swab are diagnostic.
**Control:**

Vaccination of pregnant cows with a pilus vaccine (polyvalent) is effective in raising antibody levels in the colostrum and thereby passively protecting calves. Segregate infected animals, and protect from direct sunlight and dust. Control flies (fly-repellent ear tags may be of use).

Antibiotics topically, subconjunctivally, or parenterally. Gentamicin topically and oxytetracycline (I.M) are generally used.

**Note:** A similar condition (Pink eye) in sheep is caused by *Moraxella* (previously *Neisseria*) *ovis*.