

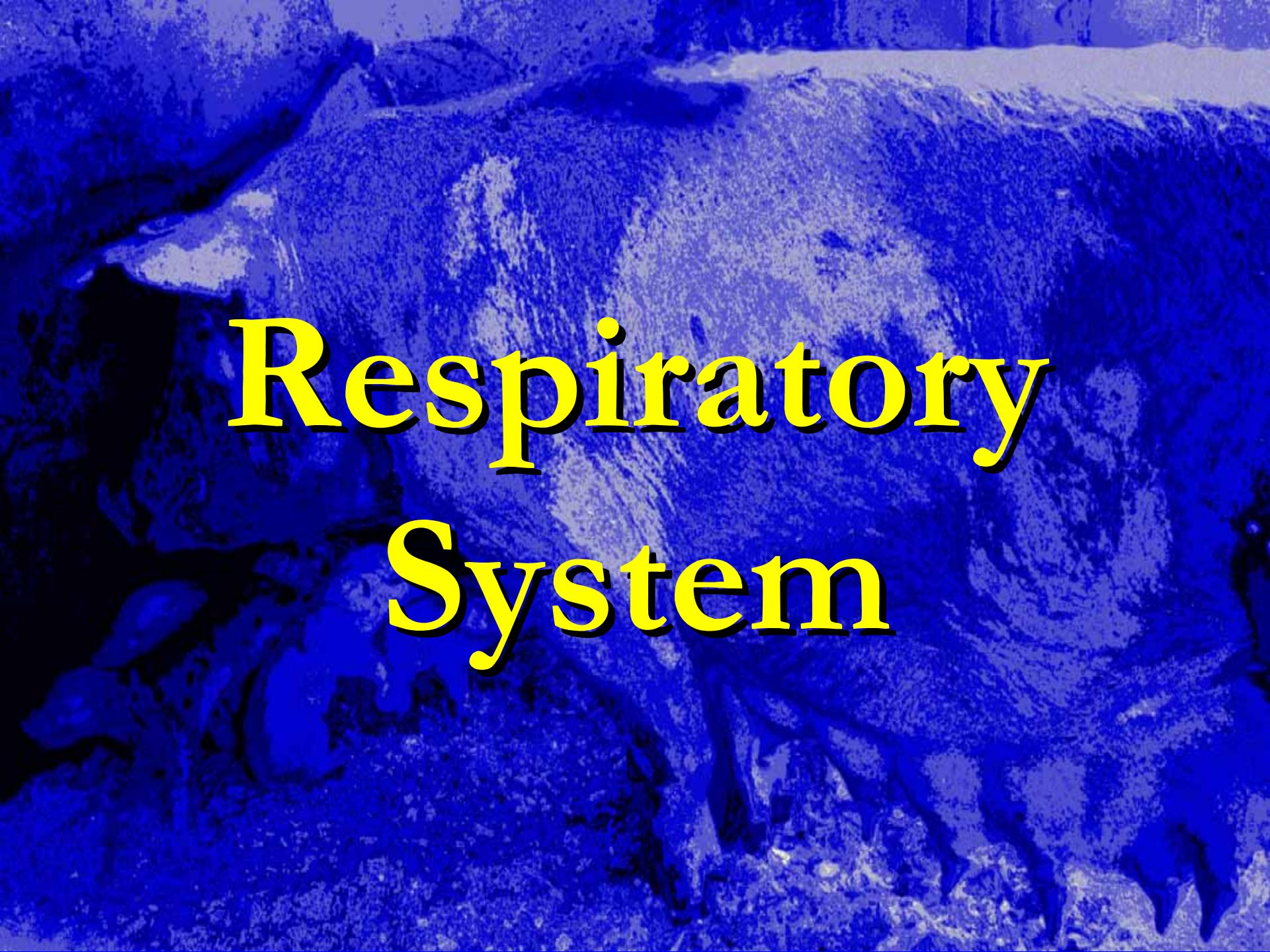
Pathology of Swine

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Respiratory System

Diagnosis of Respiratory Diseases in Swine

- Complete Necropsy on ACUTE pigs
- Submit both fixed and refrigerated samples:
 - Lung, trachea, nasal turbinates
 - Brain, spleen, tonsil, liver, kidney, lymph nodes,
 - Serum
- Serology: coincident seroconversion

Gross Examination of the Lung Distribution

- **Diffuse** ⇒ vascular or viral
- **Dispersed multifocal** ⇒ vascular and/or embolic
- **Lobular** ⇒ airway, segmental
- **Cranioventral** ⇒ dependent
- **Hilar** ⇒ App

Gross Examination of the Lung Color

- **Pink** \Rightarrow blood + air = normal
- **Purple** \Rightarrow blood - air = atelectatic
- **Red** \Rightarrow blood (congestion or hemorrhage)
- **Red spots or blotches** \Rightarrow hemorrhages
- **White** \Rightarrow WBC (Pus), Fibrin, Albumin

Gross Examination of the Lung

Consistency

- **Soft, resiliant** ⇒ normal
- **Fluctuant** ⇒ edematous
- **Firm, resiliant** ⇒ fibrinous
- **Firm, unyielding** ⇒ cellular infiltrates
- **Hard, unyielding** ⇒ scarring
- **Crepitant** ⇒ emphysema

Gross Examination of the Lung

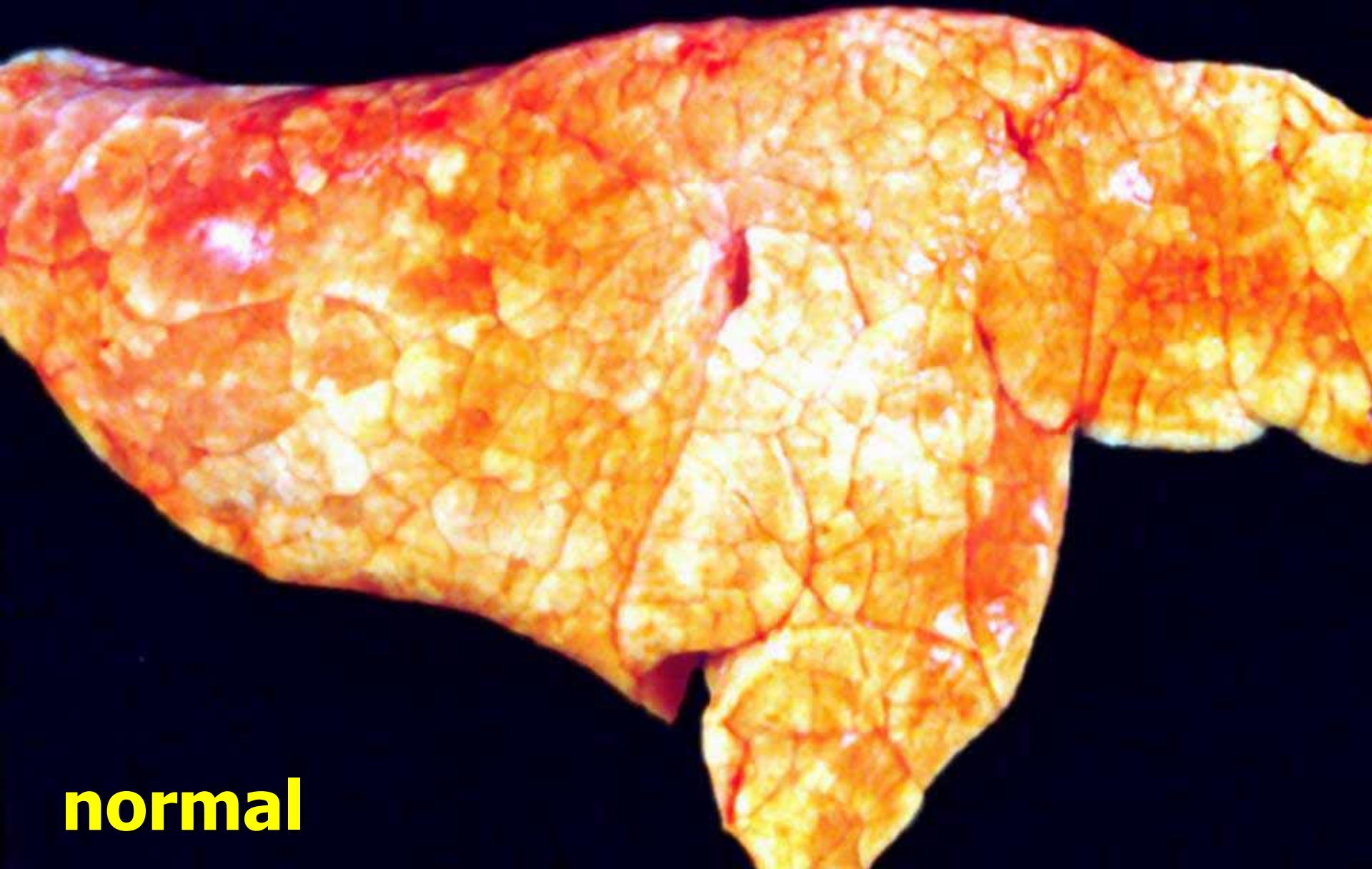
Cross-Section

- Dry, friable \Rightarrow necrosis
- White interlobular septae \Rightarrow fibrin
- White grape-like clusters \Rightarrow pus in alveoli
- Red blotches (interlobular septae) \Rightarrow hemorrhage
- Glistening stringy clear or white material in airways \Rightarrow mucous +/- WBC

Gross Examination of the Lung

Integration

- **Lesions in lungs** → differentials
- **Lesions in other organs** → differentials
- One disease? Systemic disease?
- Multiple diseases?
- Adequate samples?



normal

Progressive atrophic rhinitis

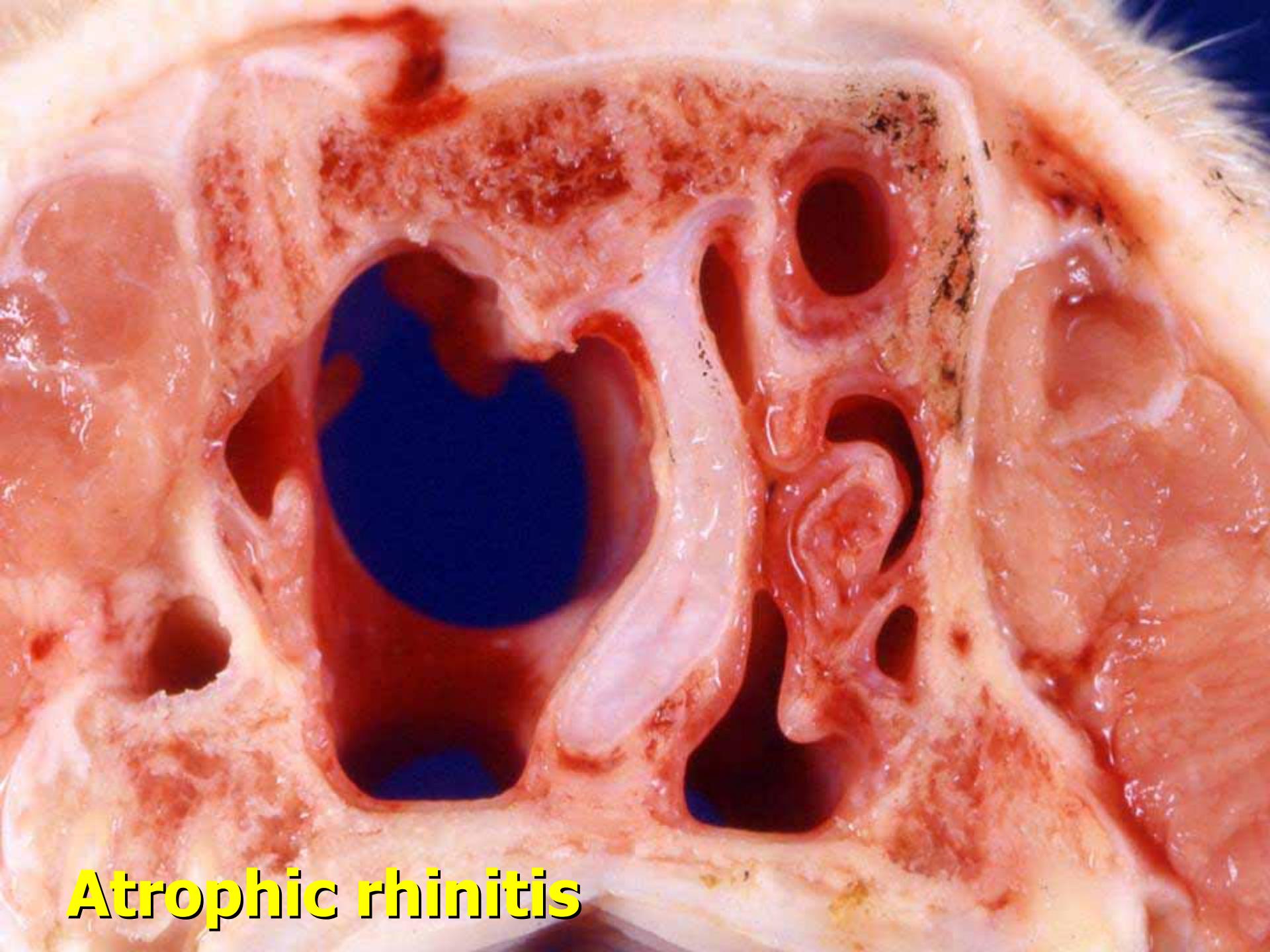
- Toxigenic *P. multocida* (usually type D)
+/- co-infection with *B. bronchiseptica*
- *P. multocida* produces dermonecrotoxin > demise of osteoblasts > enhanced osteoclast activity > turbinate atrophy > distortion of nasal septum > possibly shortening and twisting of upper jaw
- Clinical signs: Sneezing (1-8 weeks of age)
+/- epistaxis, blockage of lachrymal ducts with tear staining, mucopurulent nasal discharge, shortening of upper jaw and corrugation of the skin of snout
- Pathology: Rhinitis, turbinate atrophy and nasal distortion
- Diagnosis: Clinical signs confirmed by culture of nasal swab for toxigenic *P. multocida*



Atrophic rhinitis



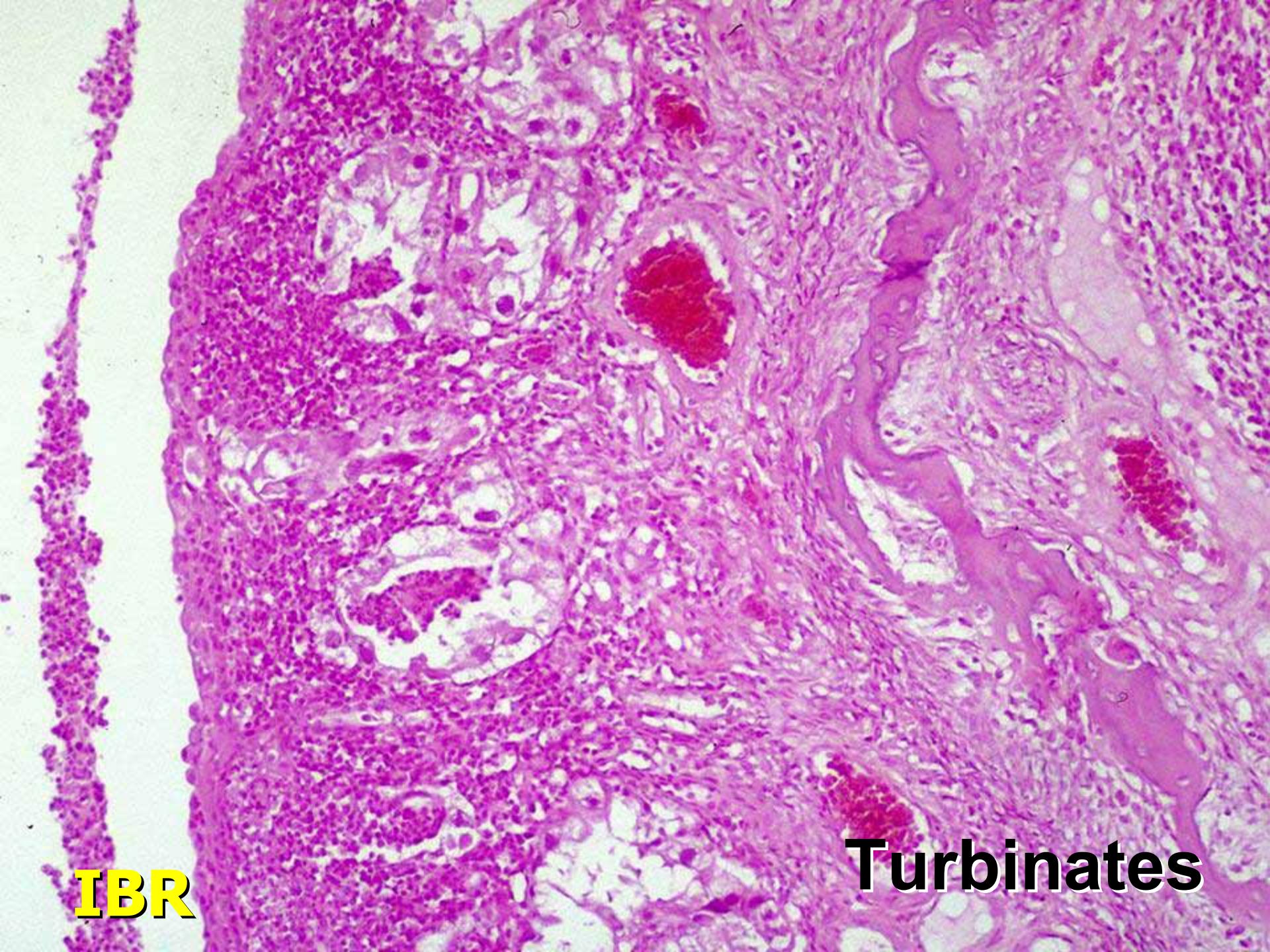
Atrophic rhinitis



Atrophic rhinitis

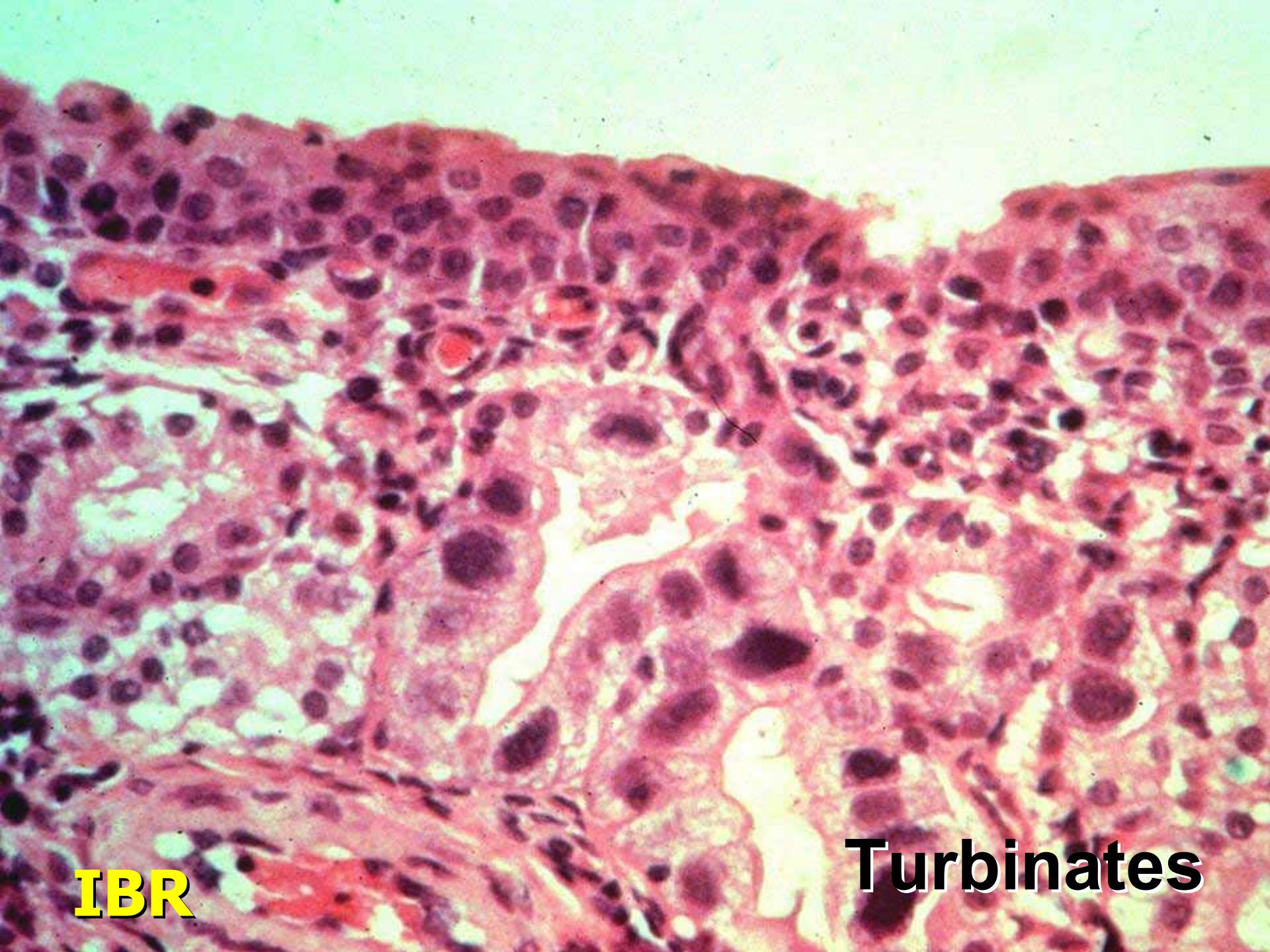
Inclusion body rhinitis

- Etiology: Porcine cytomegalovirus
- Clinical signs: Sneezing in pigs <3 weeks of age, nasal discharge and epiphora. If chronic, may lead to otitis media
- Pathology: Rhinitis and conjunctivitis
- Diagnosis: Turbinates plugged with mucus/debris, basophilic intranuclear inclusion bodies in nasal mucosa
- If herd is naïve, (eg. a new herd) any age can be affected and 25% mortality in affected litters may be seen

A histological section of tissue, likely nasal turbinates, stained with hematoxylin. The image shows various cellular components and tissue structures. In the bottom left corner, the letters "IBR" are written in yellow. In the bottom right corner, the word "Turbinates" is written in large black capital letters.

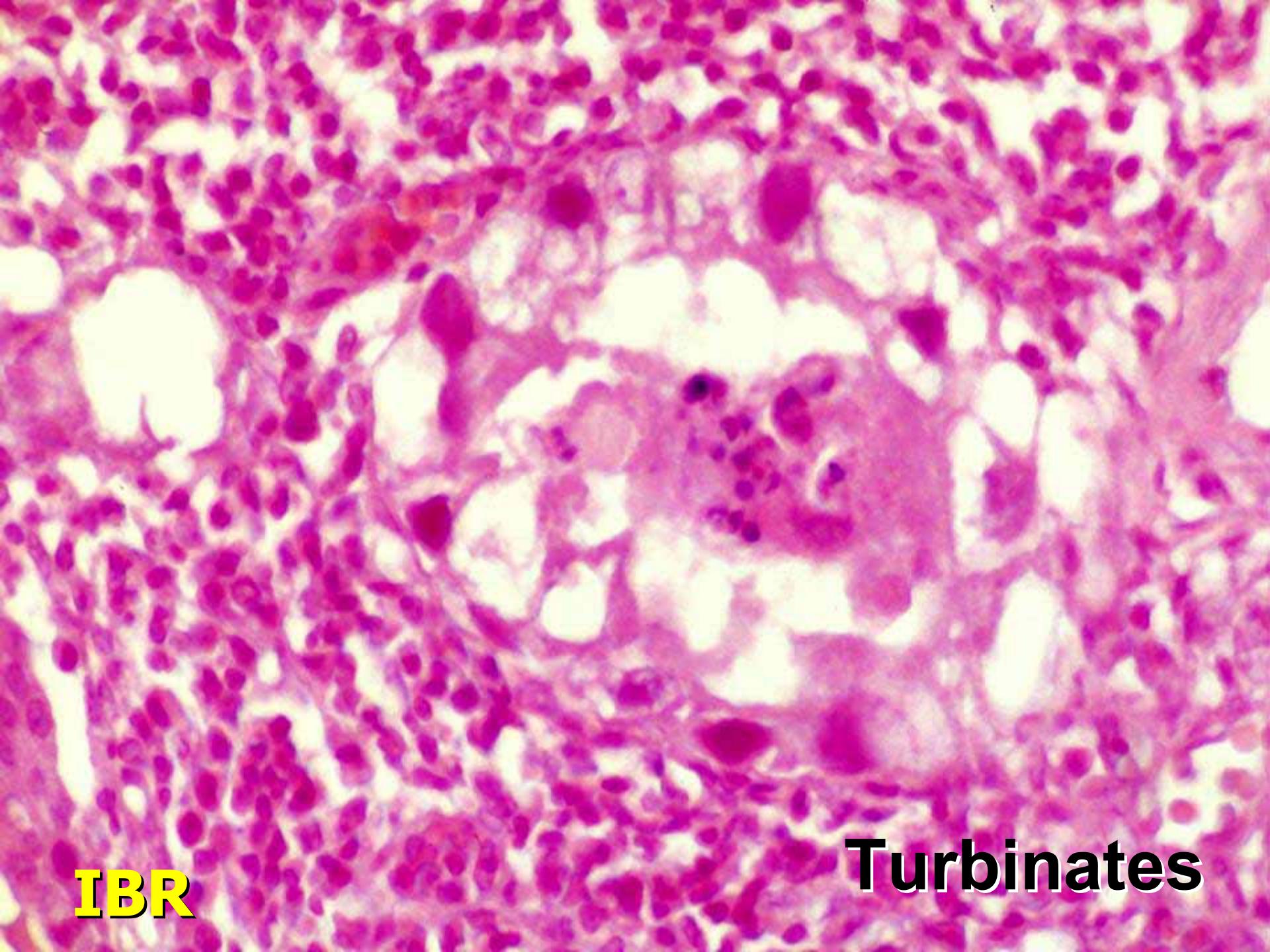
IBR

Turbinates



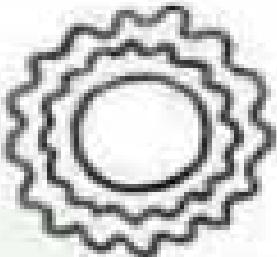
IBR

Turbinates

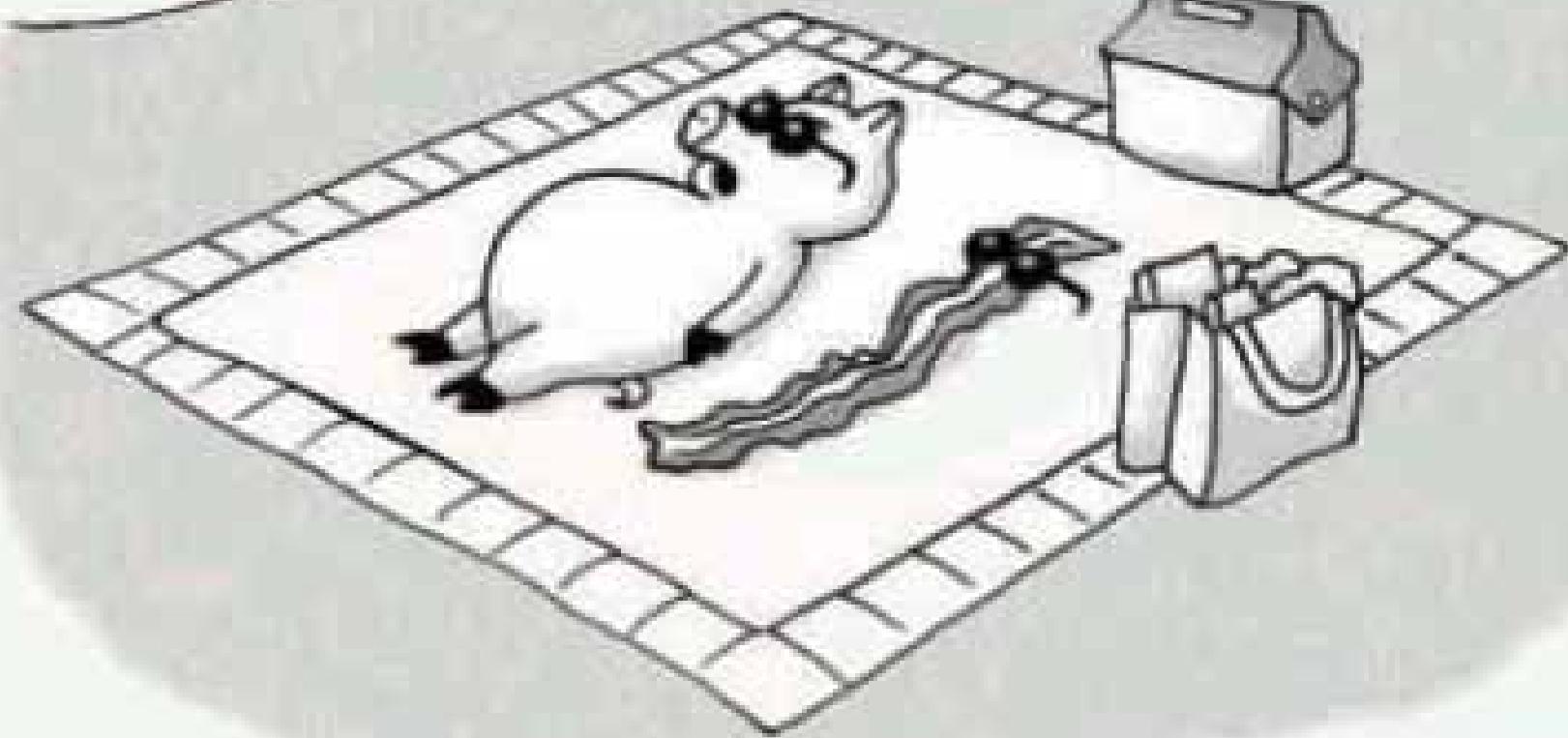


IBR

Turbinates

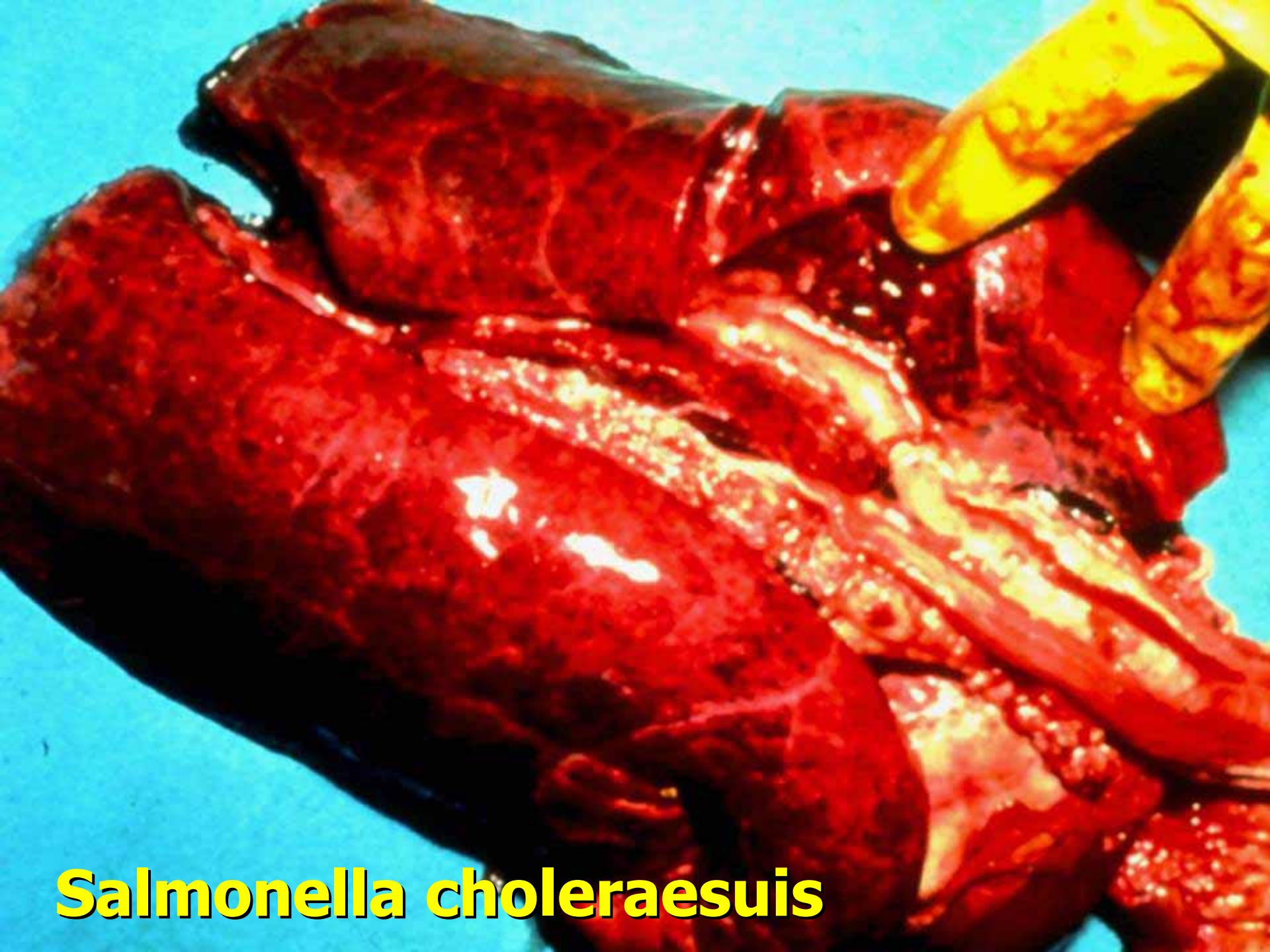


MAYBE NEXT TIME YOU'LL
TRY A LITTLE SUNSCREEN...

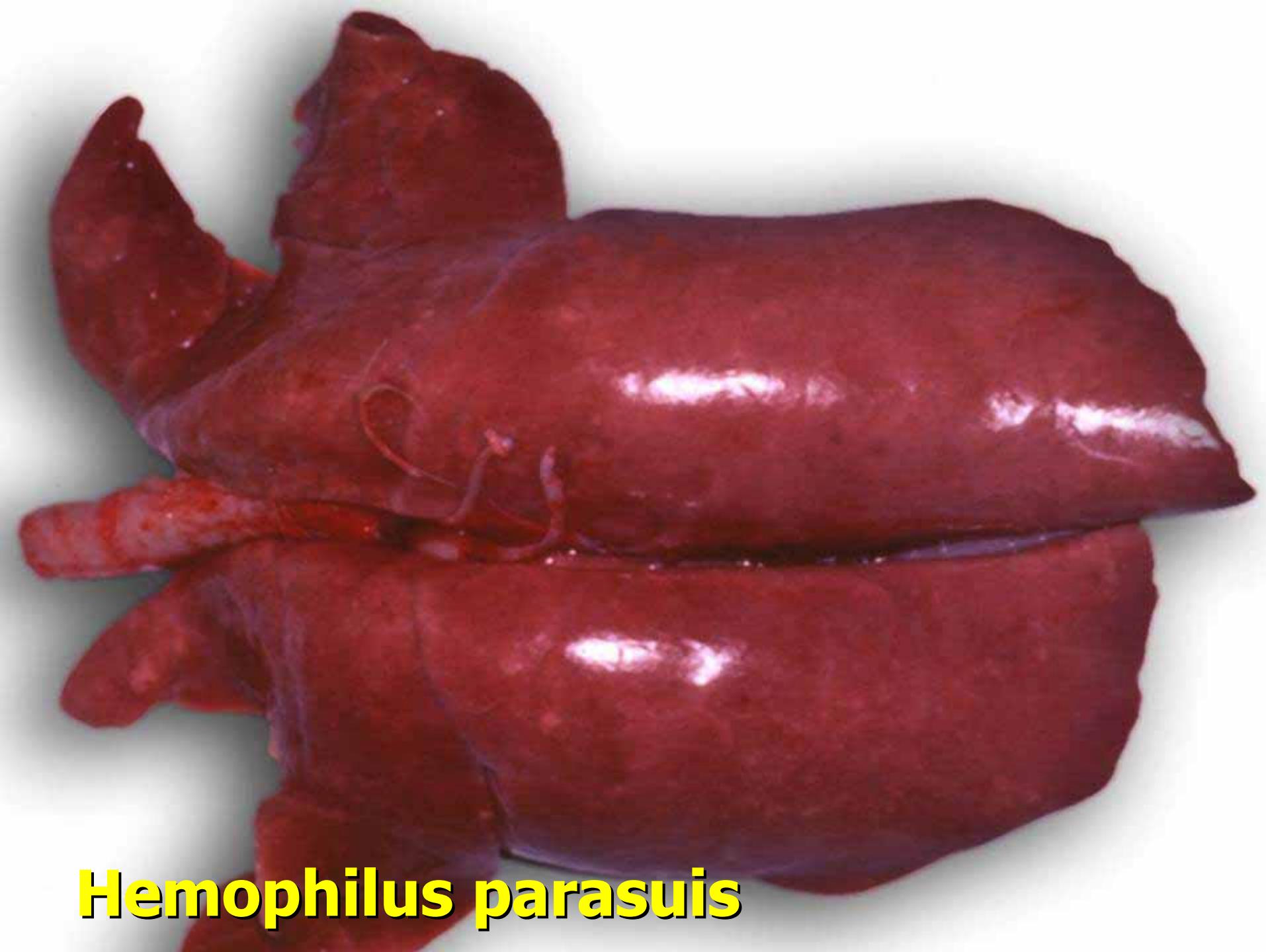


Interstitial Pneumonia in Swine

- Viral
 - Swine influenza
 - Pseudorabies
 - PRRSV
 - PRCV
 - Porcine Circovirus
- Septicemia
 - *S. cholerasuis*
 - *H. parasuis*
 - *S. suis*
 - Other
- Allergic
 - Ascarid larval migration



Salmonella choleraesuis



Hemophilus parasuis

Influenza A

- Enveloped, negative sense RNA viruses that encode 10 major viral proteins on 8 independent segments of RNA
- Two major structural proteins (determine A, B, C):
 - Nucleoprotein
 - M1 matrix protein
- Envelope glucoproteins:
 - Hemagglutinin (H) – 15 subtypes
 - Neuraminidase (N) – 9 subtypes
- Historically only limited number of subtypes in mammals
- Subclinical infection of waterfowl with all subtypes

Swine Influenza

- Epizootic and endemic form
- Replicates in:
 - Epithelium of small airways within 2 h
 - Epithelium of nasal cavity, trachea, alveoli by 24 h > alveolar macrophages
- Small bronchi blocked by neutrophil-rich exudate
- Alveolar necrosis/bronchial epithelial hyperplasia causes clinical signs
- Type A
 - H1N1 later H3N2
 - also H1N2, H4N6

SIV Induced Disease

- Rapid course
 - incubation: 12-24 hours, almost 100% of animals
 - shed in nasal secretions for 4-5 days
 - recovery, if uncomplicated, in 7 days
- Symptoms
 - coughing
 - apathetic, prostrate, erythema of skin, anorexia
 - dyspnea, sneezing, pyrexia, “drop-off”
 - reddened eyes and conjunctival discharge
 - abortions

SIV Lesions

- Gross
 - “checkerboard” cranioventral pattern
 - diffuse interstitial pattern
 - firm (proliferative) cranioventral pattern
- Microscopic
 - necrotizing bronchiolitis
 - proliferation of type II pneumocytes
 - peribronchial and perivascular cuffing

Diagnosis of SIV

- Typical lesions
- Demonstration of SIV or antigens
 - FA/IFA lung
 - VI lung, nasal swabs
 - IHC fixed lung
 - ELISA nasal swabs, bronchial swabs
- Serology: paired samples
 - HI



SIV



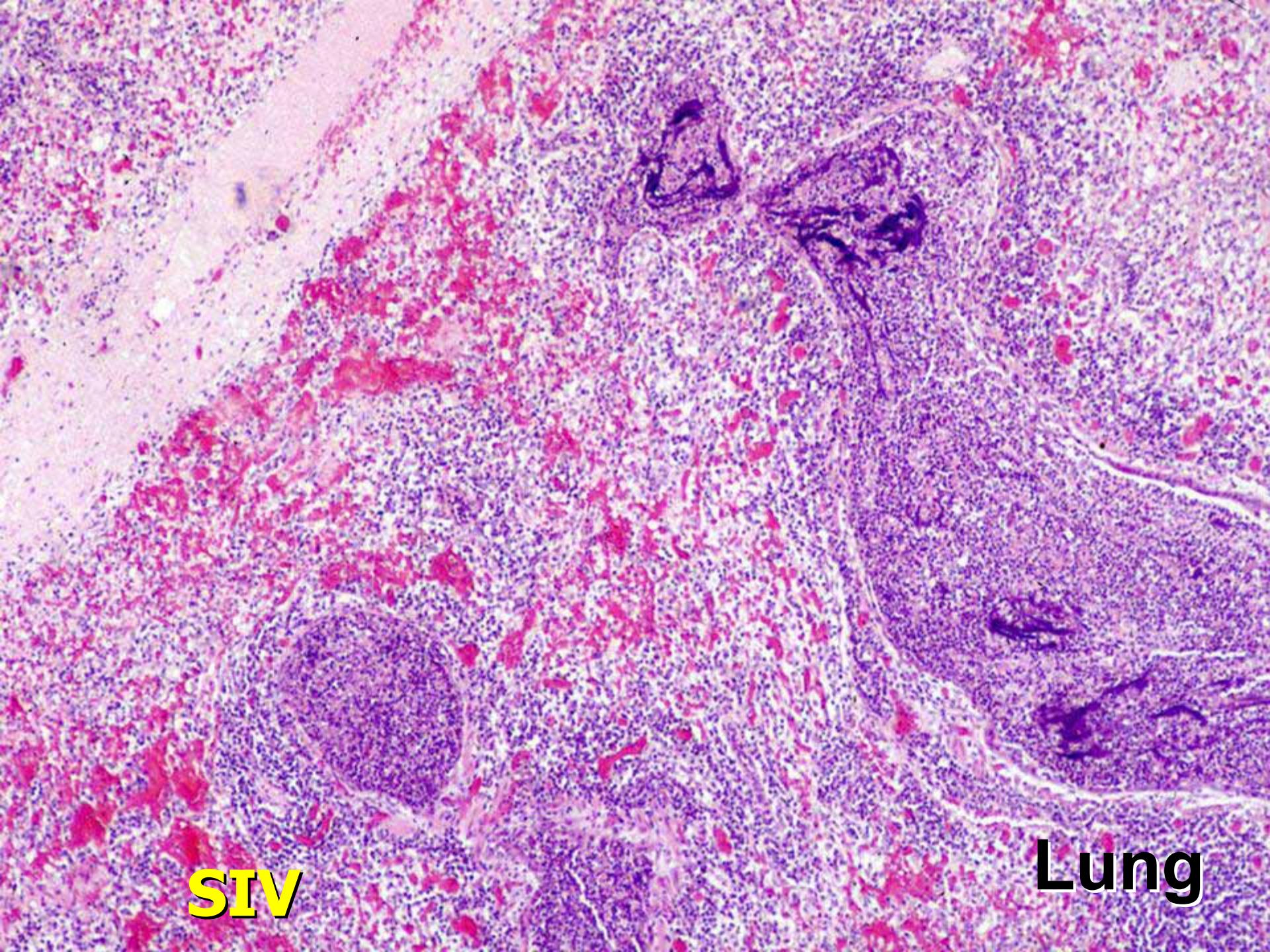
SIV



SIV

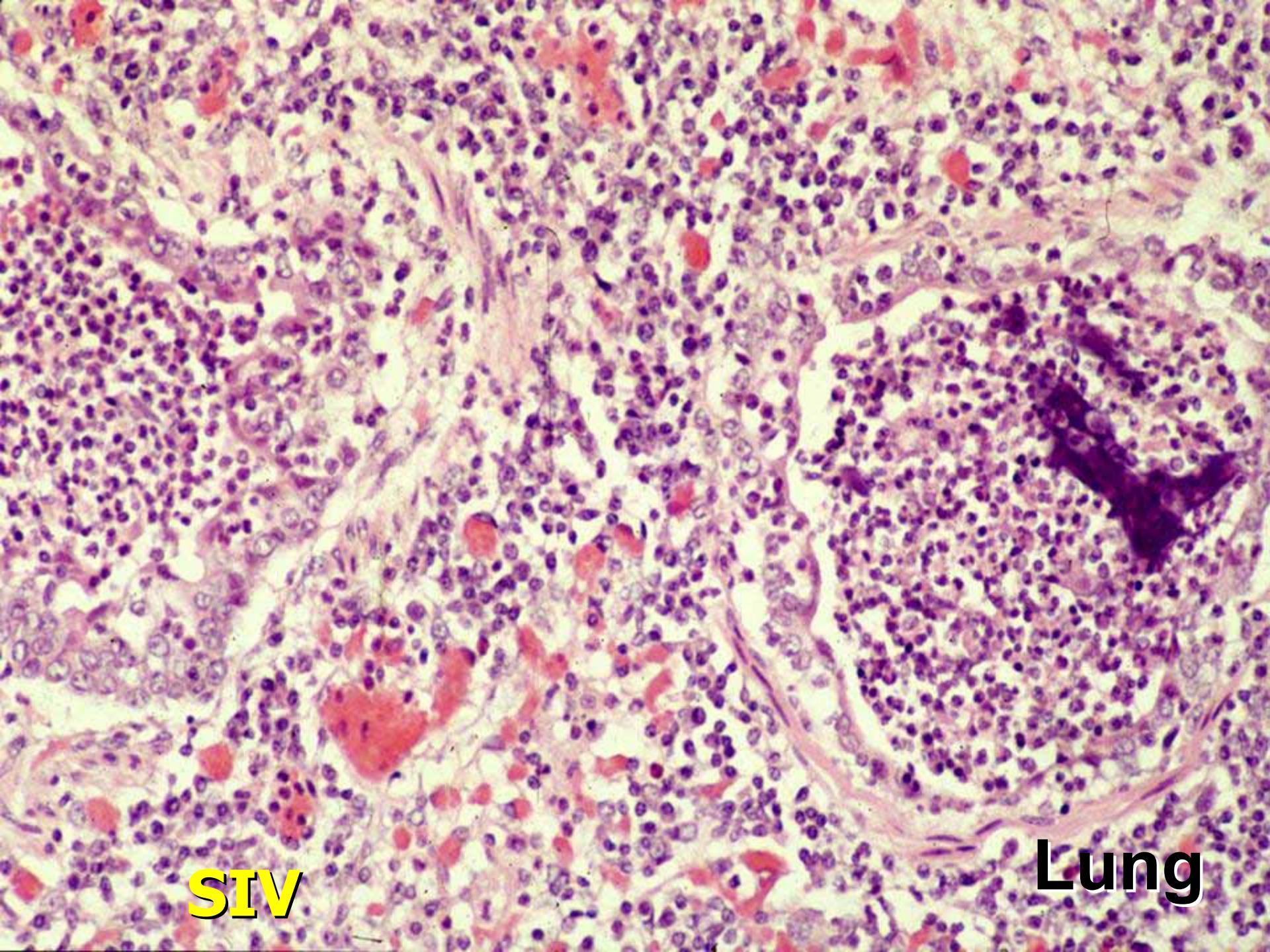


SIV



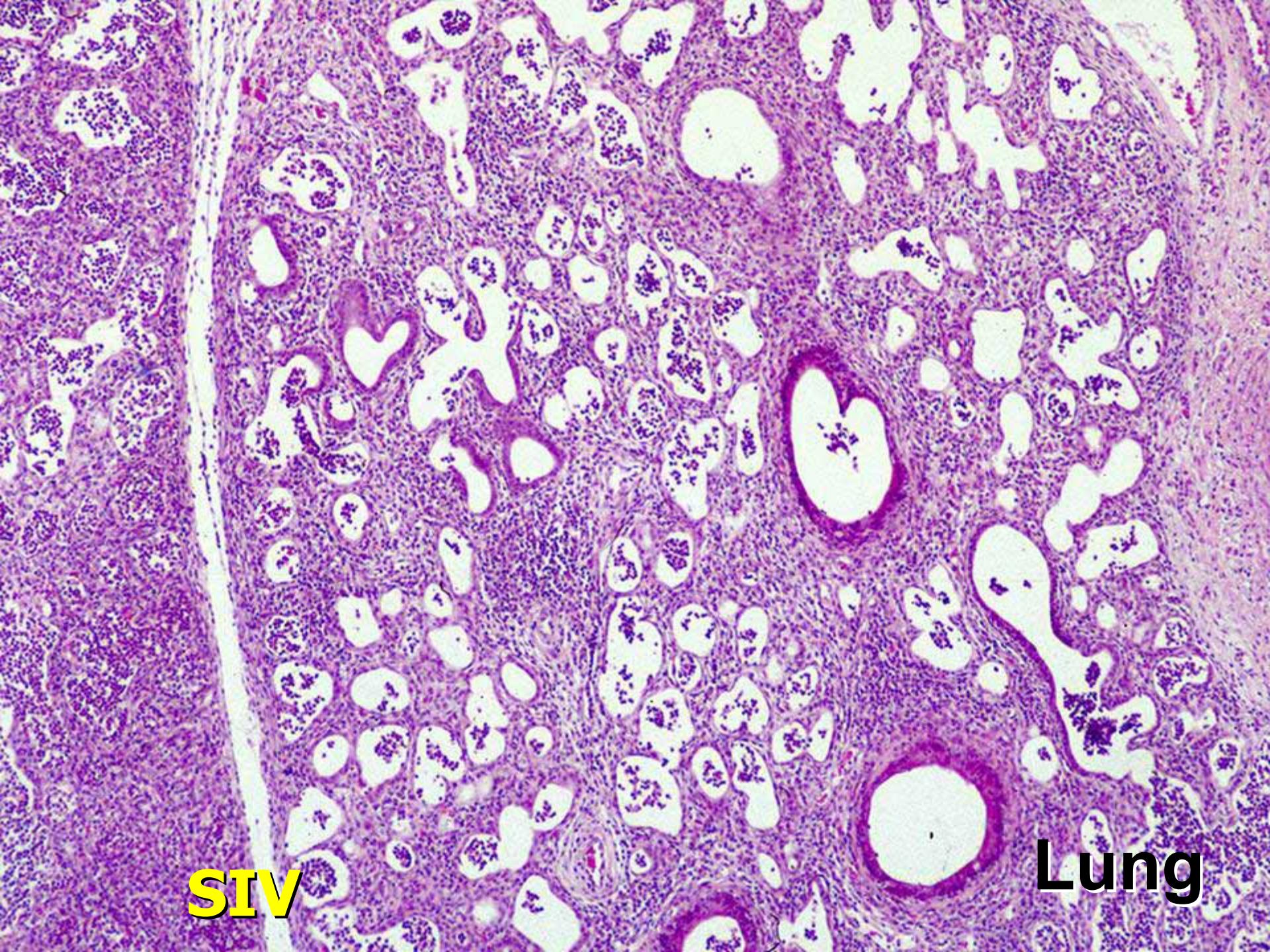
SIV

Lung



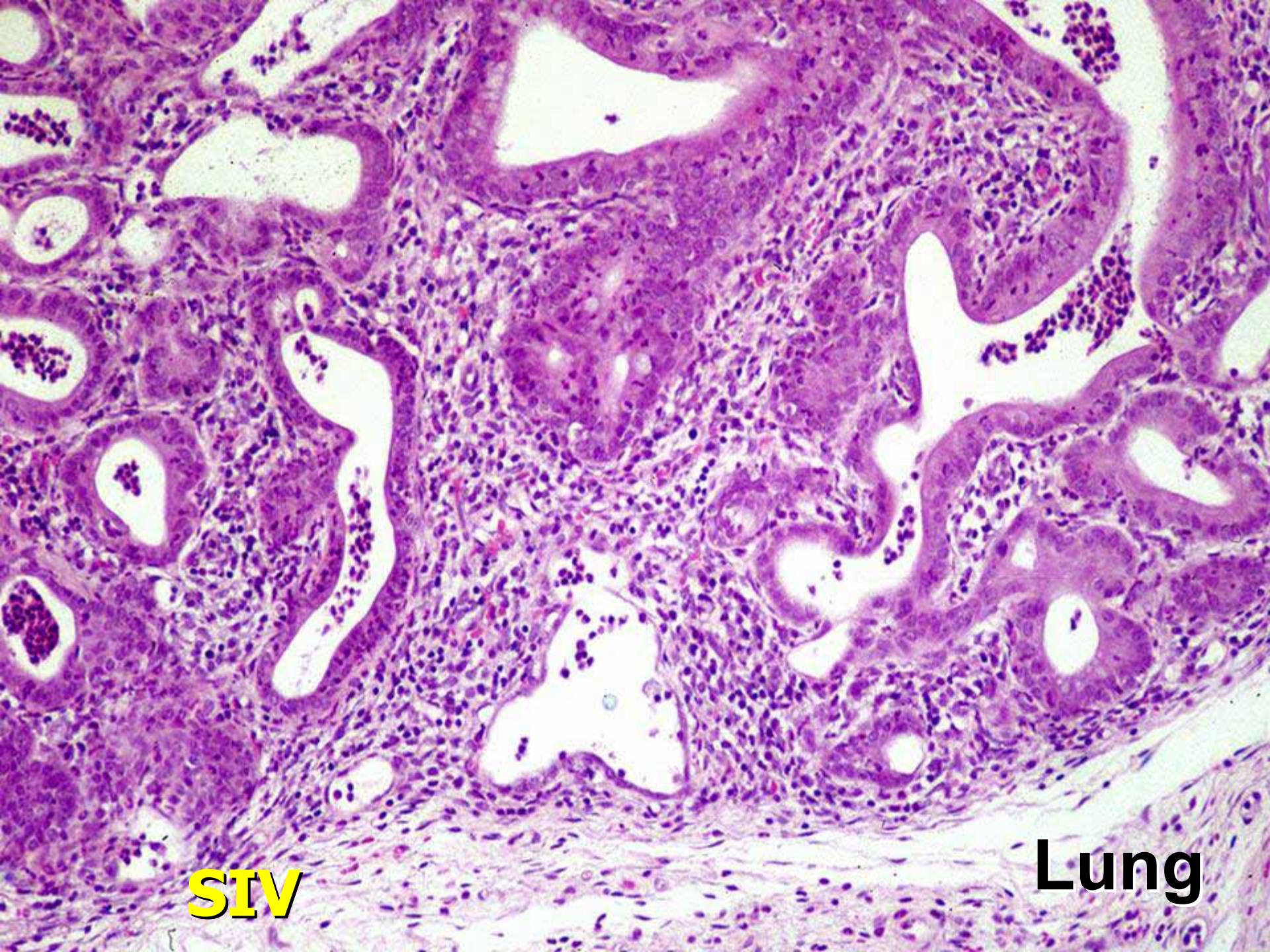
SIV

Lung



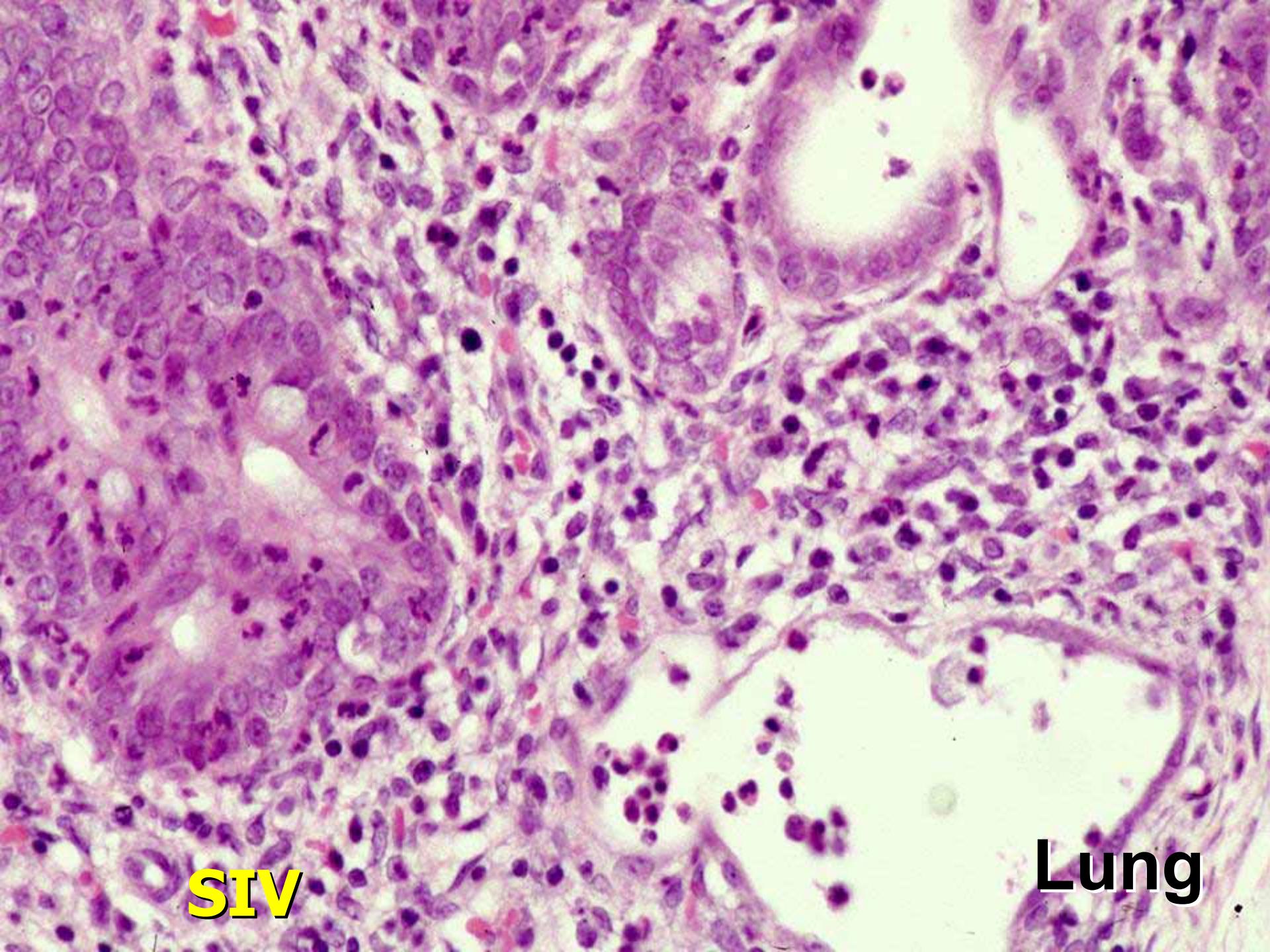
SIV

Lung



SIV

Lung



SIV

Lung

Lung

This electron micrograph shows numerous spherical viral particles, identified as SIV (Simian Immunodeficiency Virus), distributed throughout a tissue section. The particles have a dense, granular internal structure. Two specific particles are highlighted with black arrows: one in the upper left and another in the upper right. The surrounding tissue consists of various cellular structures.

SIV

As the two friends wandered through the snow on their way home, Piglet grinned to himself, thinking how lucky he was to have a best friend like Pooh.



Pooh thought to himself:
“If the pig sneezes,
he’s fucken dead.”

Swine Influenza in Humans

- Pigs are important role in inter-species transmission, because they have receptors to both avian and human influenza virus strains: “mixing vessel”
- Outbreaks and sporadic human infection with swine influenza have been occasionally reported
- Influenza outbreak caused by swine H1N1 virus in Fort Dix, New Jersey in 1974
- Outbreak in Wisconsin in 1988 resulted in multiple human to human infections
- People in contact with swine have higher antibody levels
- Swine influenza viruses have been isolated from turkeys, indicating transmission between pigs and avian species
- Pigs can be infected with the highly pathogenic avian influenza (HPAI) H5N1 virus





Porcine Respiratory Coronavirus

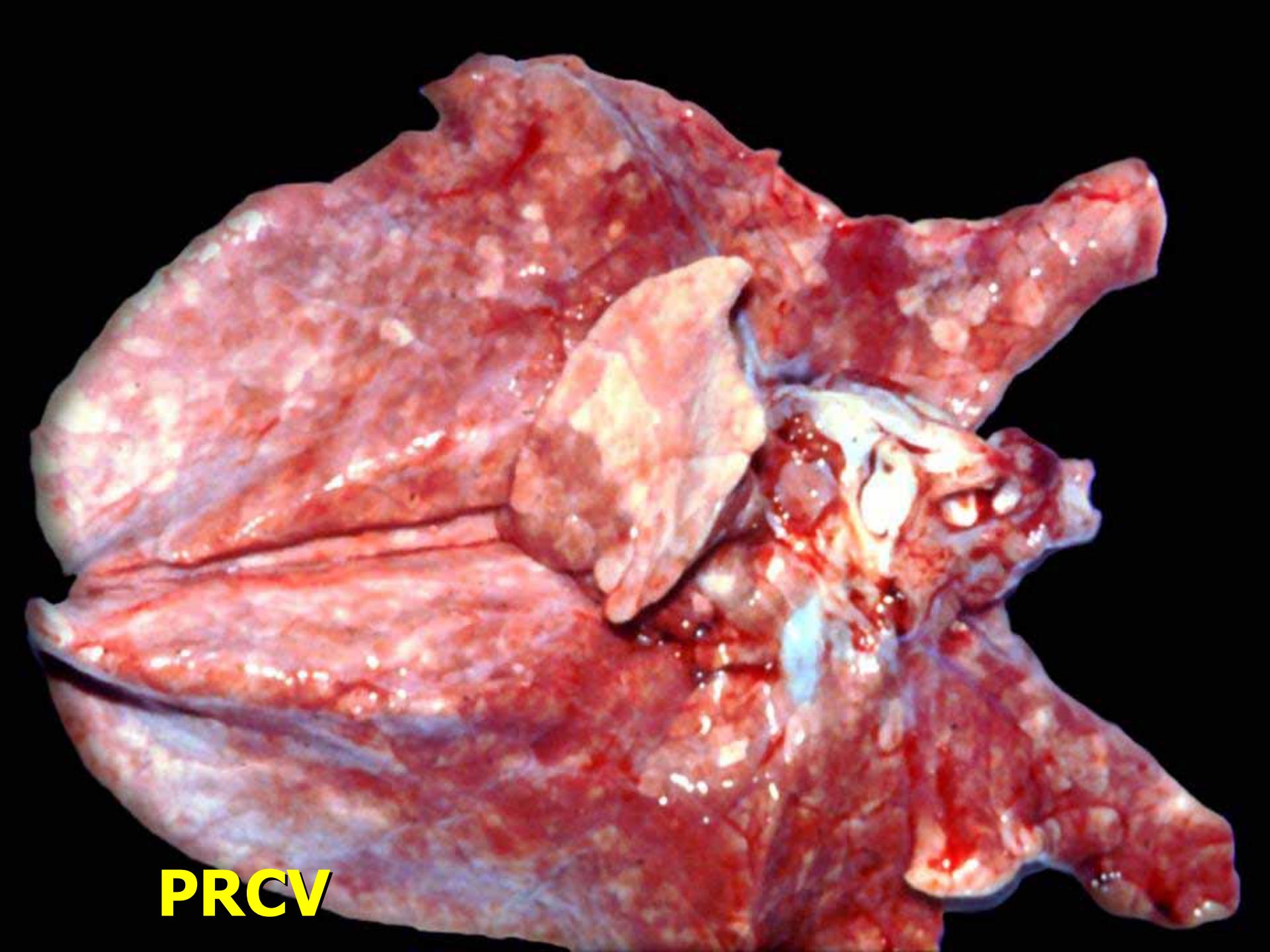
- Deletion mutant of TGE virus
- Replicates in epithelium of nasal cavity and airways
- Shed in nasal secretions for up to 10 days
- Virus is transient in lungs

Symptoms and Lesions of PRCV

- Symptoms
 - usually none **seroconversion to TGE**
 - mild dyspnea, pyrexia, coughing
- Lesions
 - slight increased firmness of apical, cardiac lobes
 - mild interstitial pneumonia with necrosis of epithelium of terminal airways

Diagnosis of PRCV

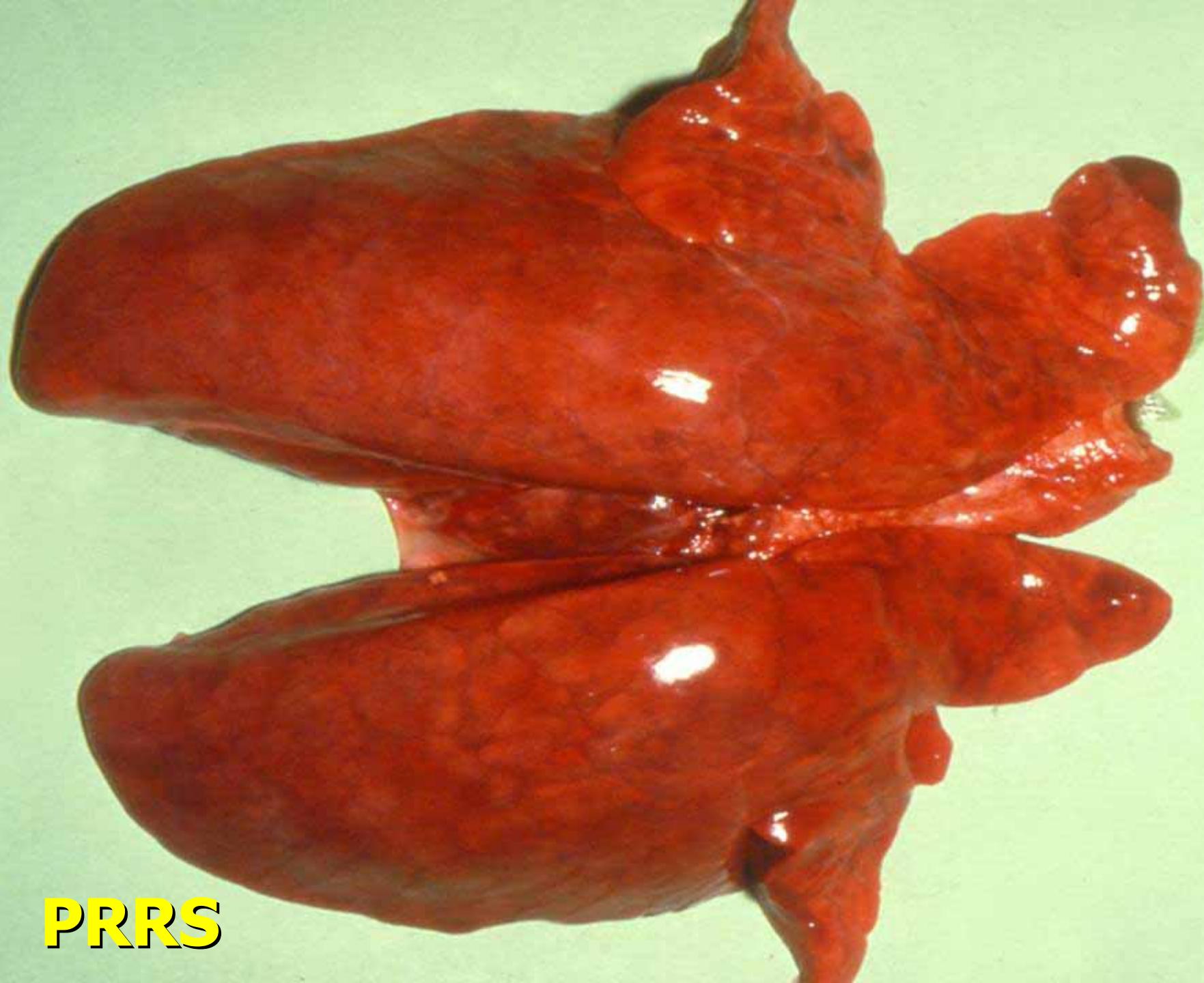
- Typical lesions
- Demonstration of PRCV or antigen
 - FA lung
 - VI nasal swabs, lung
 - Differentiate from TGE: cDNA probe
 - PCR followed by cDNA probe
- Serology
 - SN for TGE and on sera > or = 1:64
 - Competitive ELISA



PRCV



PMWS



PRRS



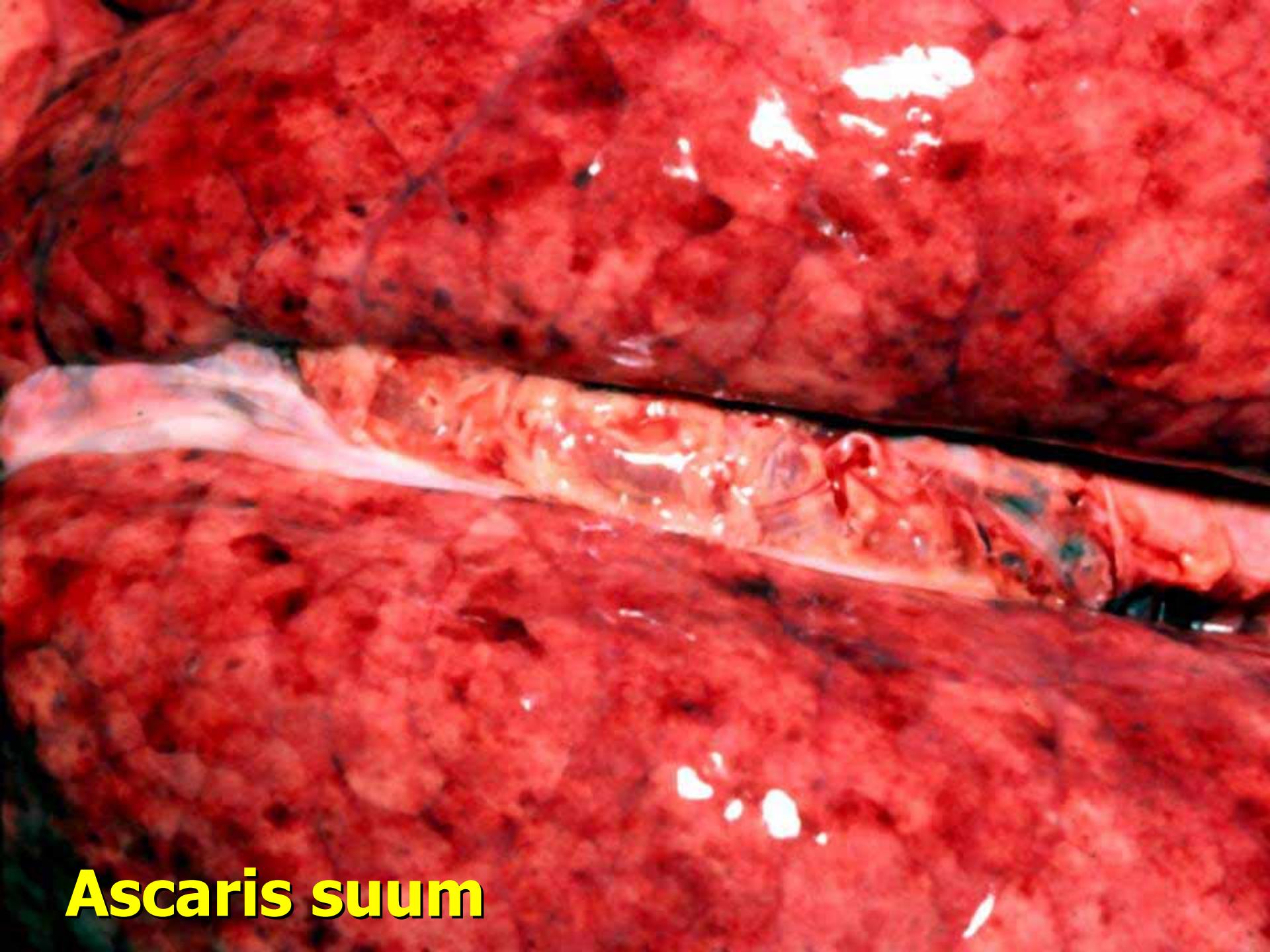
Pseudorabies



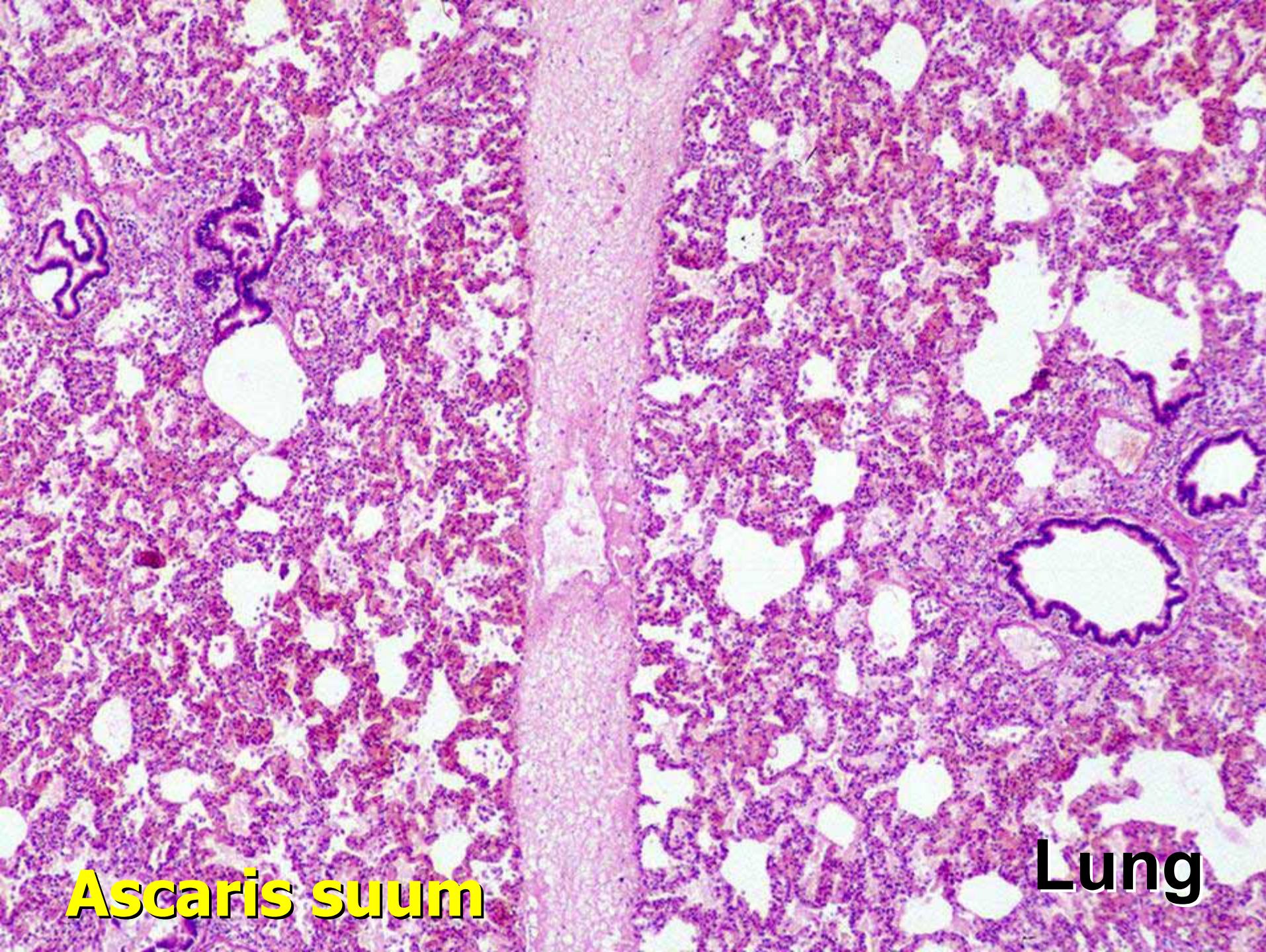
SIV + PRRSV



Ascaris suum

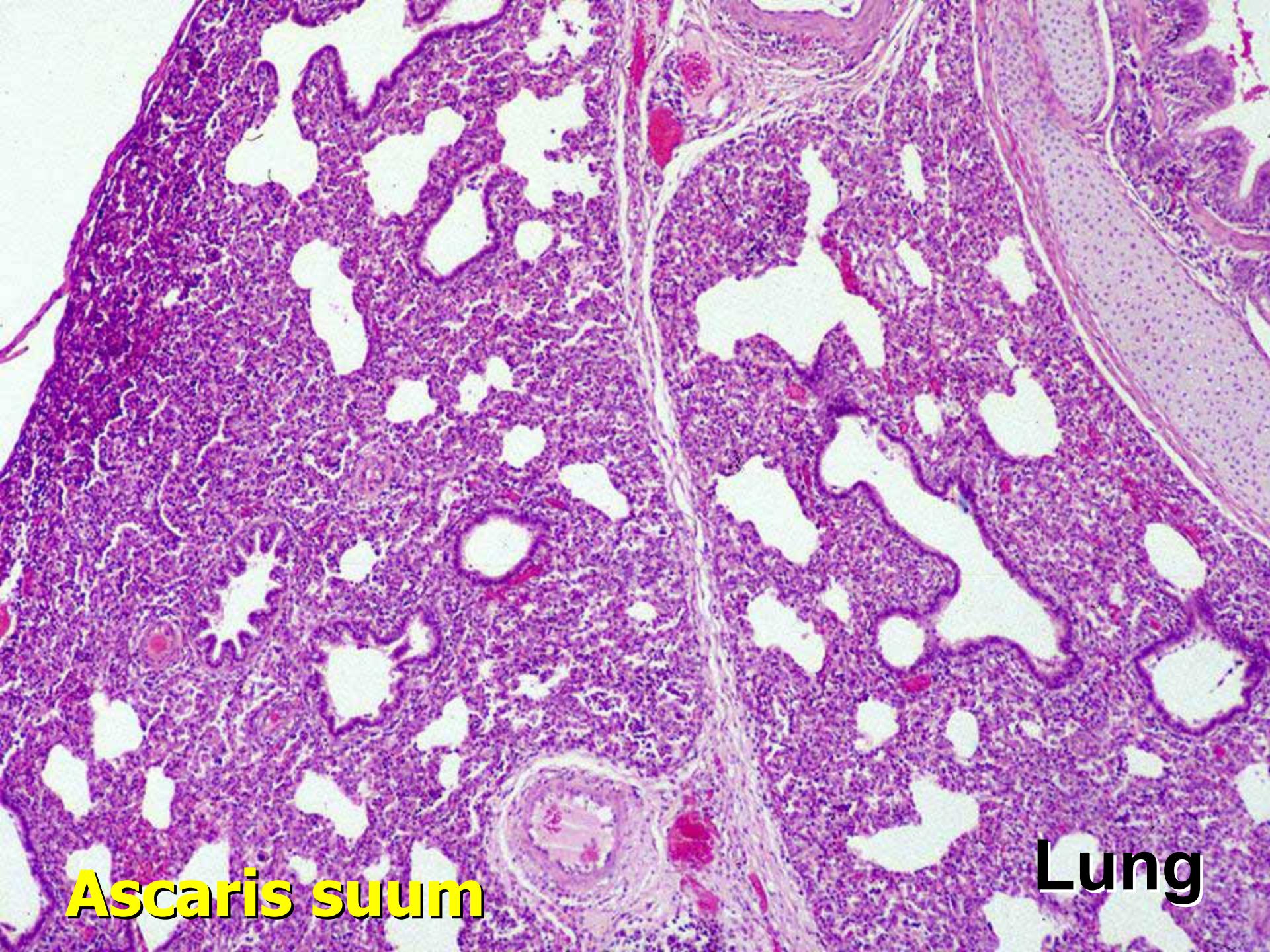


Ascaris suum



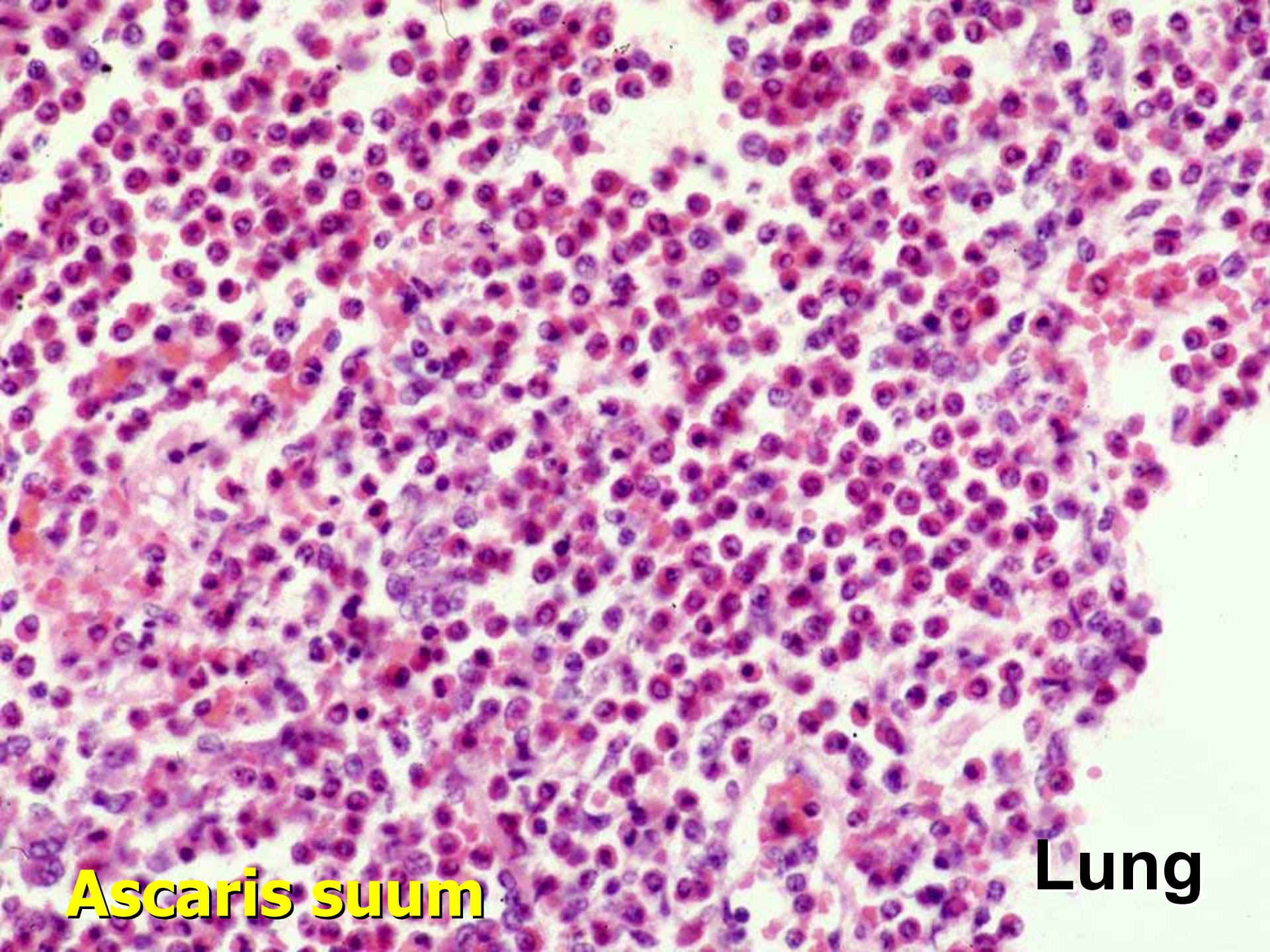
Ascaris suum

Lung



Ascaris suum

Lung

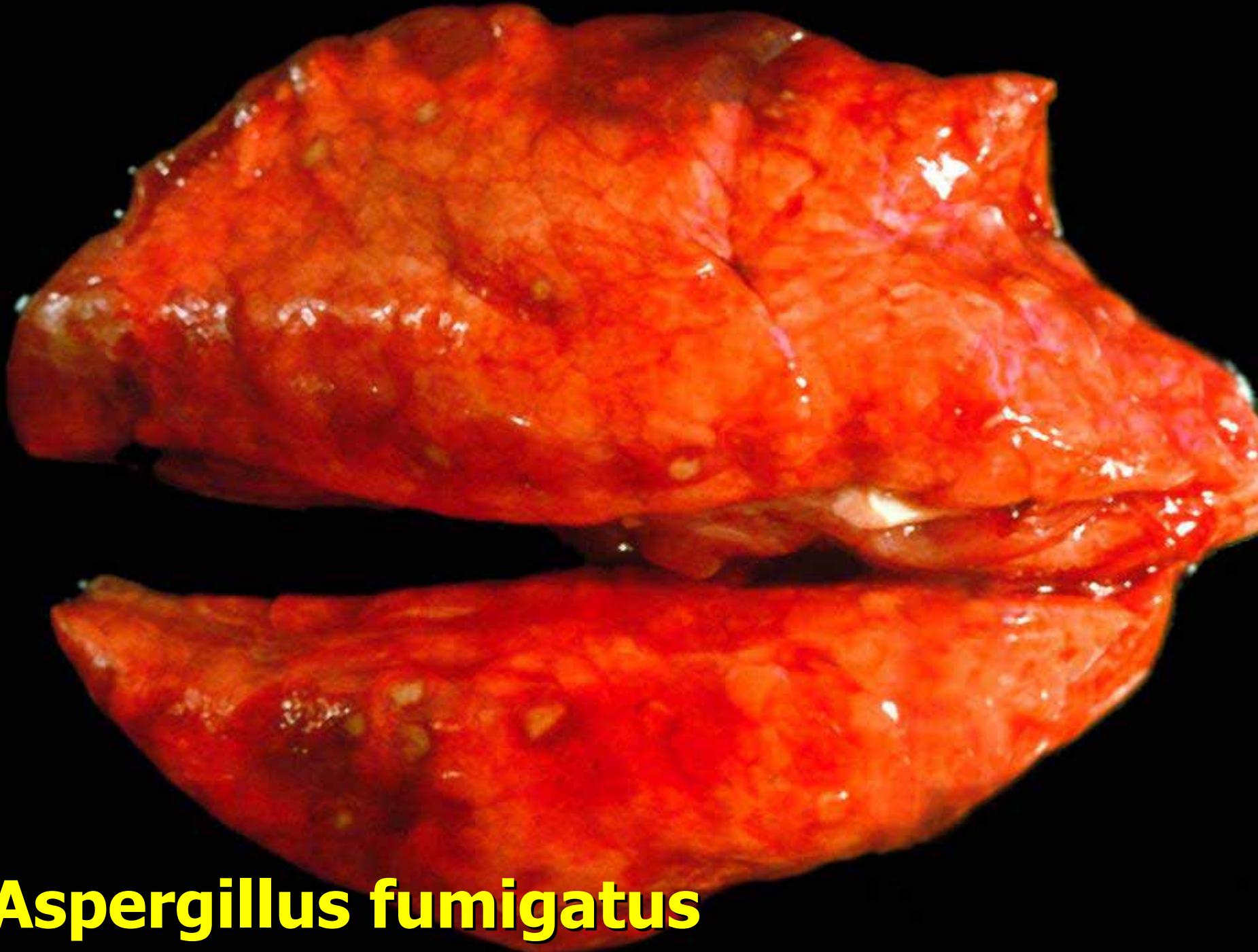


Ascaris suum

Lung



A. suis

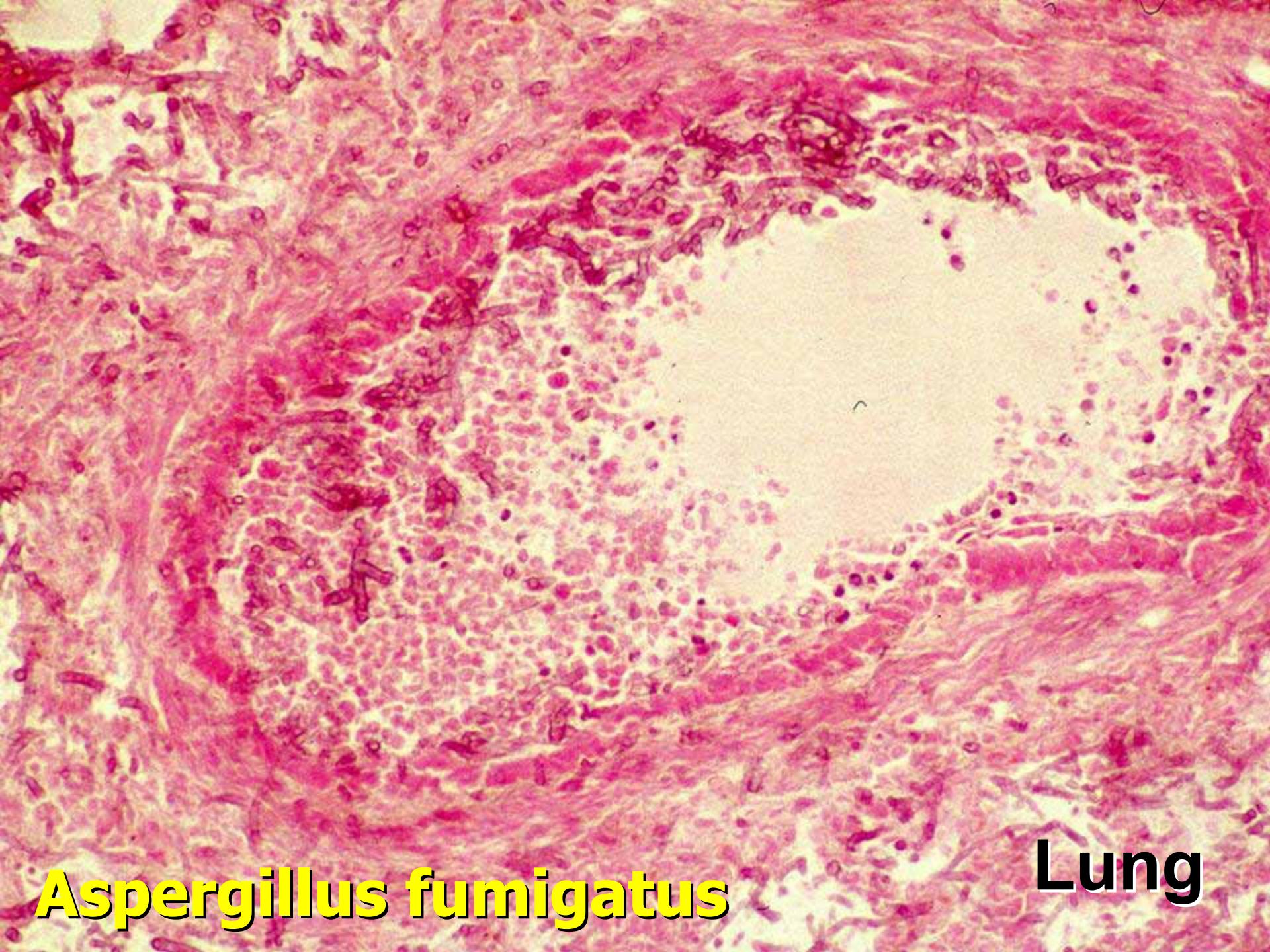


Aspergillus fumigatus



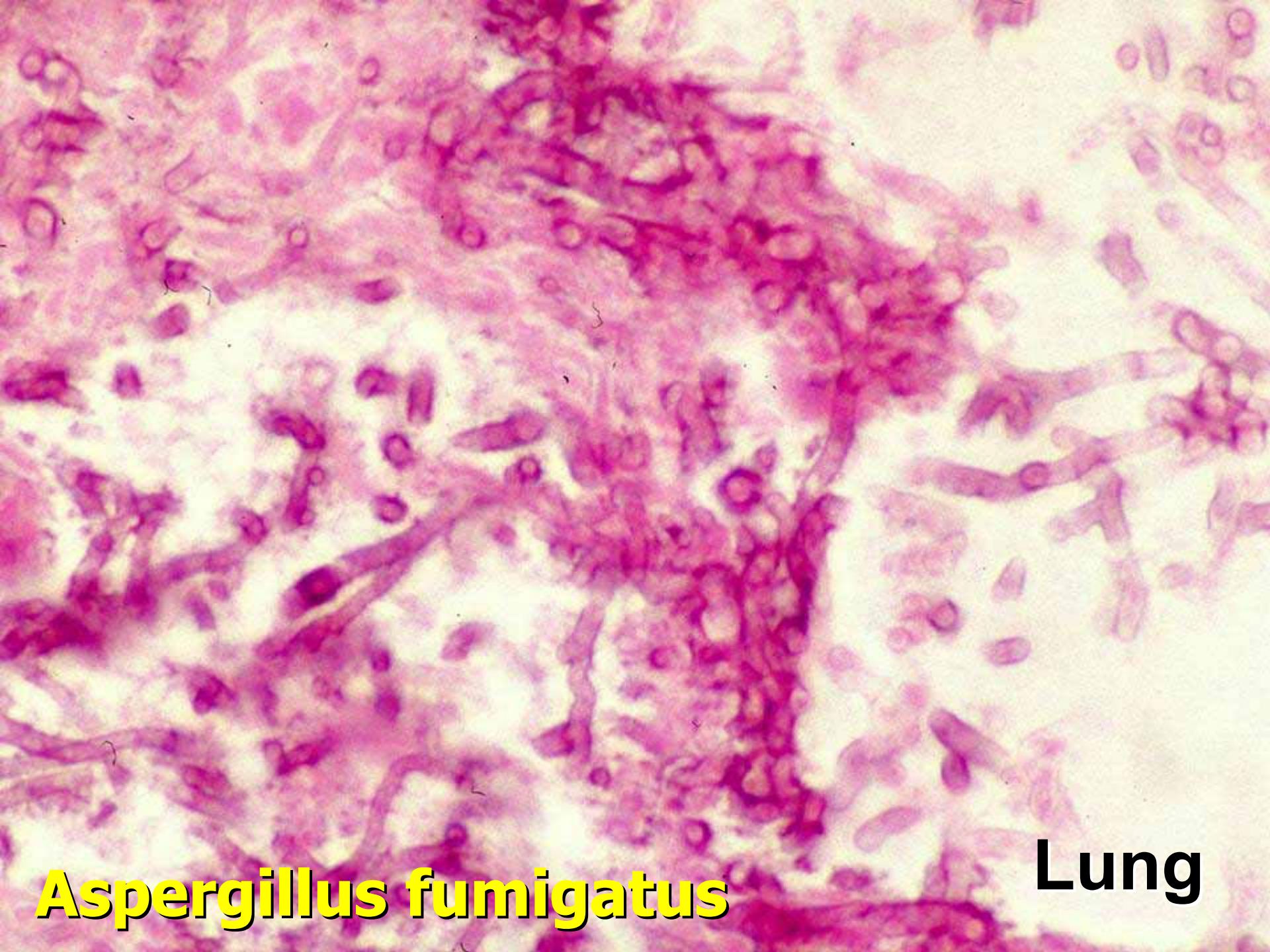
Aspergillus fumigatus

Lung



Aspergillus fumigatus

Lung



Aspergillus fumigatus

Lung



Mycobacterium avium



Mycobacterium avium



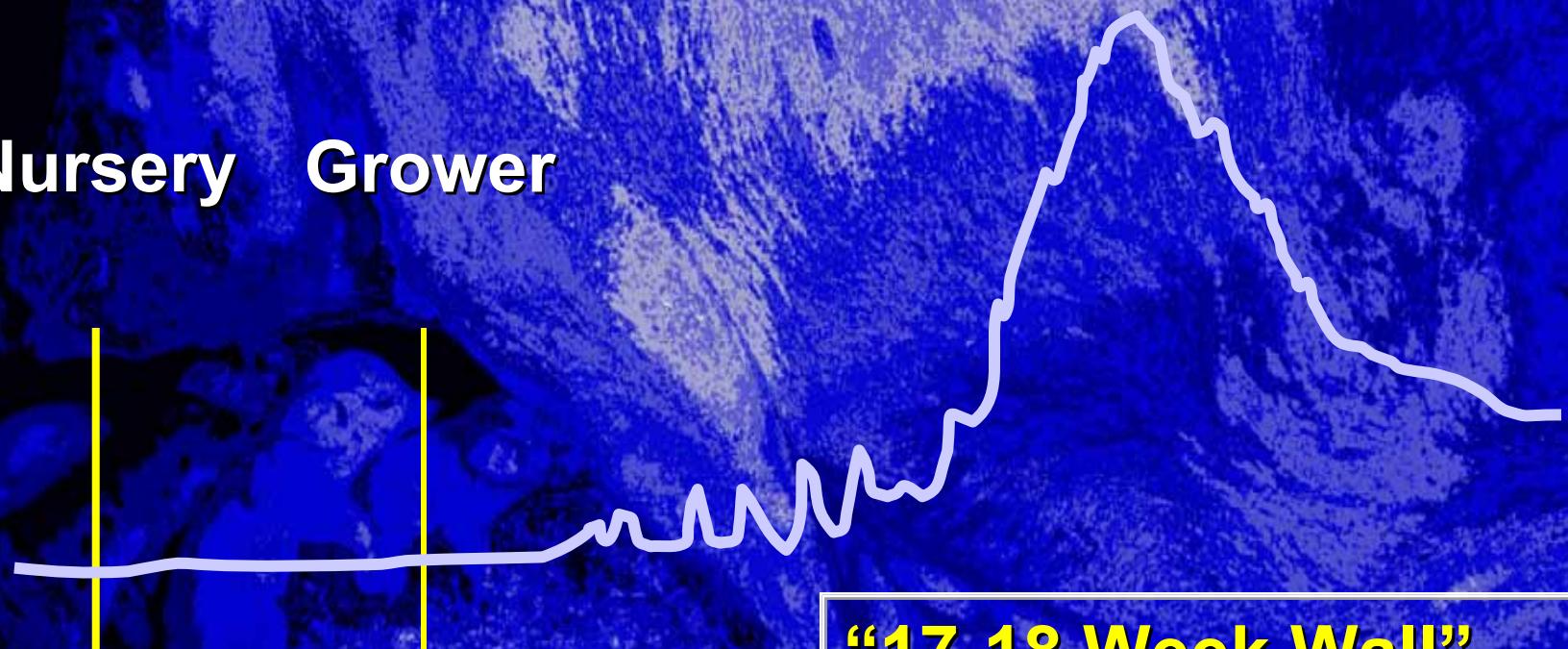
Bacterial Pneumonia in Swine

Infection versus Disease

- **subclinical upper respiratory bacterial infection ≠ pneumonia**
- **risk factors for bacterial pneumonia**
 - **impaired lung defenses**
 - **level of challenge**
 - **virulence of bacterial organism**

Bacterial Initiators of Porcine Respiratory Disease Complex

Nursery Grower



“17-18 Week Wall”
increased morbidity
increased mortality

Lung Defenses

Native

Mucociliary Apparatus

Alveolar macrophages

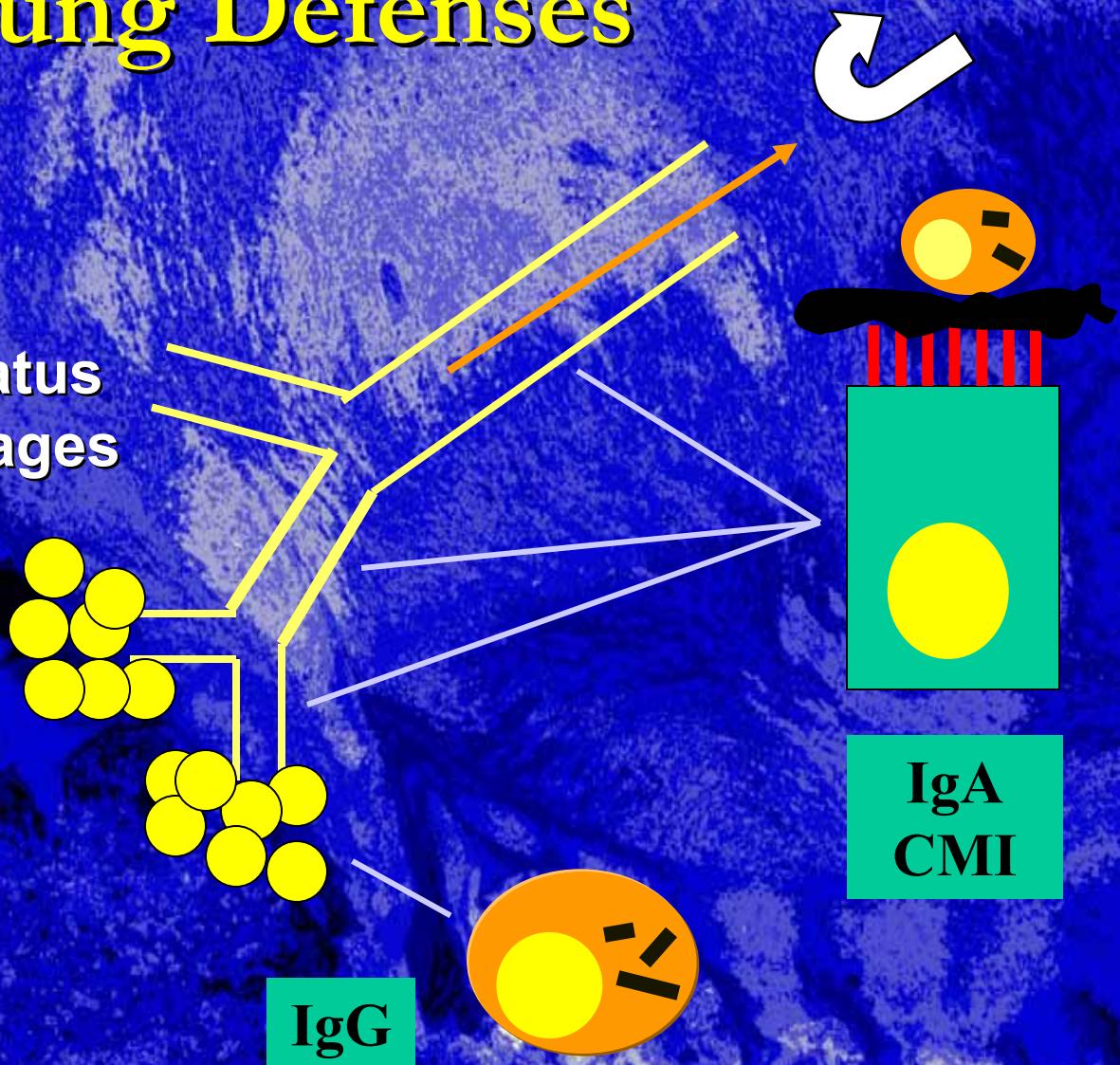
Inflammation

Acquired

IgA

CMI

IgG



Damage to Lung Defenses

Mucociliary
Apparatus

M. hyopneumoniae

SIV

PRV

PRCV

PCV?

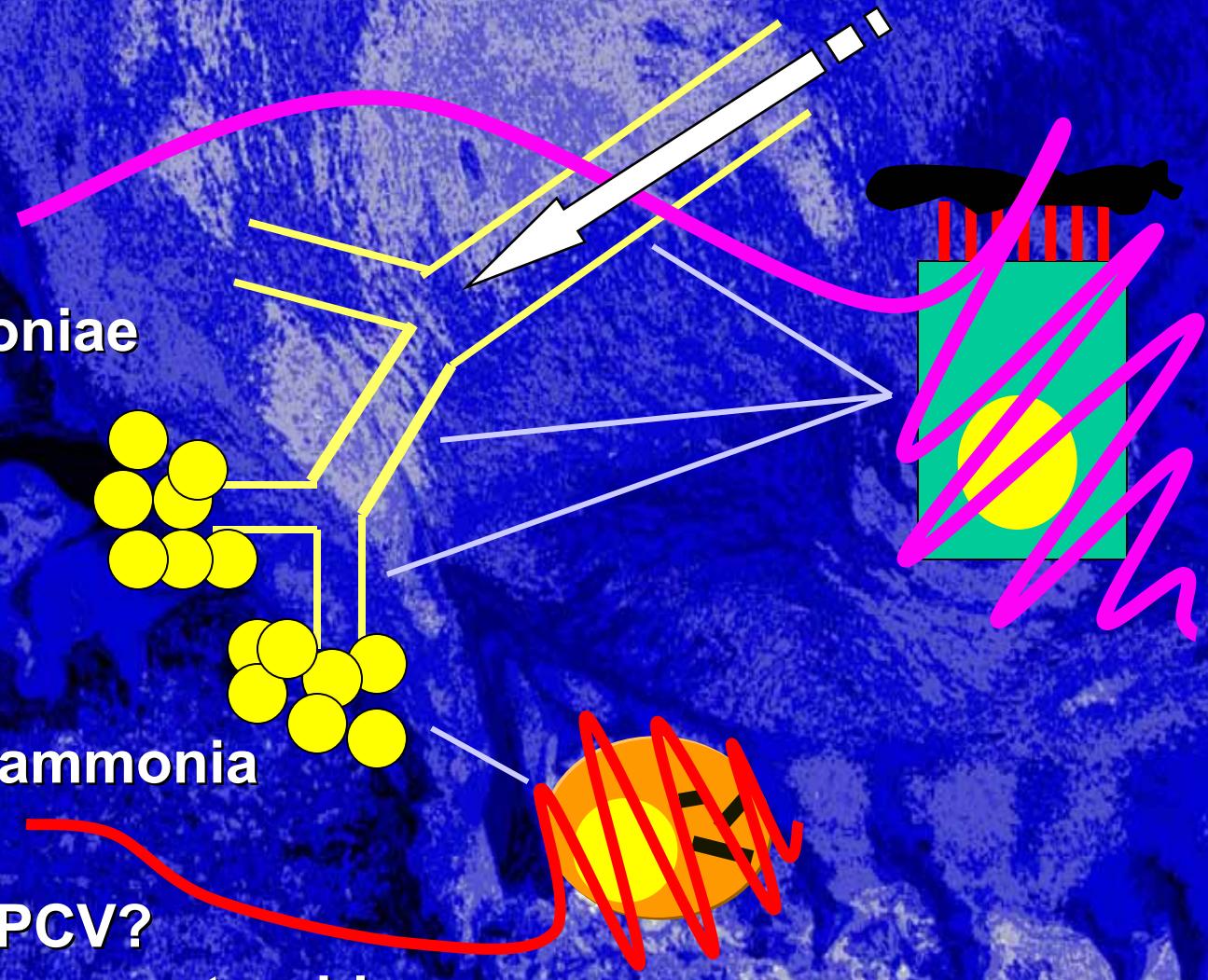
Chilling

high levels of ammonia

Macrophages

PRRSV, APP, PCV?

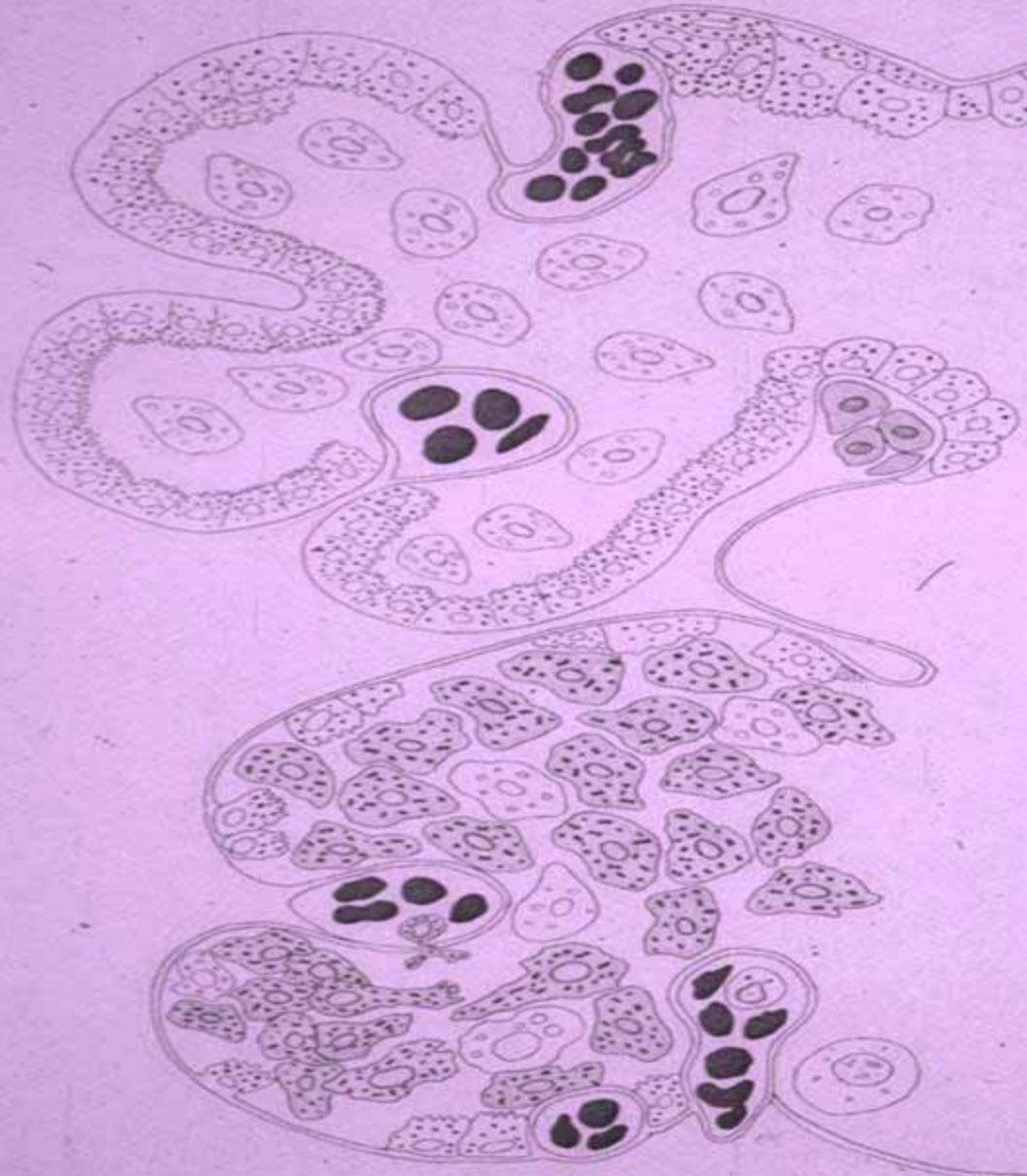
Stress, endogenous steroids



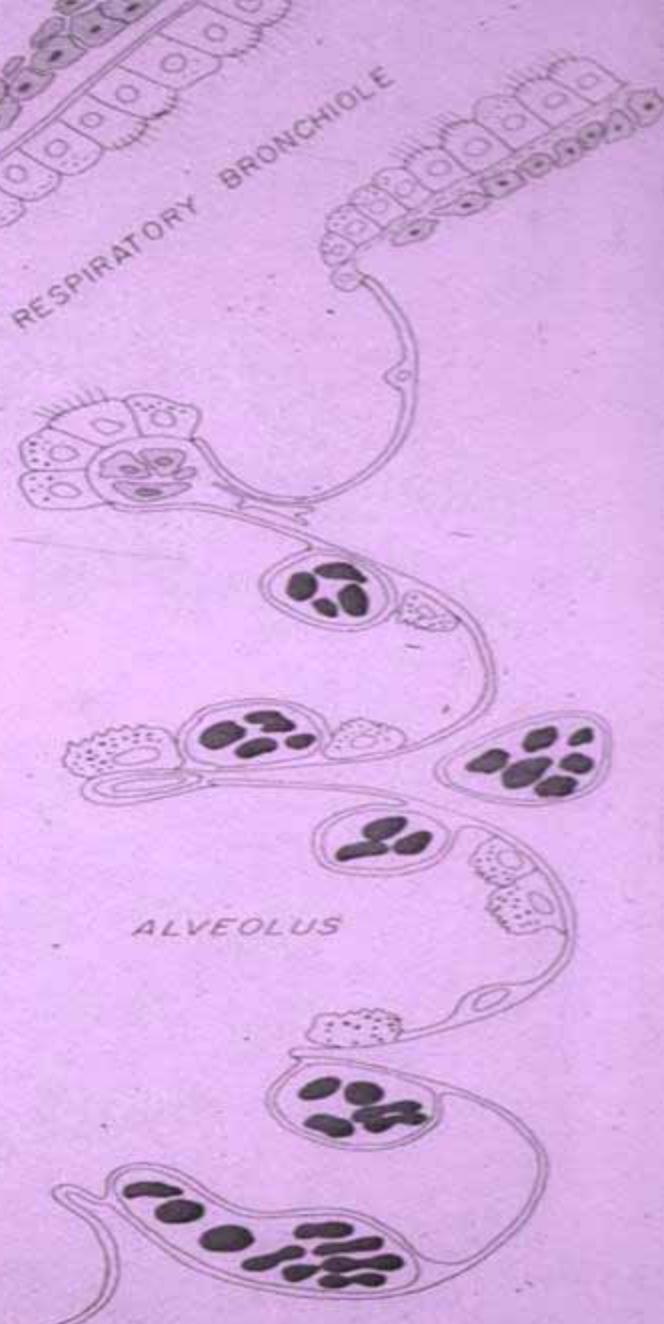
PRDC is a “Complex”

- Primary pathogens (Initiators)
 - *M. hyopneumoniae*
 - *A. pleuropneumoniae*
 - SIV
 - PRRSV
 - PRV
 - PRCV
 - PCV
- Secondary pathogens (Followers)
 - *P. multocida*
 - *S. suis*
 - *H. parasuis*
 - *B. bronchiseptica*
 - *A. pyogenes*
 - ***A. pleuropneumoniae*

EPITHELIALIZATION



PNEUMONIA

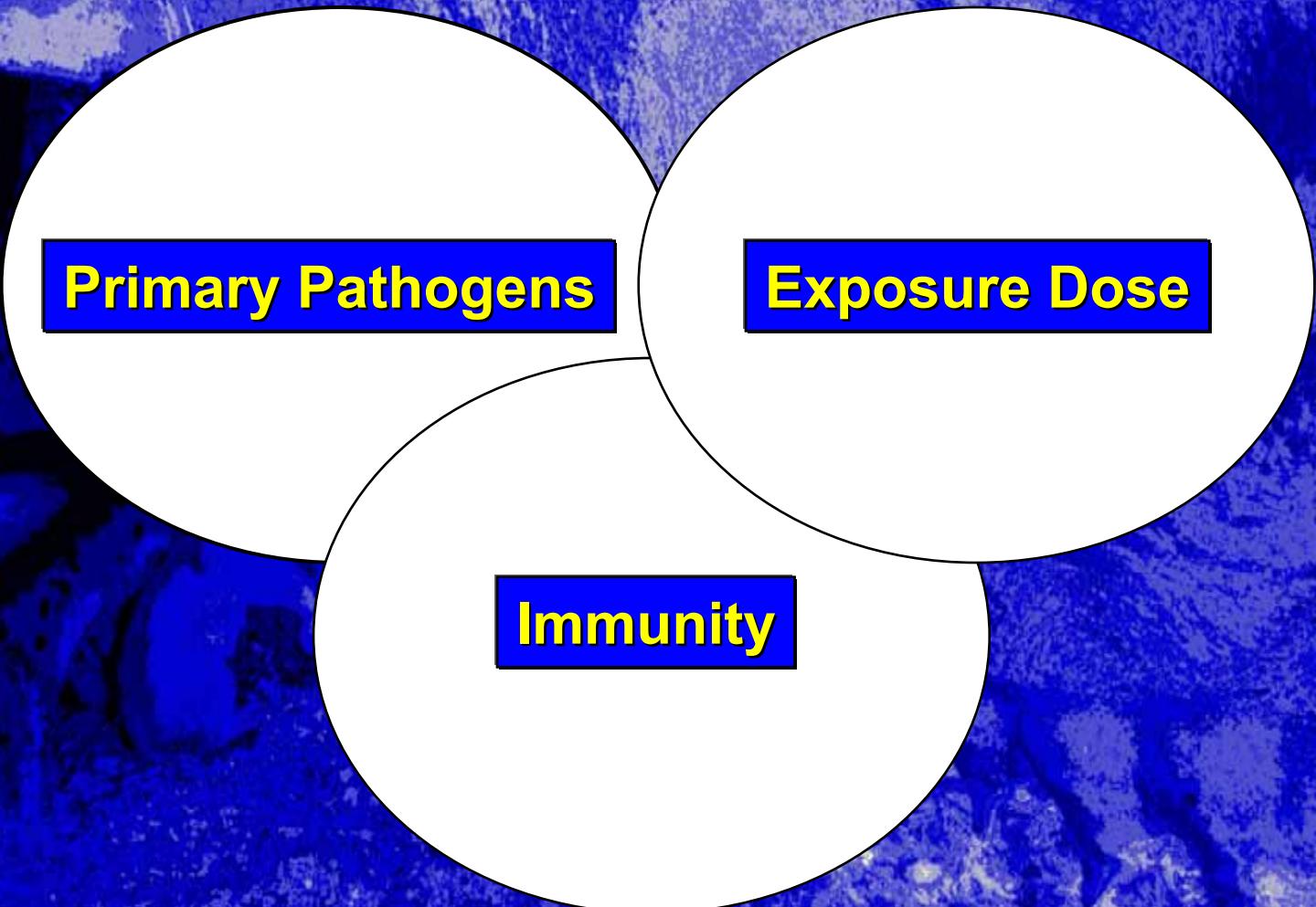


NORMAL

RESPIRATORY BRONCHIOLE

ALVEOLUS

Risk Factors for PRDC



Prevention and Control of PRDC

- **focus on Initiators**
- **farm or system specific**
 - **identify initiators**
 - **evaluate shedding and immunity**
 - **evaluate management practices, exposure**
 - **develop plan**
 - **work plan**
 - **monitor and re-evaluate**

Increased Bacterial Challenge

- High stocking density
- Frequent mixing of pigs
- Large variation in age in pens or rooms
- ☰ 200 pigs per air space
- Low ventilation rates
- Non-solid partitions between pens
- Breaches in biosecurity

Virulence of Organisms

- Primary Inhaled
 - *M. hyopneumoniae*
 - *A. pleuropneumoniae*
 - *B. bronchiseptica*
- Secondary Inhaled
 - *P. multocida*
 - *S. suis*
 - *H. parasuis*
 - *M. hyorhinis**
 - *A. pyogenes**
- Primary Blood-borne
 - *S. choleraesuis*
 - *A. suis*
 - *A. pyogenes*

Mycoplasma hyopneumoniae

- fastidious pleomorphic bacterium
- infects only pigs
- colonizes respiratory epithelium - cilia
 - catarrhal bronchopneumonia - resolves ↗75d
 - secondary purulent bacterial bronchopneumonia
 - “Enzootic pneumonia”
 - protracted disease
 - marked reduction in rate-of-gain

Mycoplasma hyopneumoniae

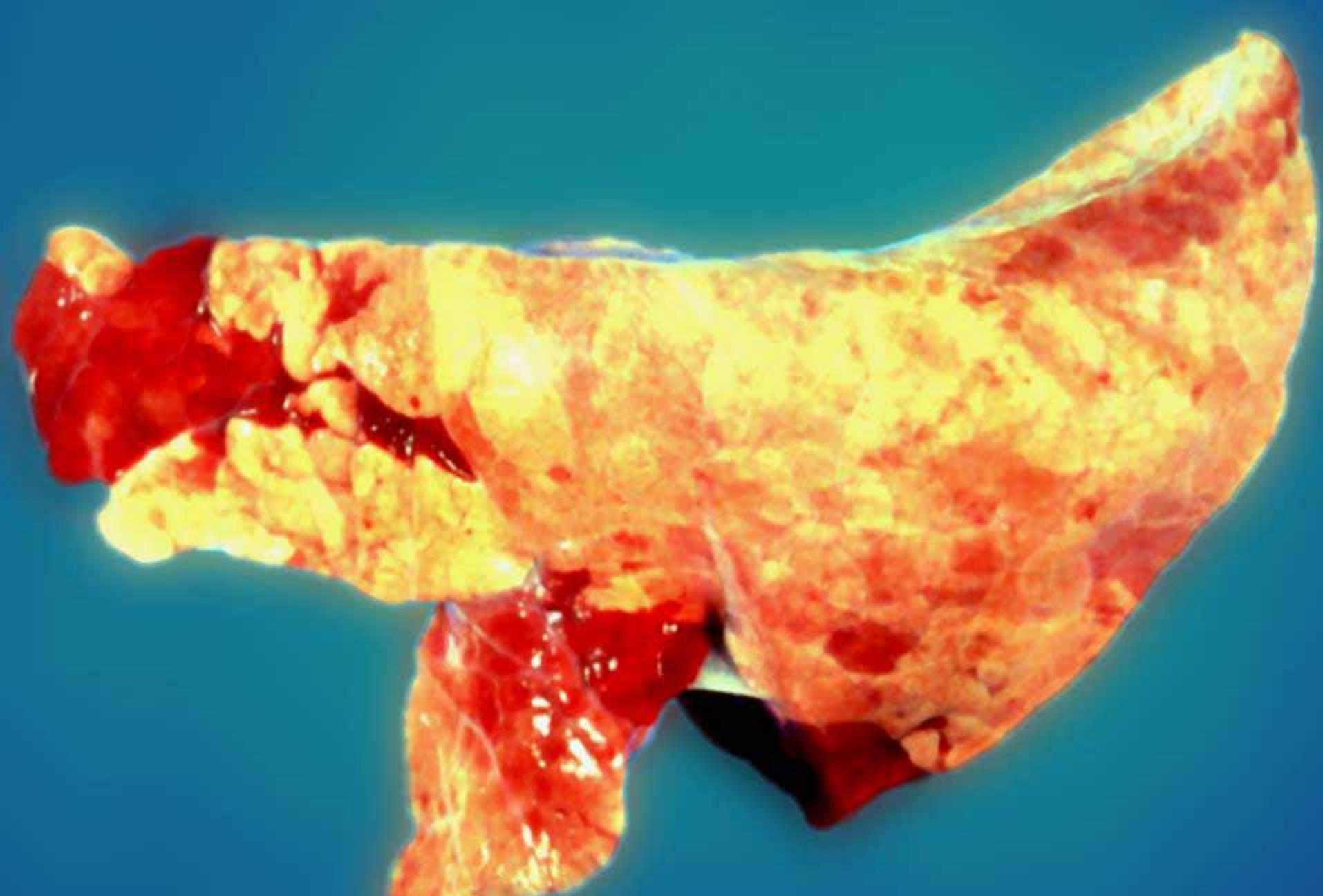
Epidemiology

- **Transmission is inefficient**
 - Nose-to-nose
 - Clinical disease ⊕ Subclinical disease
 - Older animals ⊕ Young animals
 - Aerosol
- **Disease is dose-dependent**
 - Subclinical infection vs. clinical disease
 - Incubation period: 11 days to 6 weeks

Mycoplasma hyopneumoniae

Diagnosis

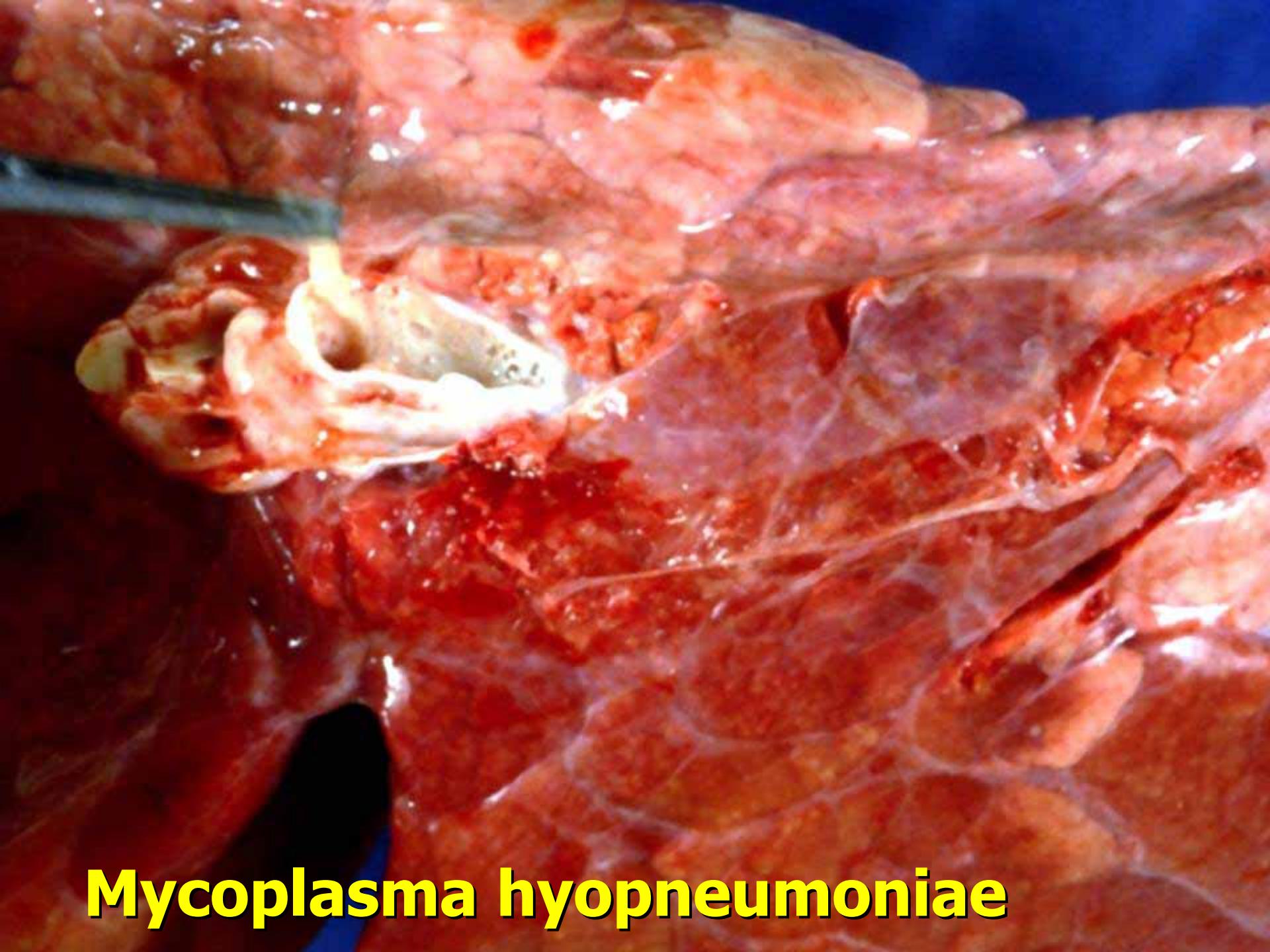
- **Clinical signs**
 - Slowly spreading nonproductive cough
 - Depression in growth rate
 - Dyspnea, anorexia, death
- **Lesions:** Catarrhal bronchopneumonia
- **Confirmation:**
 - Antigen: Tissue FA, IHC
 - Nucleic Acid: PCR
 - *Antibodies: CF, ELISA



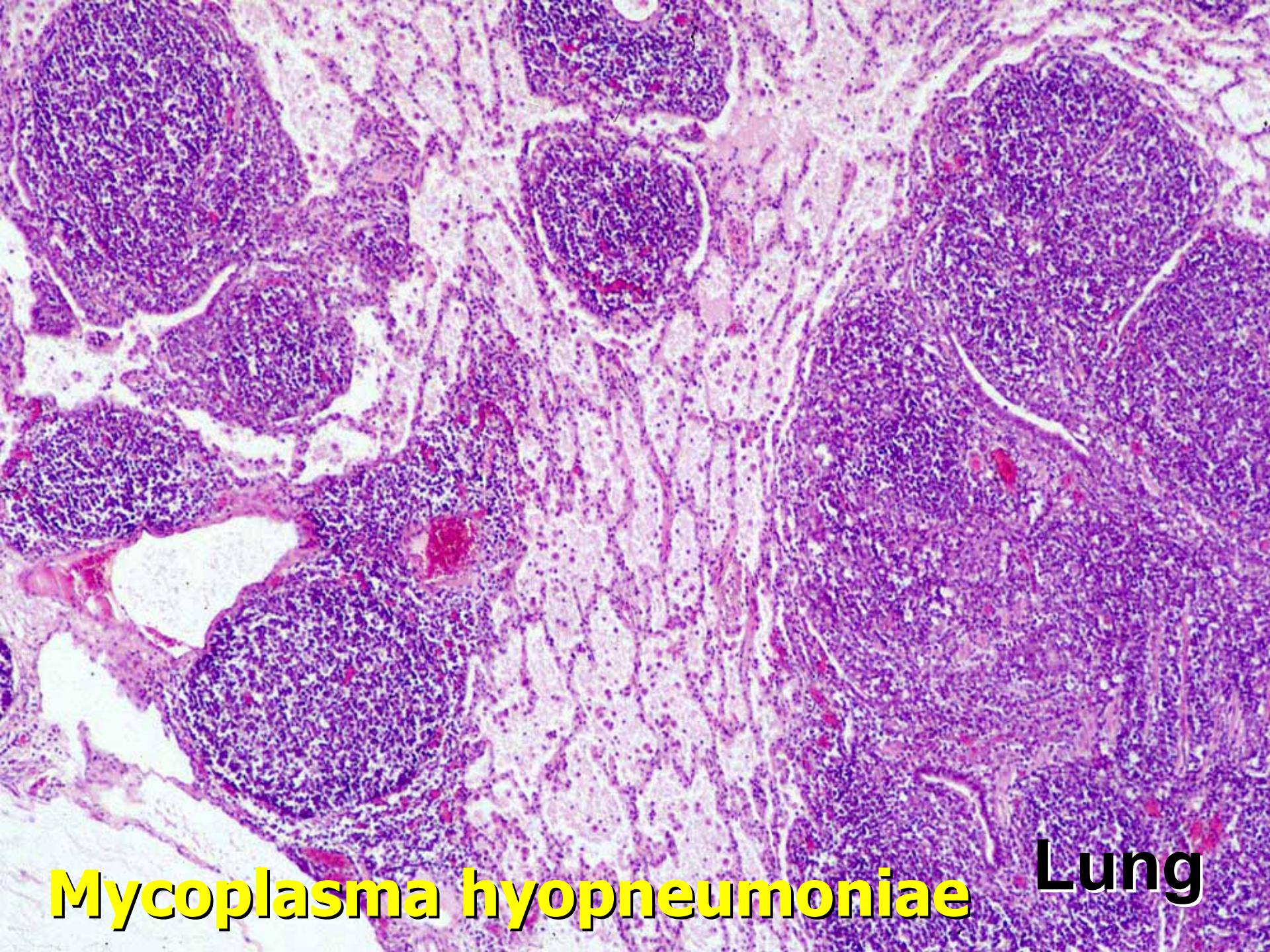
Mycoplasma hyopneumoniae



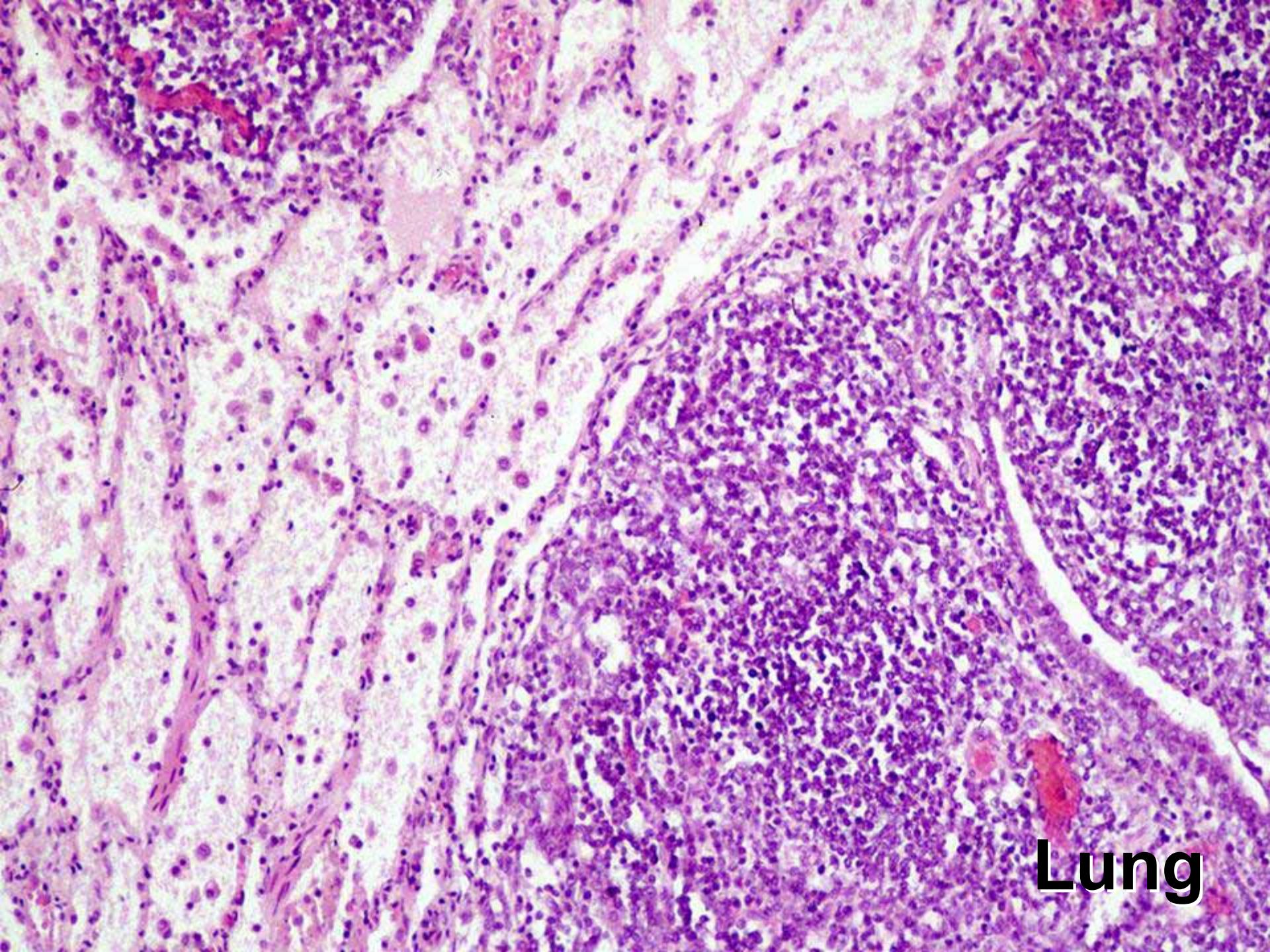
Mycoplasma hyopneumoniae



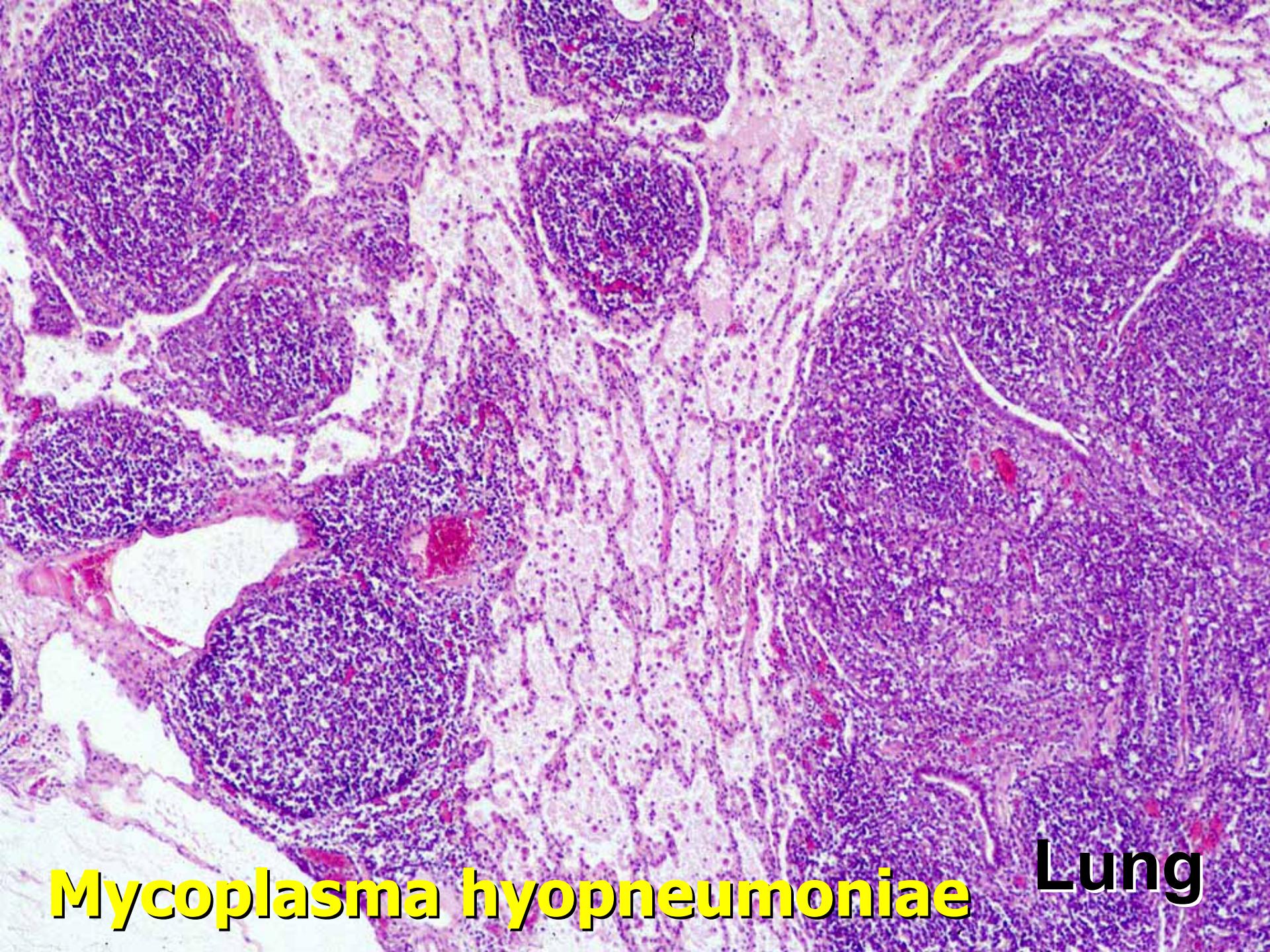
Mycoplasma hyopneumoniae



Mycoplasma hyopneumoniae Lung



Lung



Mycoplasma hyopneumoniae Lung

Mycoplasma hyopneumoniae

Trachea



Trachea

Mycoplasma hyopneumoniae

Secondary Inhaled Pathogens

Purulent Bronchopneumonia

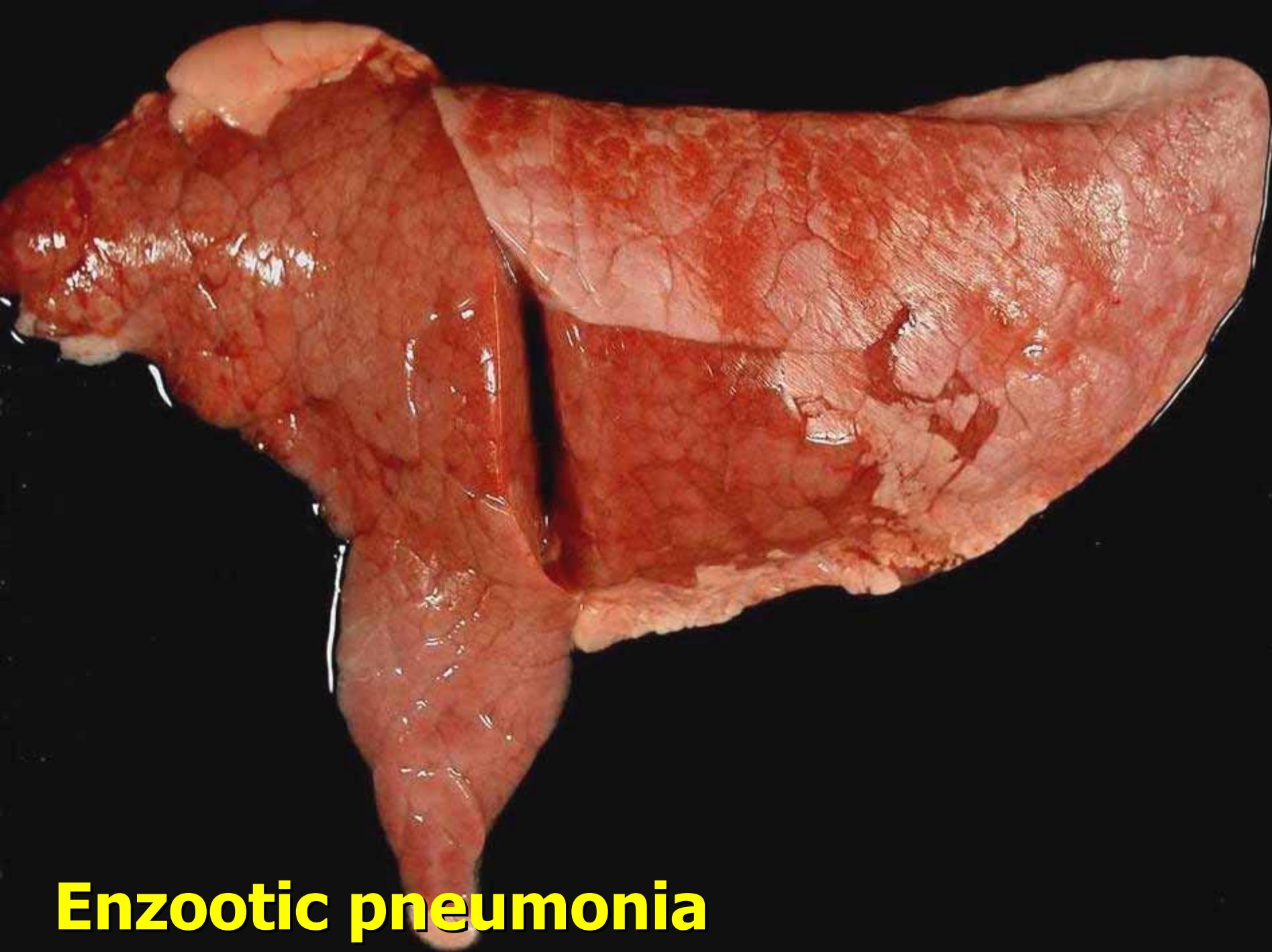
- upper respiratory commensals
- follow Mycoplasma or viral infections
- “Enzootic Pneumonia” - may not be!
- most common:
 - *P. multocida*
 - *S. suis*
 - *H. parasuis*
- diagnosis: culture
 - *M. hyorhinis*
 - *A. pyogenes*
 - *B. bronchiseptica*

Pasteurella multocida

- common in nearly all swine herds
- non-toxigenic and toxigenic strains
- most common bacterial isolate from pneumonic lungs in slaughter swine
- lung isolates
 - most are capsular type A
 - most are serotypes 3 or 5 (of 16 total)
 - toxin as a virulence factor??
 - some strains: pleuritis, abscessation



Enzootic pneumonia



Enzootic pneumonia



A. pyogenes

Bordetella bronchiseptica

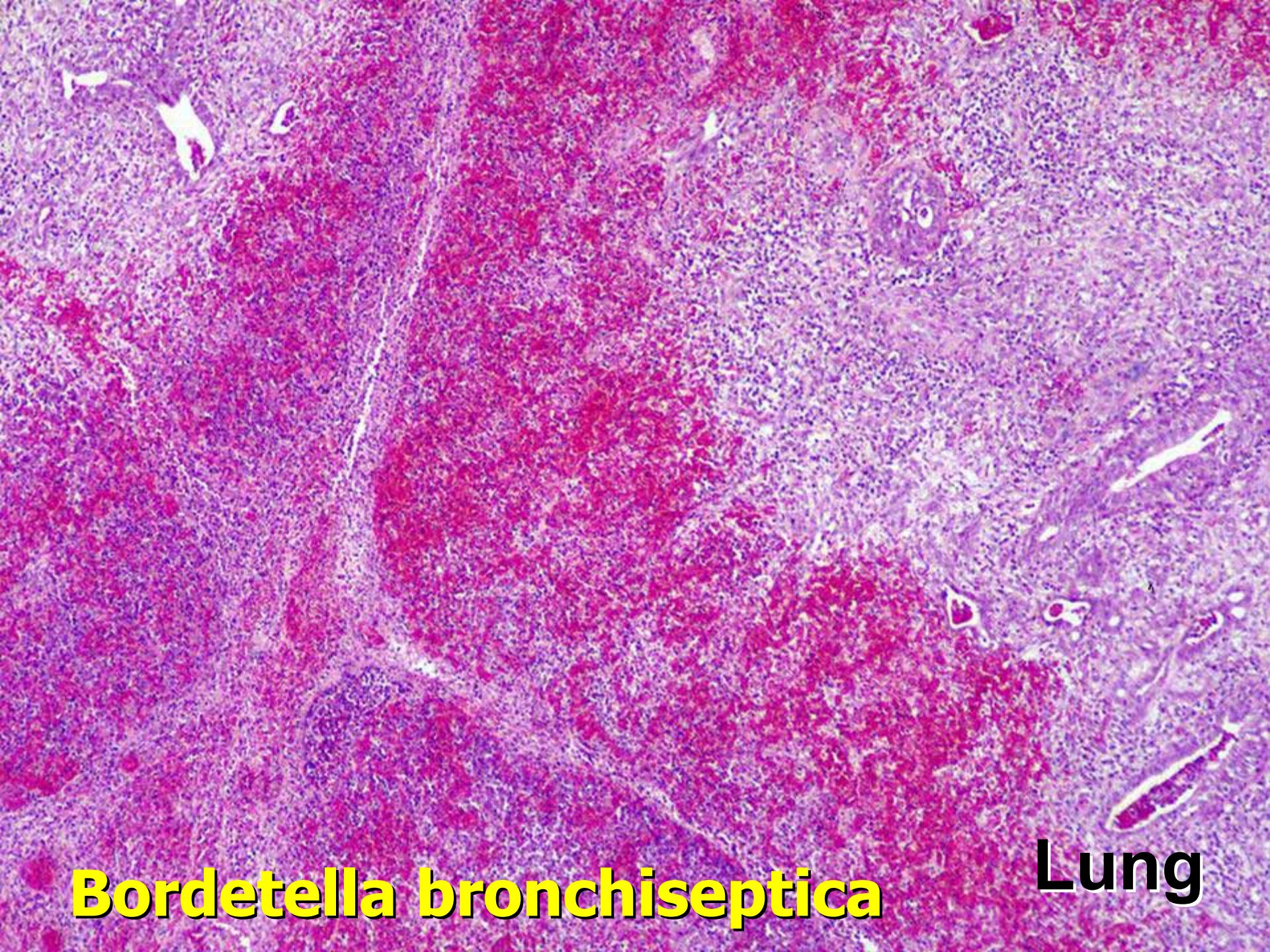
- primary or secondary inhaled pathogen
- Pathogenesis: Colonisation and destruction of cilia in upper respiratory tract, may colonize lung causing broncho-pneumonia (cranial and middle lobes)
- primary pathogen: first few weeks of life
- lobular necrohemorrhagic bronchopneumonia
 - Coughing, sneezing +/- epistaxis and mucopurulent nasal discharge, mild (reversible) turbinate atrophy (regressive atrophic rhinitis), death
- chronic progressive bronchopneumonia
 - Coughing and poor growth



Bordetella bronchiseptica

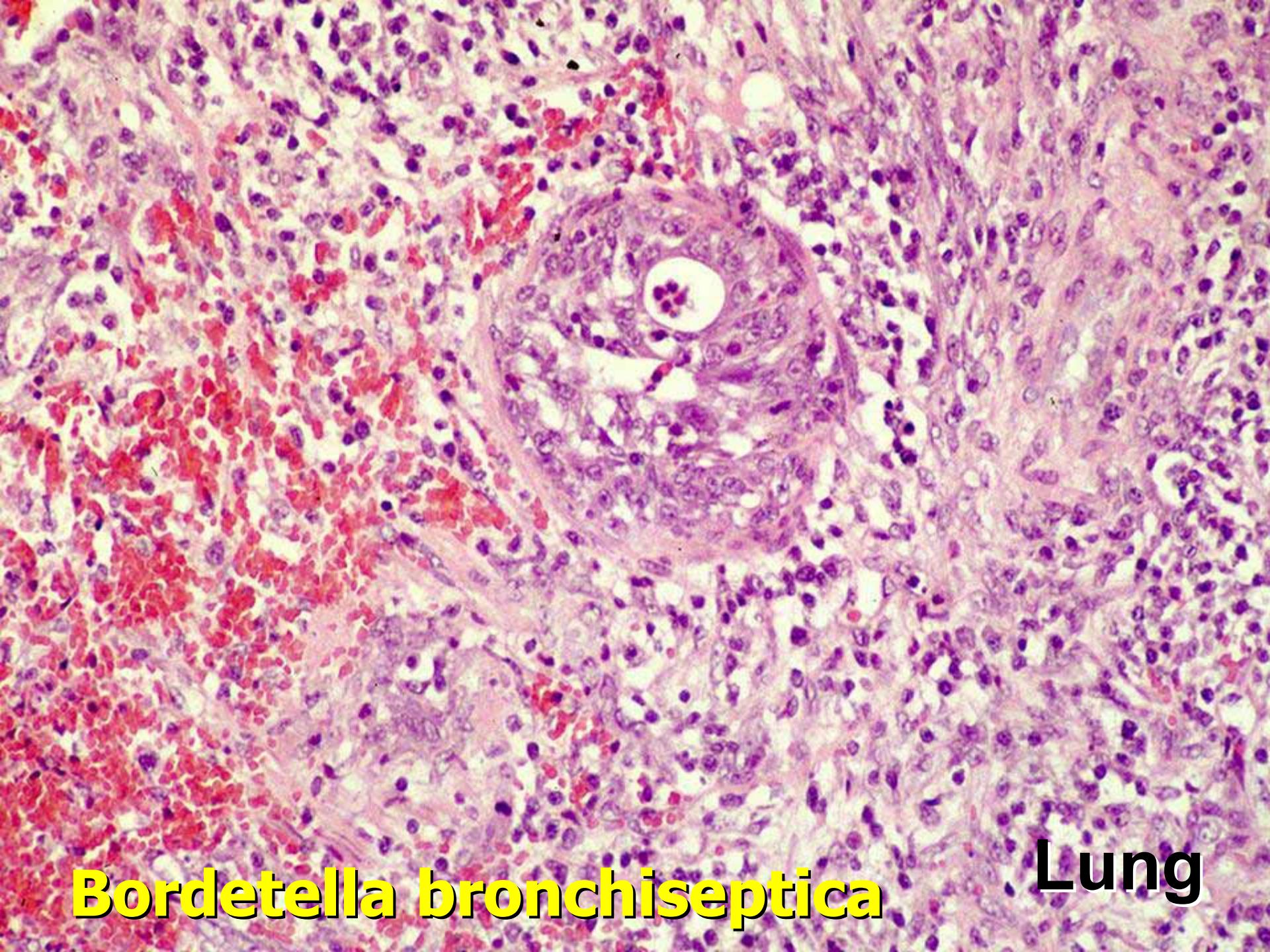


Bordetella bronchiseptica



Bordetella bronchiseptica

Lung



Bordetella bronchiseptica

Lung



Actinobacillus pleuropneumoniae

- fastidious encapsulated coccobacillus found only in swine
- biovar 1: require NAD for growth
- biovar 2: NAD not required for growth
- 15 serotypes
 - predominant serotypes vary by region
 - serotypes and strains vary in virulence

Actinobacillus pleuropneumoniae

Virulence Factors & Epidemiology

- Virulence factors
 - Capsule
 - Endotoxin
 - Exotoxins: APX I, II, III
 - Hemolysin APX I, II
 - Cytolysin APX I, II, III
- Epidemiology
 - Carriers: tonsil, nasal cavity, lung
 - Carriers may be seronegative

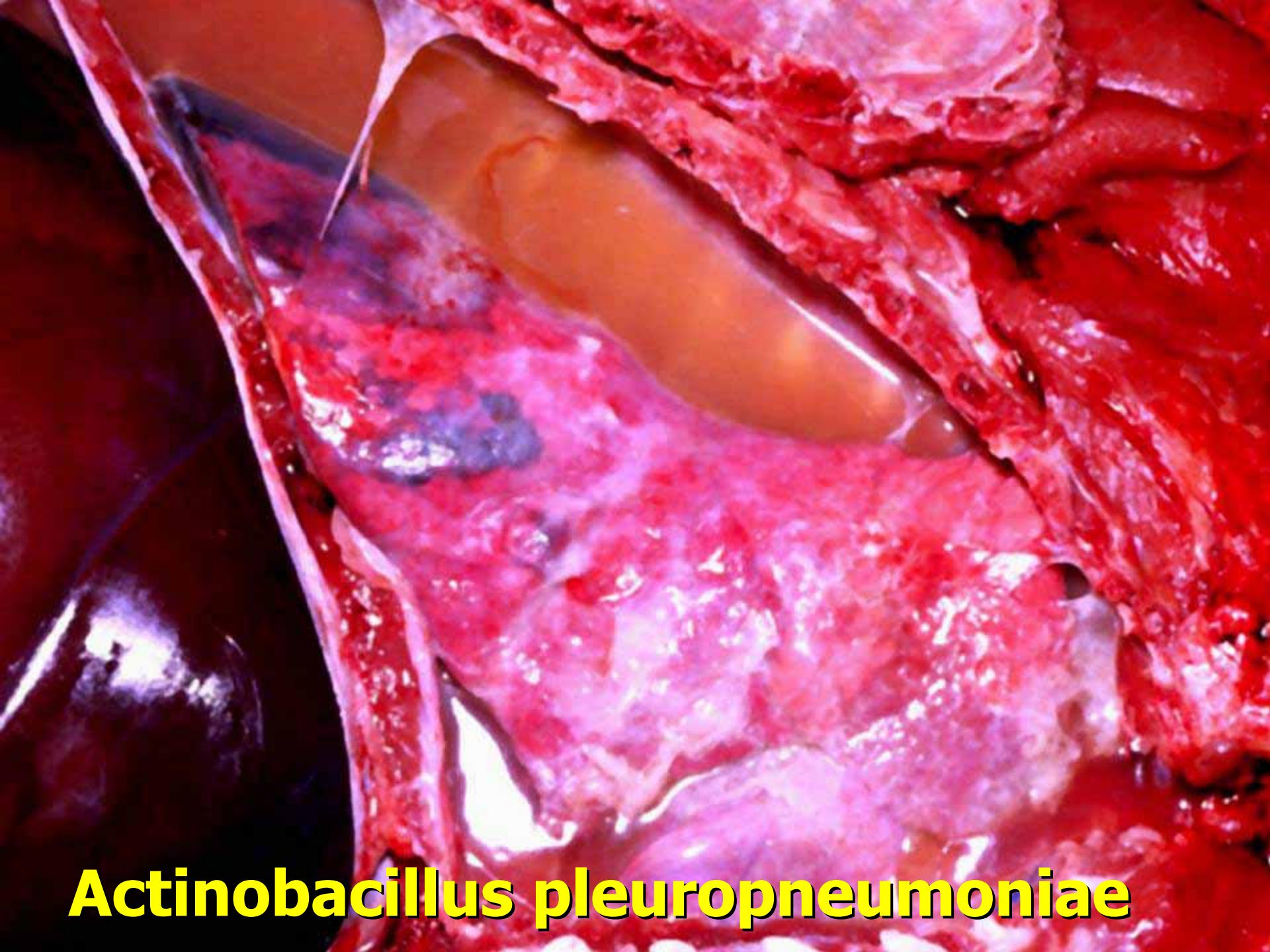
Actinobacillus pleuropneumoniae

Diagnosis

- Clinical signs:
 - most common in 3-6 month-old pigs
 - peracute - chronic
 - pyrexia, dyspnea, hemoptosis, death, anorexia
- Lesions: Fibriononecrotic hemorrhagic pleuropneumonia
- Confirmation:
 - Culture: fresh lung from untreated animals
 - Antigen: Agglutination, Co-agglutination, ELISA
 - Antibodies: CF, HN, ELISA



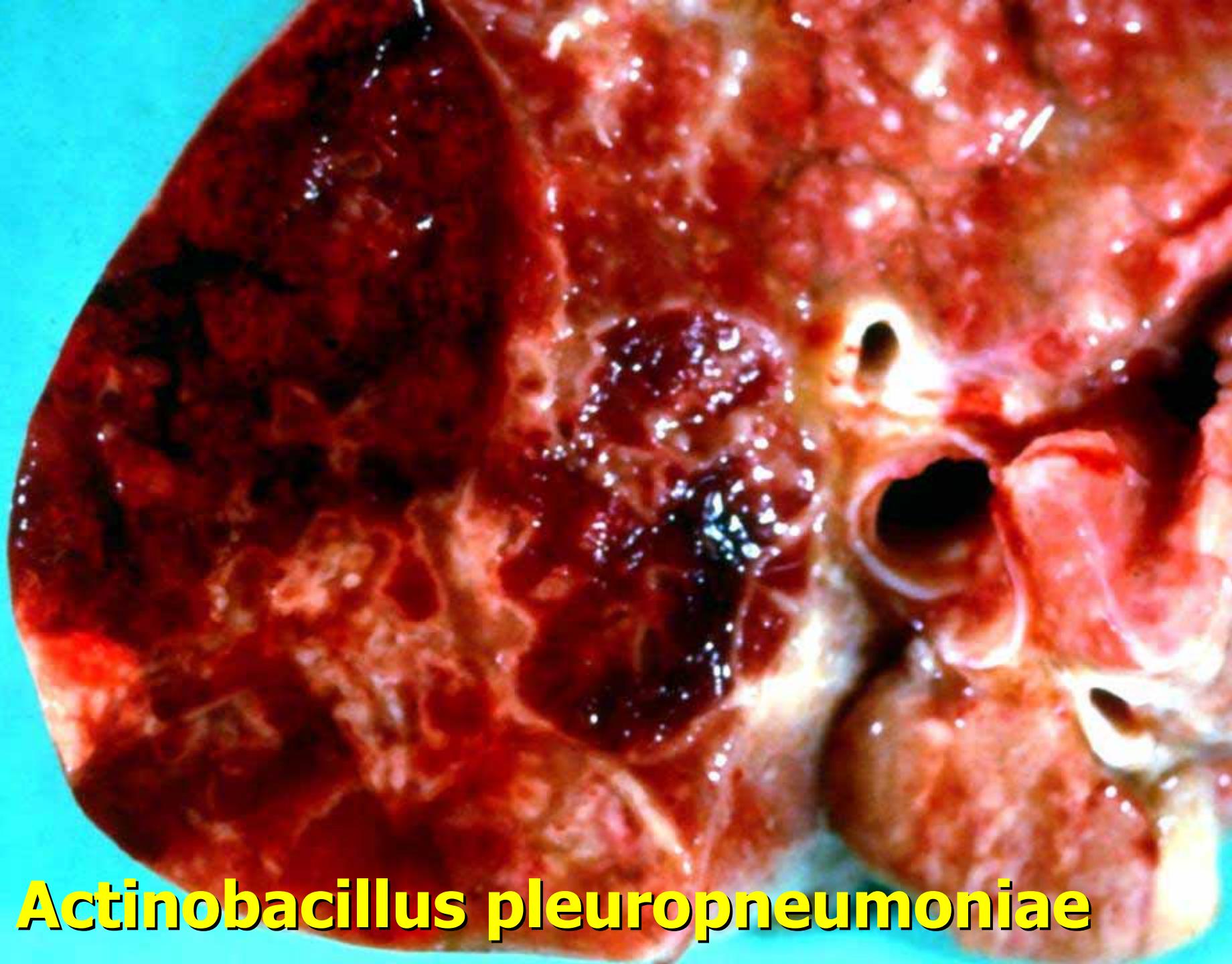
Actinobacillus pleuropneumoniae



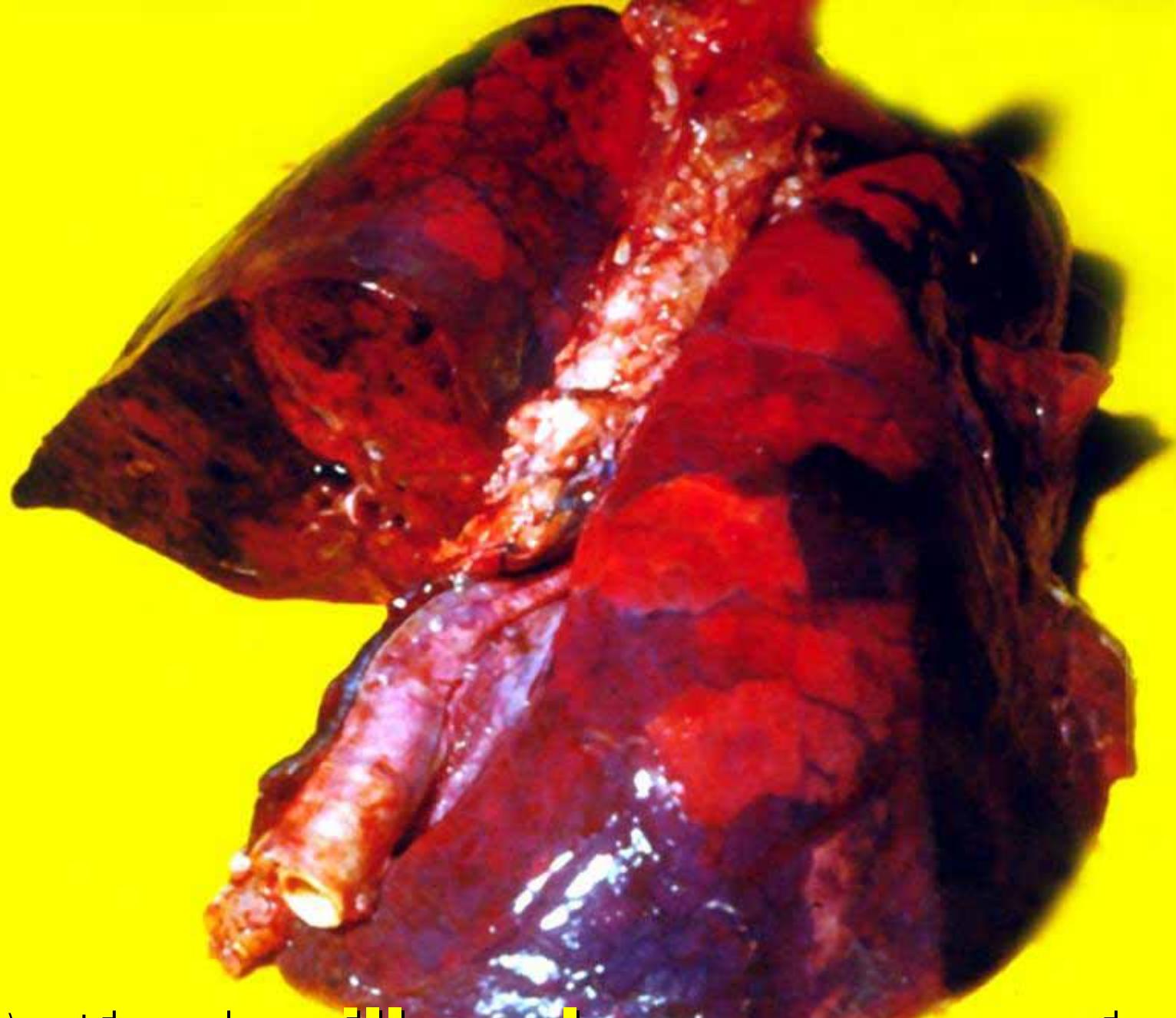
Actinobacillus pleuropneumoniae



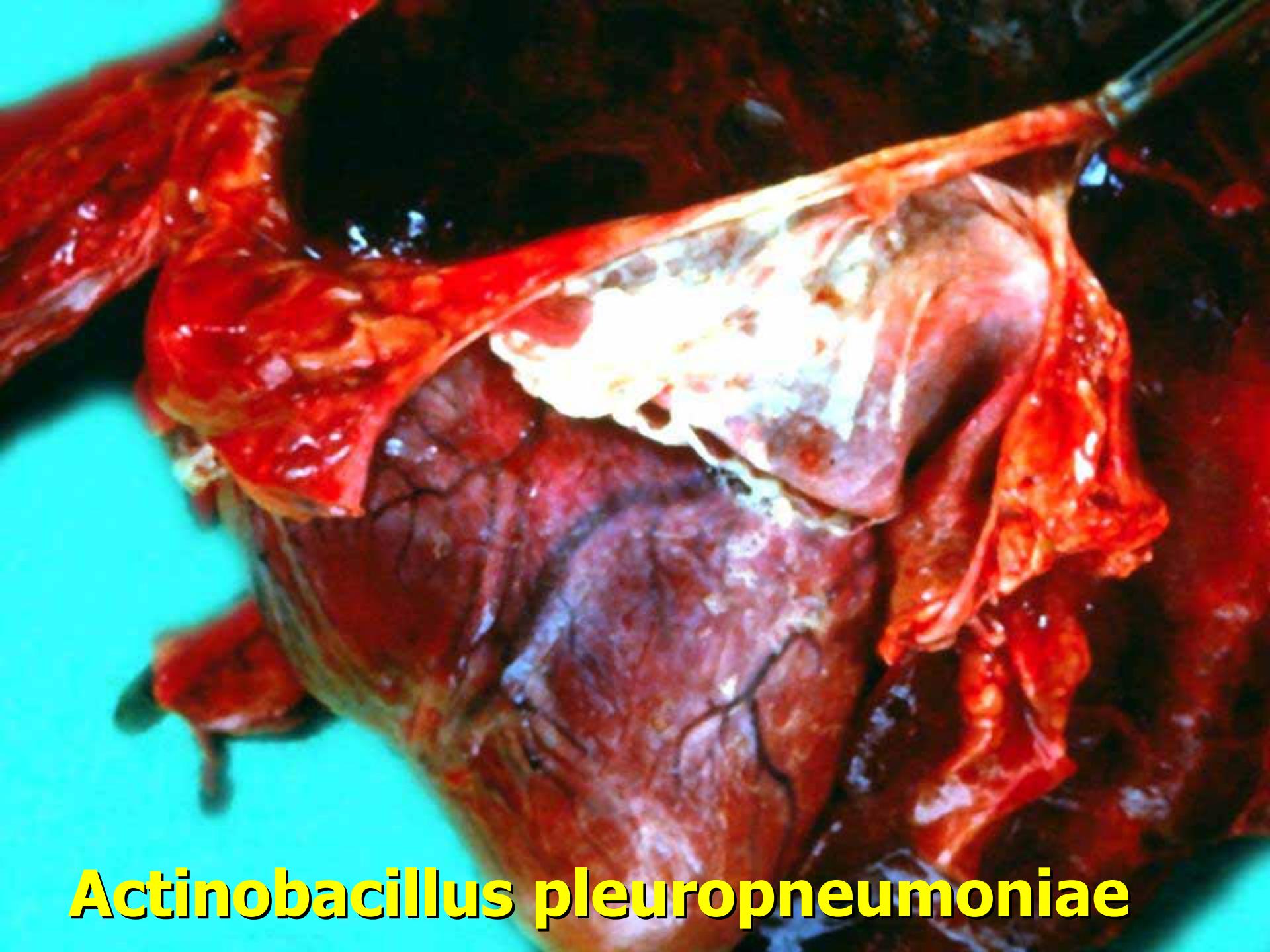
Actinobacillus pleuropneumoniae



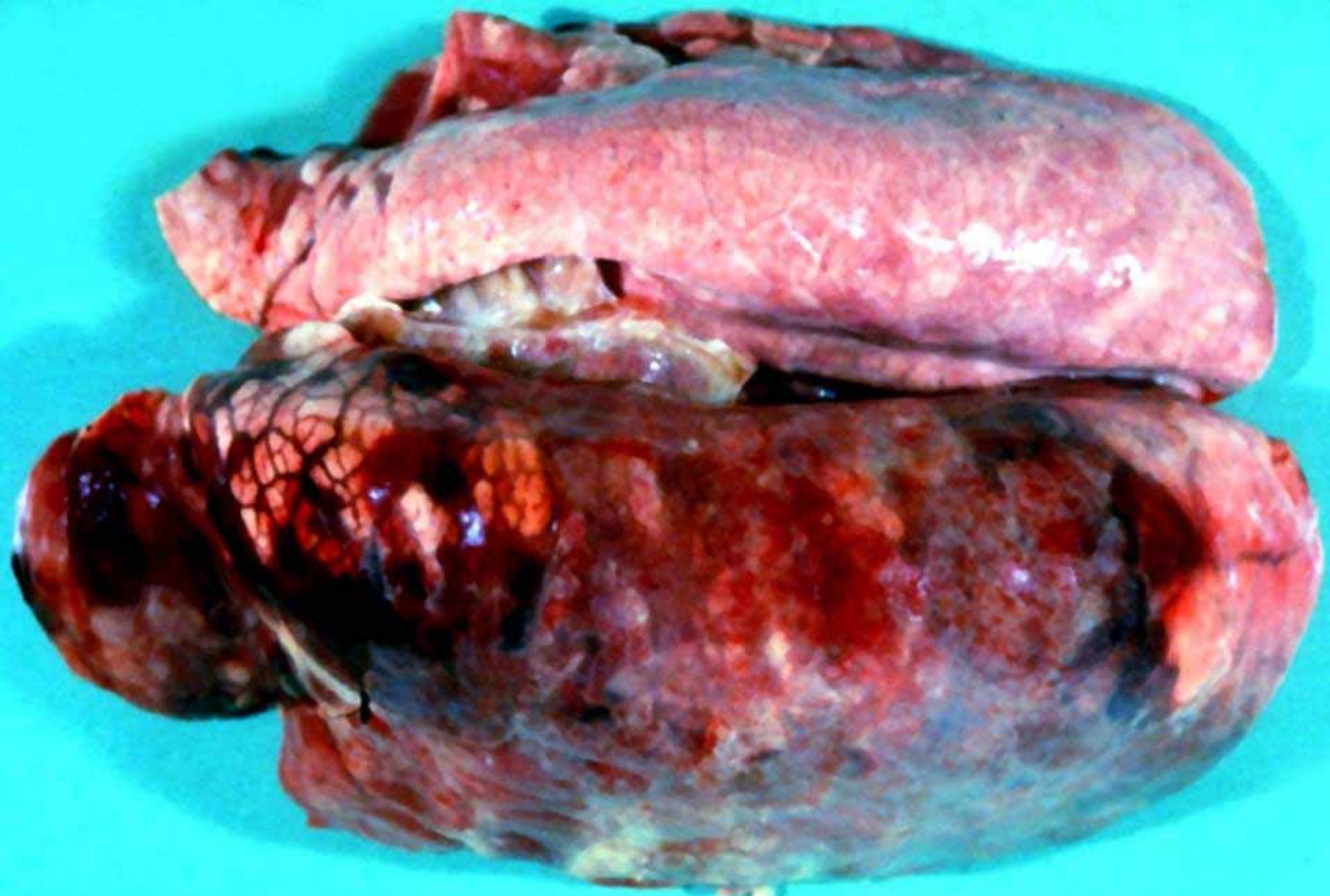
Actinobacillus pleuropneumoniae



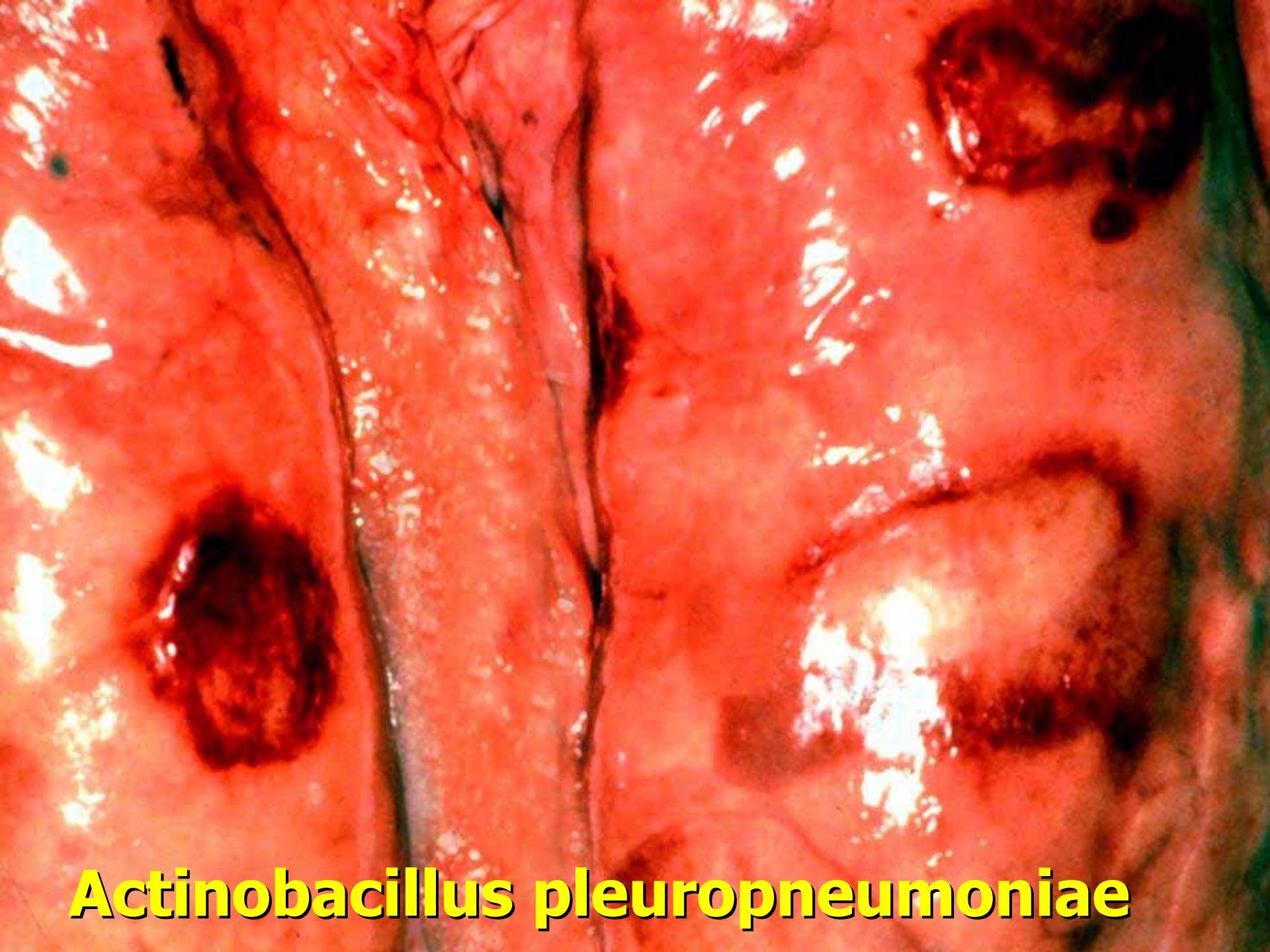
Actinobacillus pleuropneumoniae



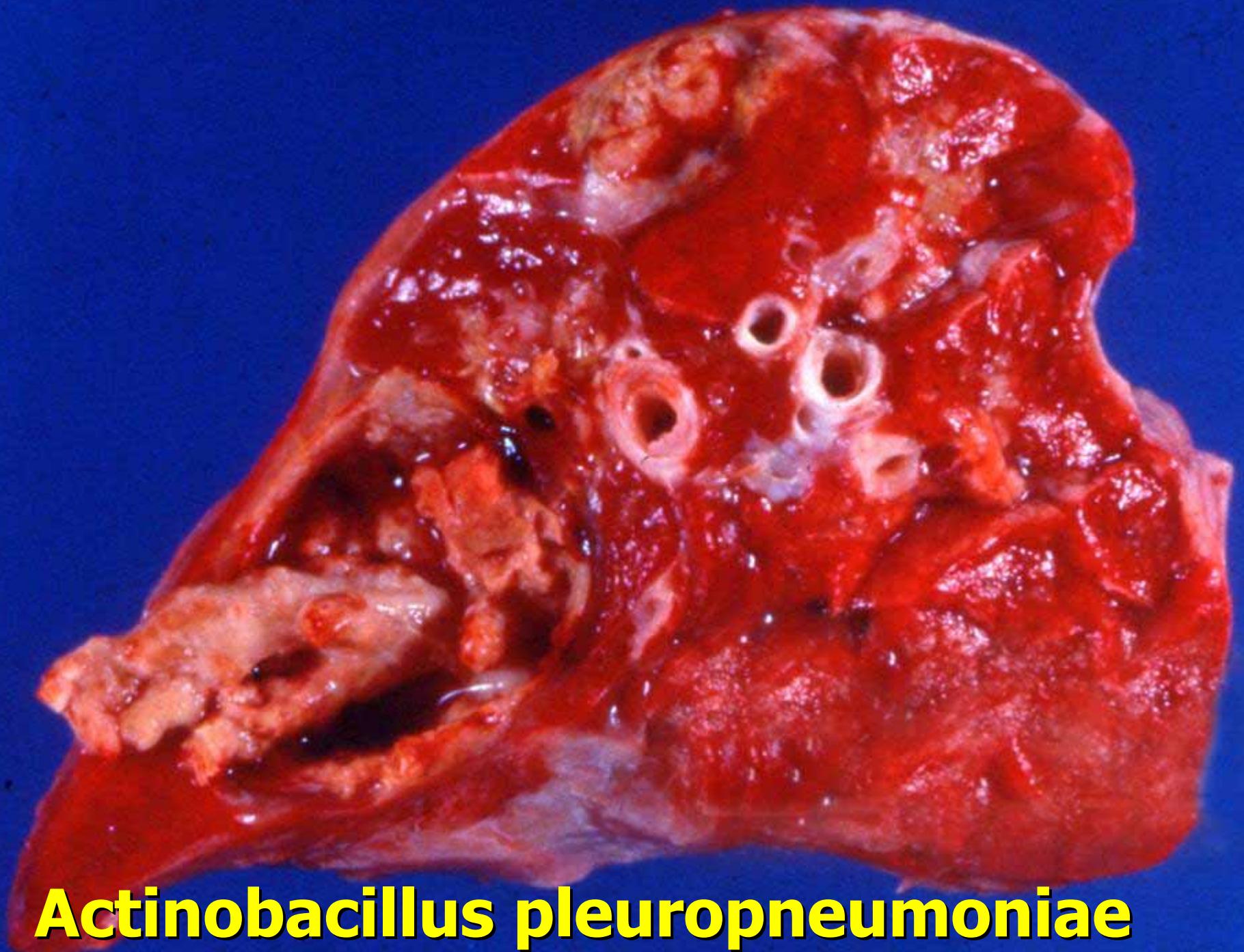
Actinobacillus pleuropneumoniae



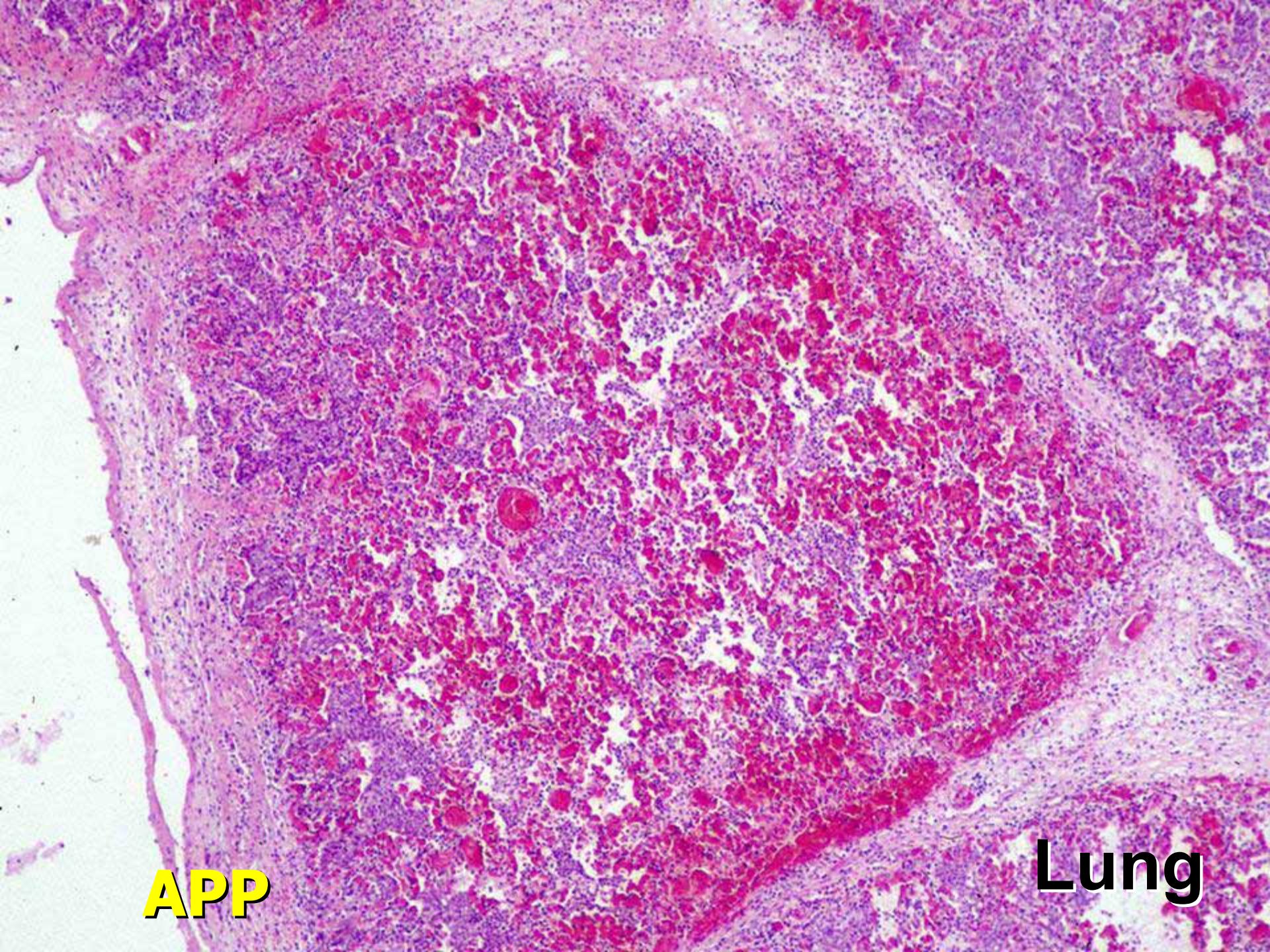
Actinobacillus pleuropneumoniae



Actinobacillus pleuropneumoniae

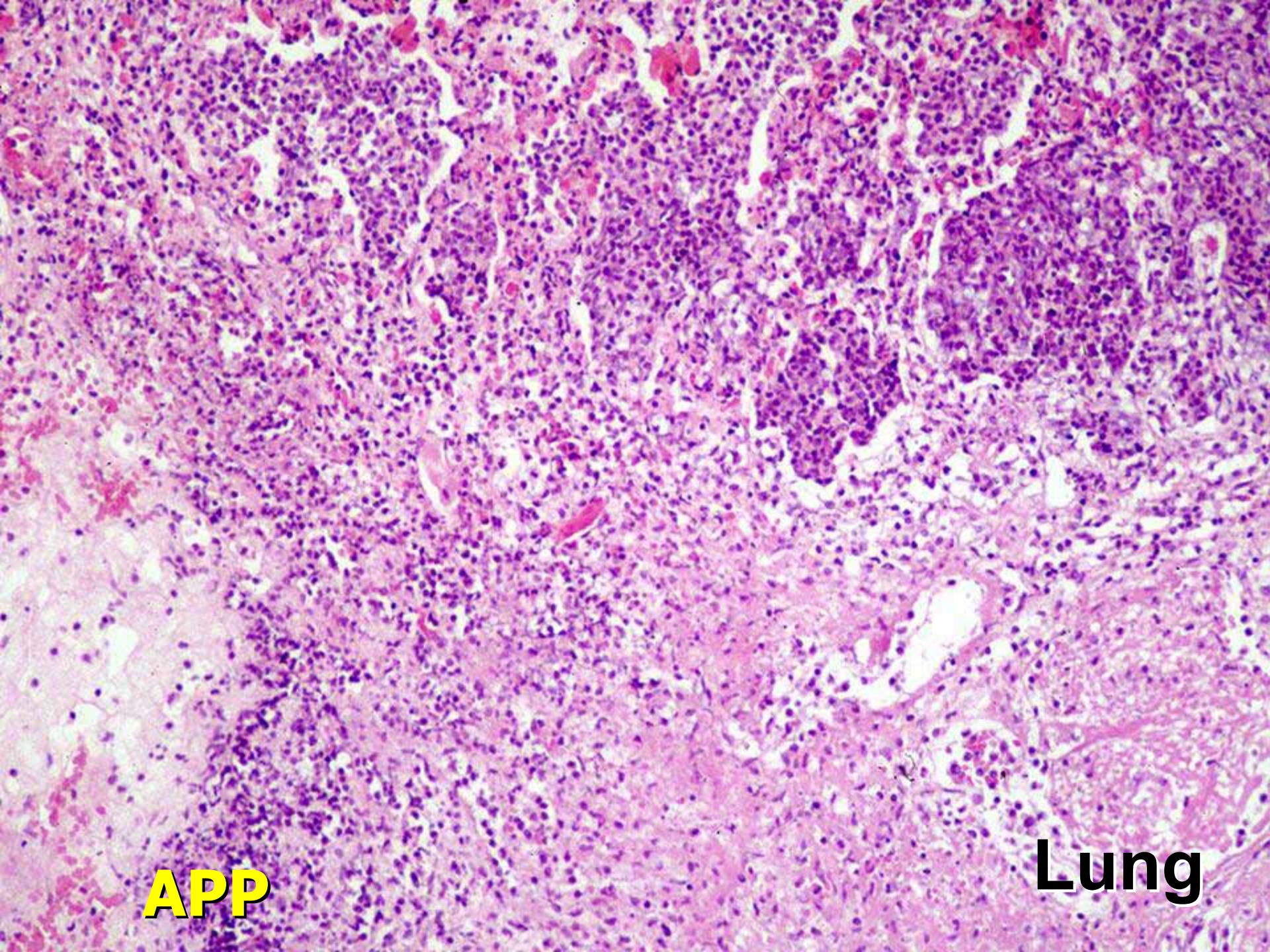


Actinobacillus pleuropneumoniae



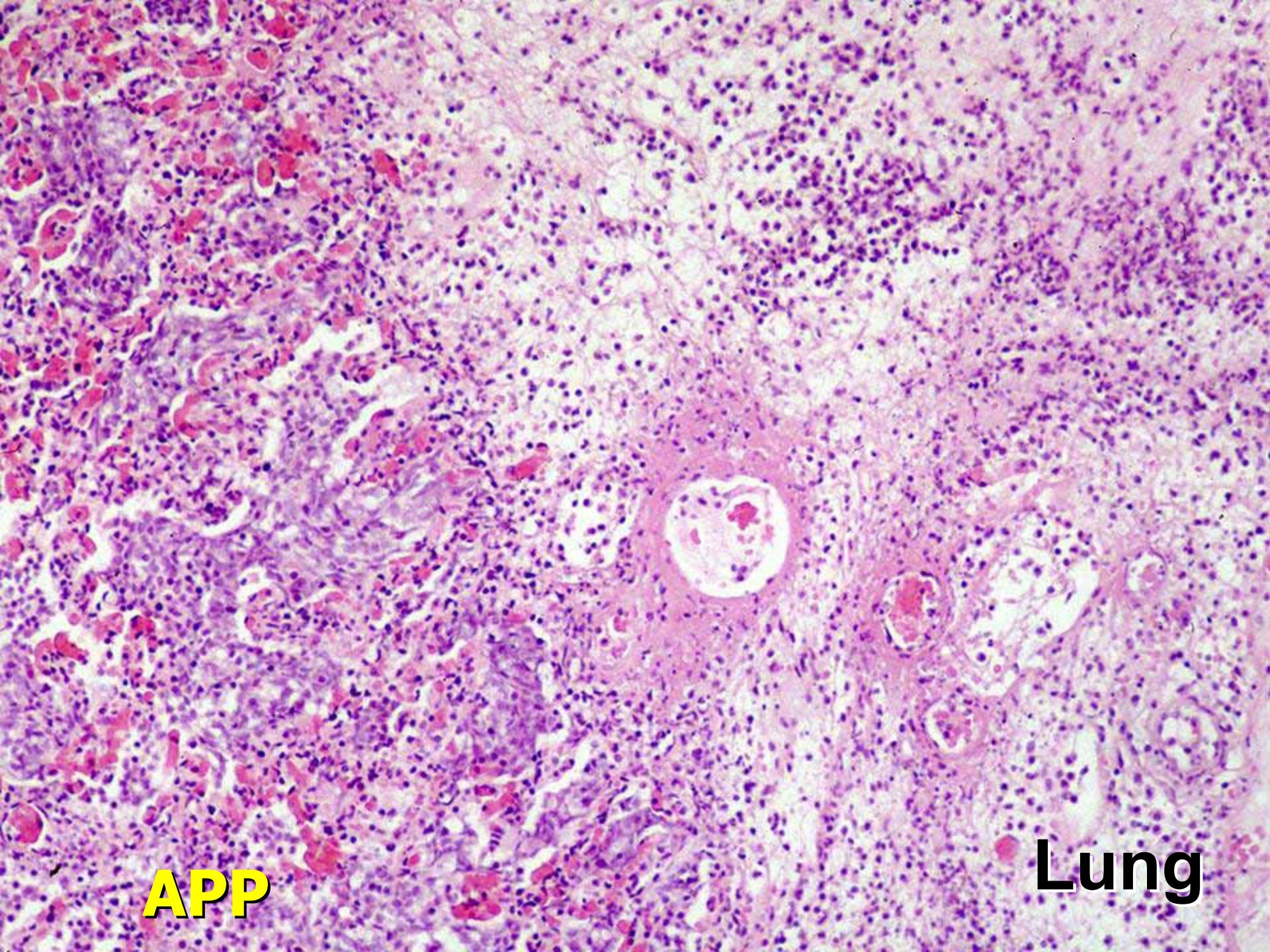
APP

Lung



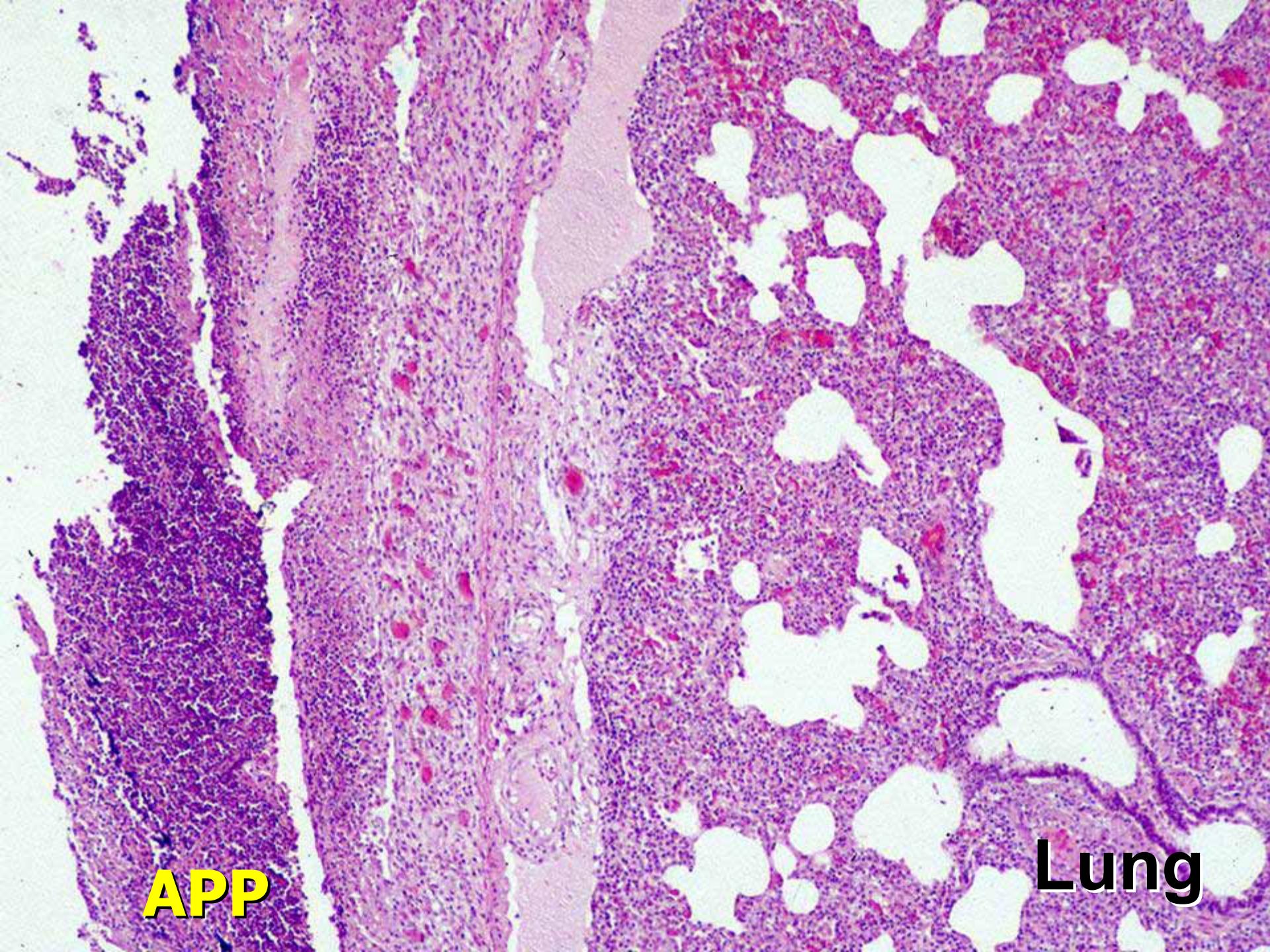
APP

Lung



APP

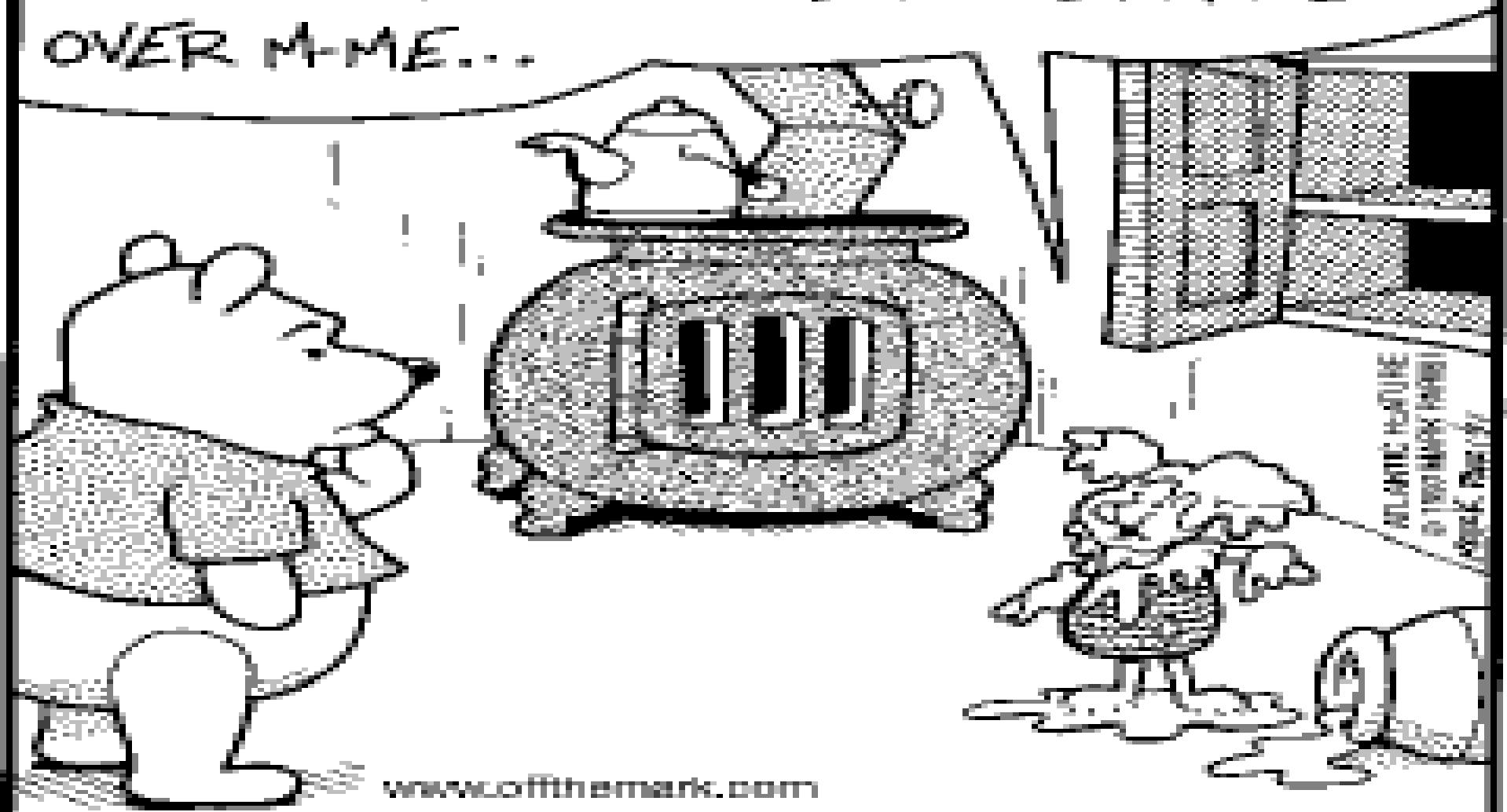
Lung



APP

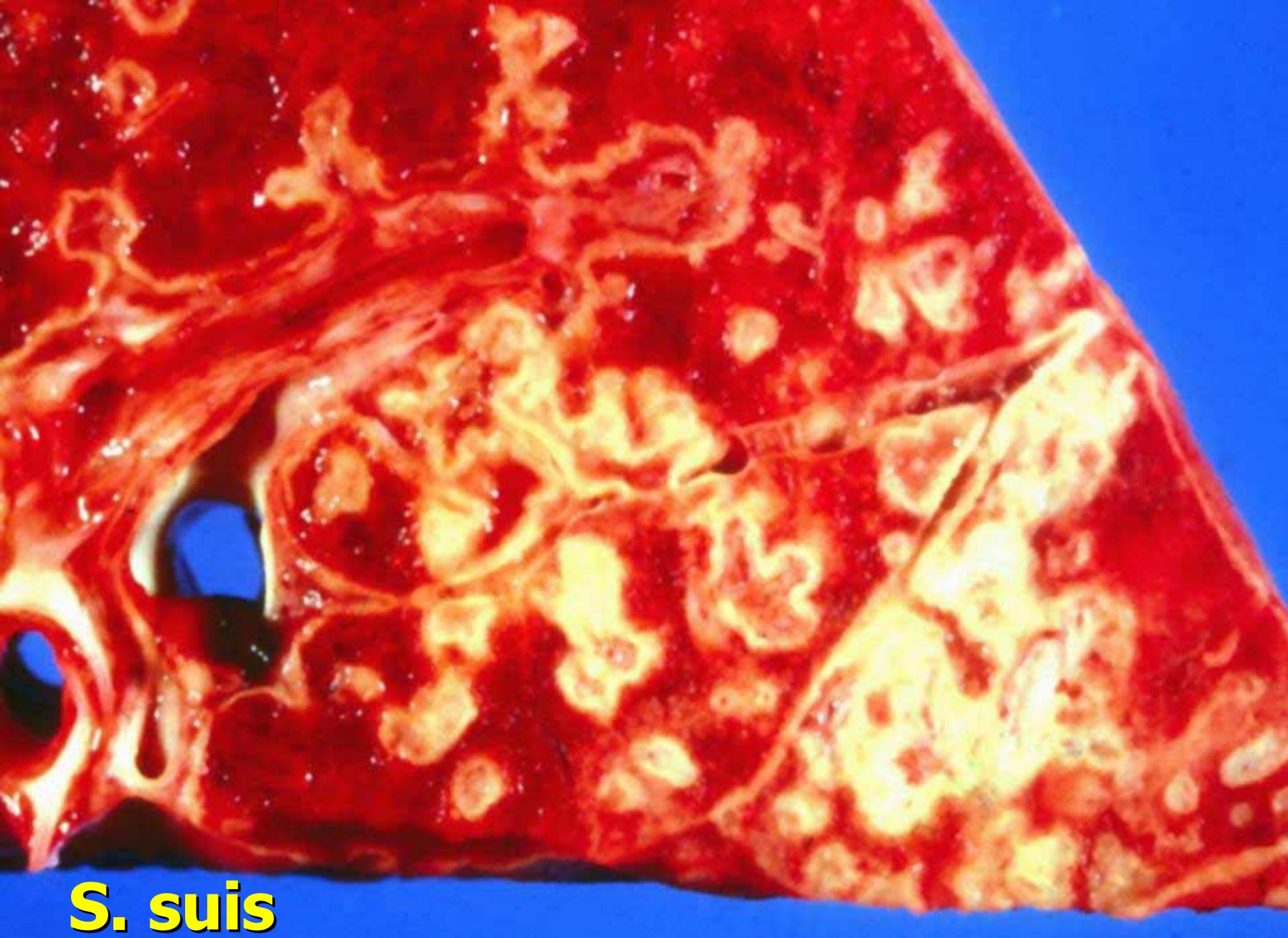
Lung

OH, O-O-DEAR DEAR... I SEEM TO
HAVE SPILLED YOUR D-DINNER ALL
OVER ME...



www.coffthemark.com

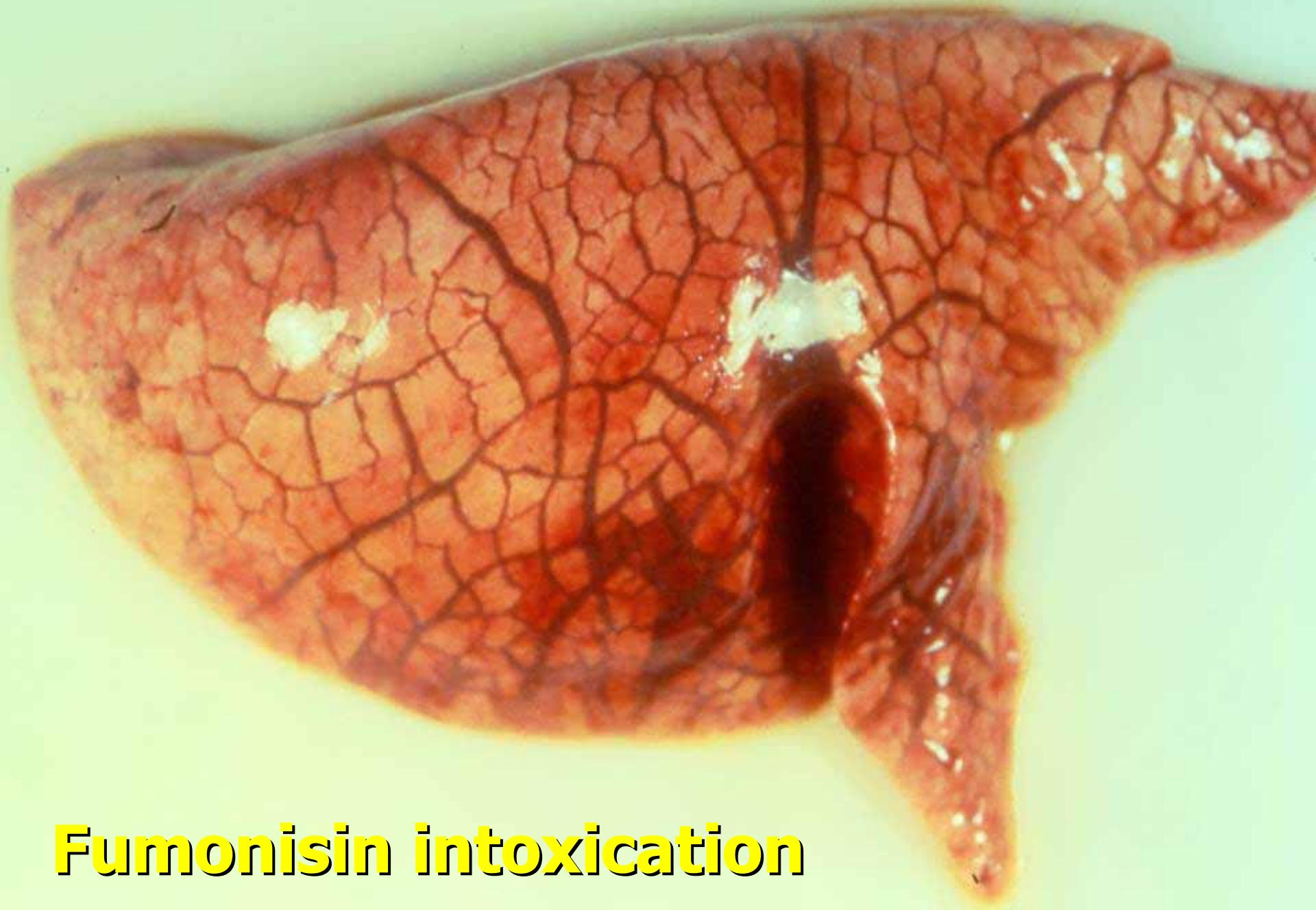
WINNIE THE POOH, ABOUT TO INVENT
THE FIRST HONEY-ROASTED HAM



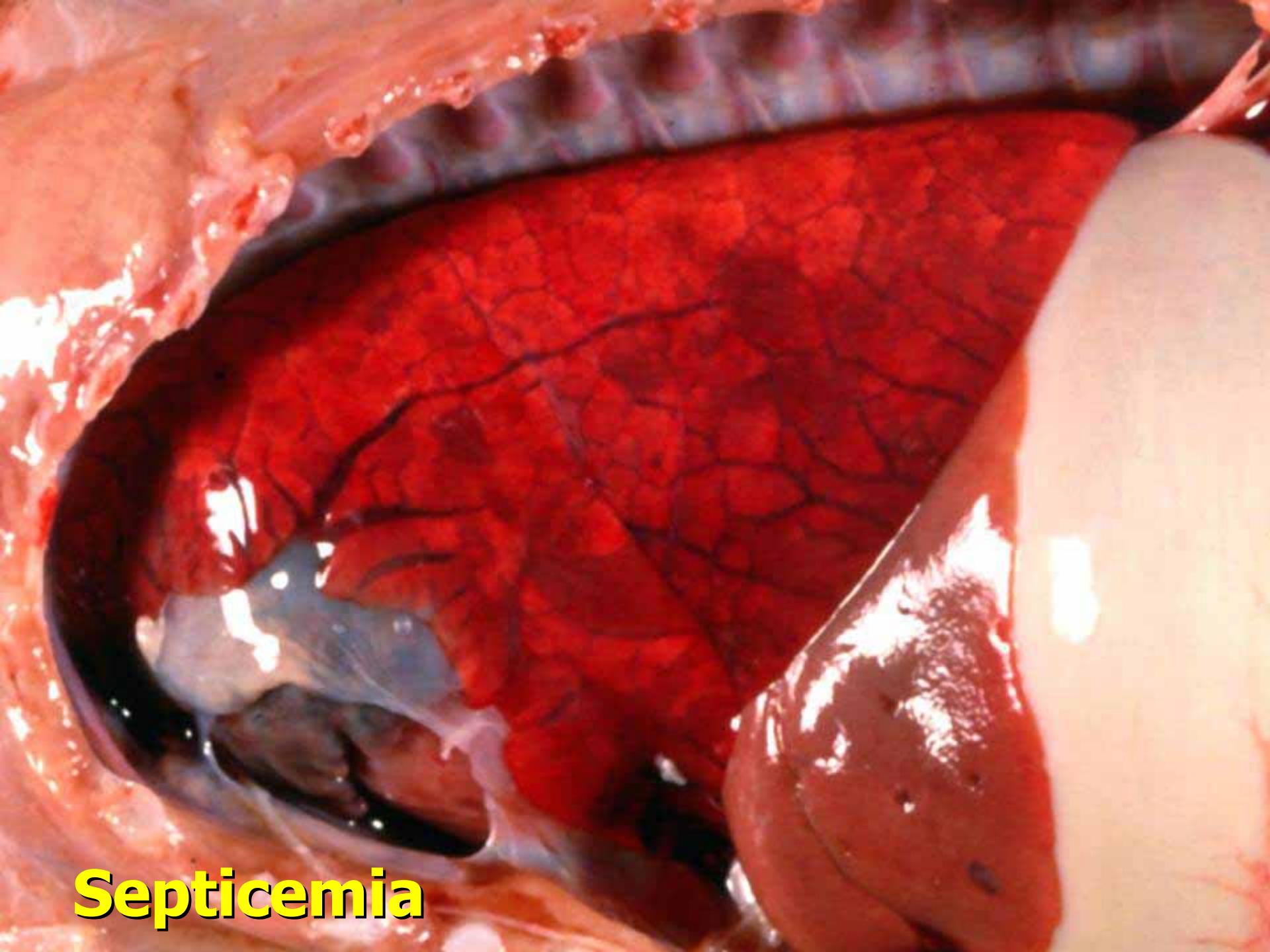
S. suis



Fumonisin intoxication



Fumonisin intoxication



Septicemia



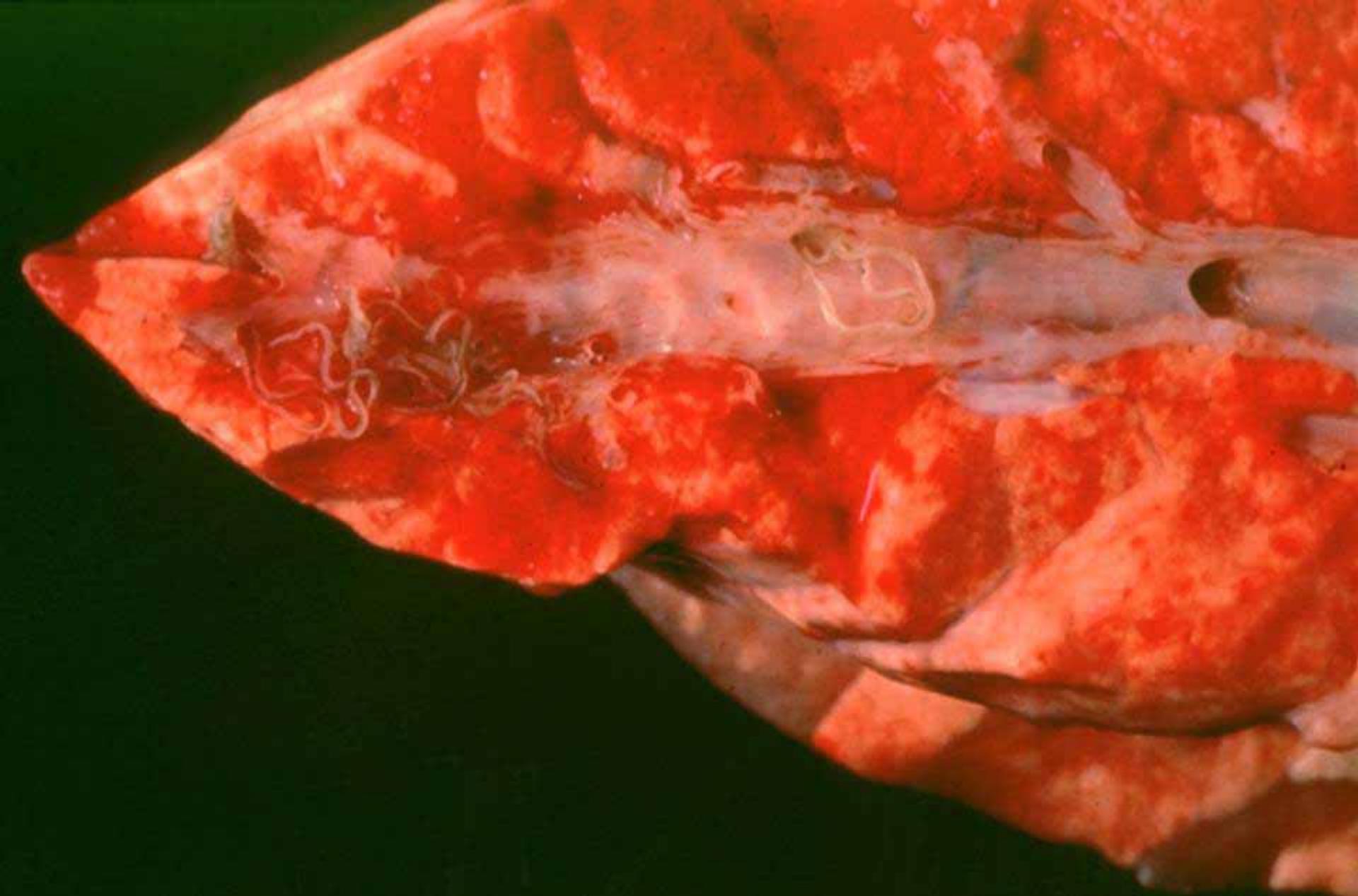
Septicemia

COMMON CAUSES AND GROSS TYPES OF PNEUMONIA IN SWINE

Cause	Type of Pneumonia	Ages Commonly Affected	Is Coughing Typical?
<i>Aujeszky's virus</i>	Interstitial pneumonia	Any age	Sometimes
Swine Influenza virus	Interstitial pneumonia	Any age	Yes (loud, harsh)
PRRS virus	Interstitial pneumonia	Any age	No
Ascarid larval migration	Interstitial pneumonia	After weaning	No
<i>S. choleraesuis</i> septicemia	Interstitial pneumonia	1 – 5 months	No
<i>Mycoplasma hyopneumoniae</i>	Bronchopneumonia	1 ½ – 6 months	Yes (dry, soft)
<i>Actinobacillus pleuropneumoniae</i>	Pleuropneumonia	Any age	No
<i>Bordetella bronchiseptica</i>	Bronchopneumonia	1-5 weeks	Yes
<i>Pasteurella multocida</i> <i>Streptococcus suis</i> <i>Haemophilus parasuis</i> <i>Actinomyces pyogenes</i>	Secondary purulent bronchopneumonia	Any age	Yes



Metastrengylus elongatus

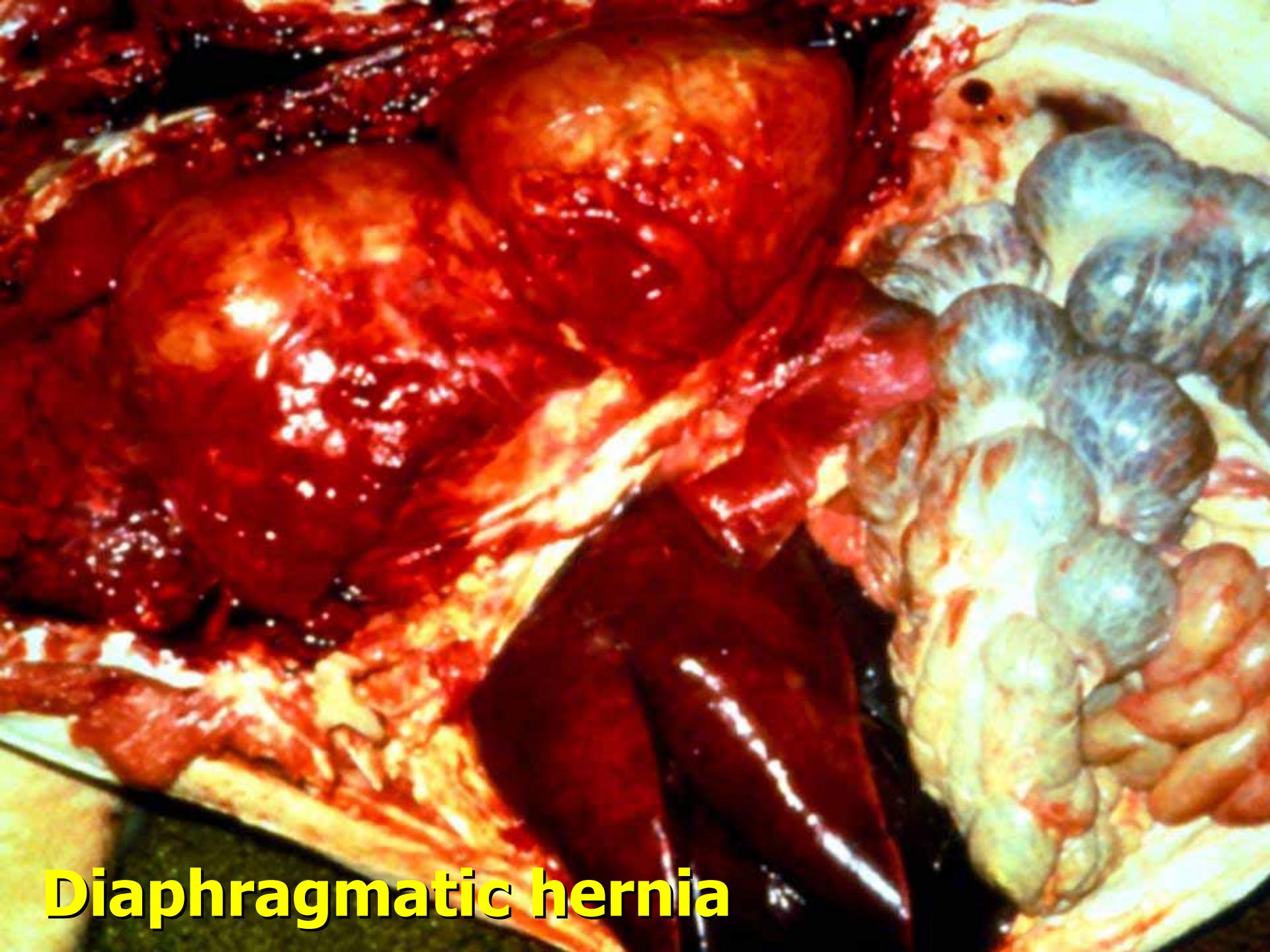


Metastrengylus elongatus

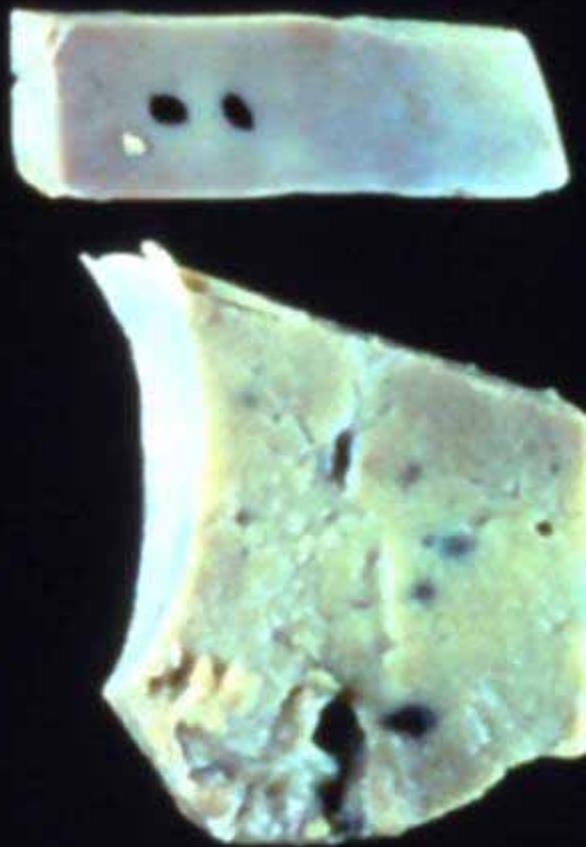


Metastrongylus elongatus

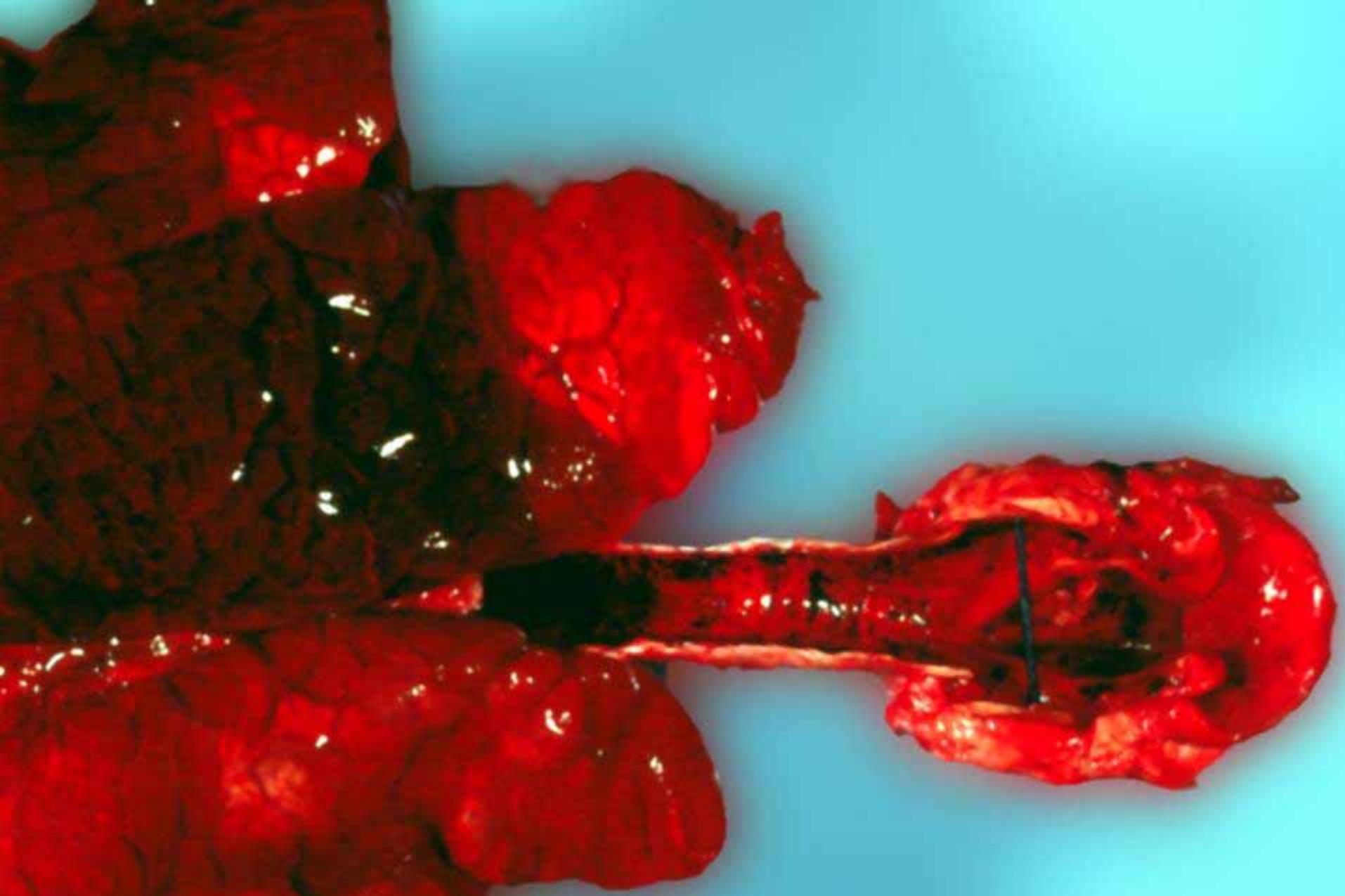
Lung



Diaphragmatic hernia



Melanosis

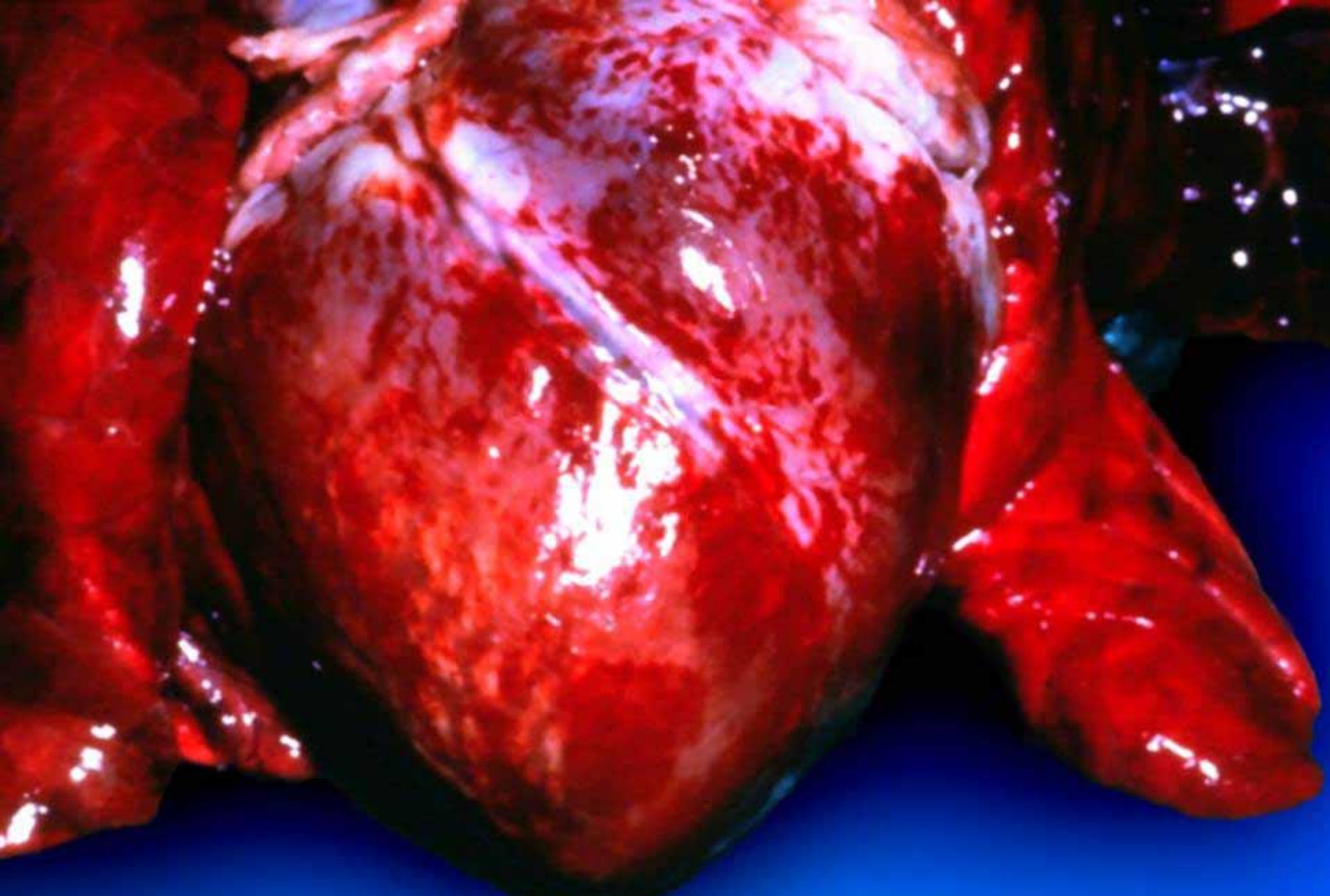


Carboxyhemoglobinemia

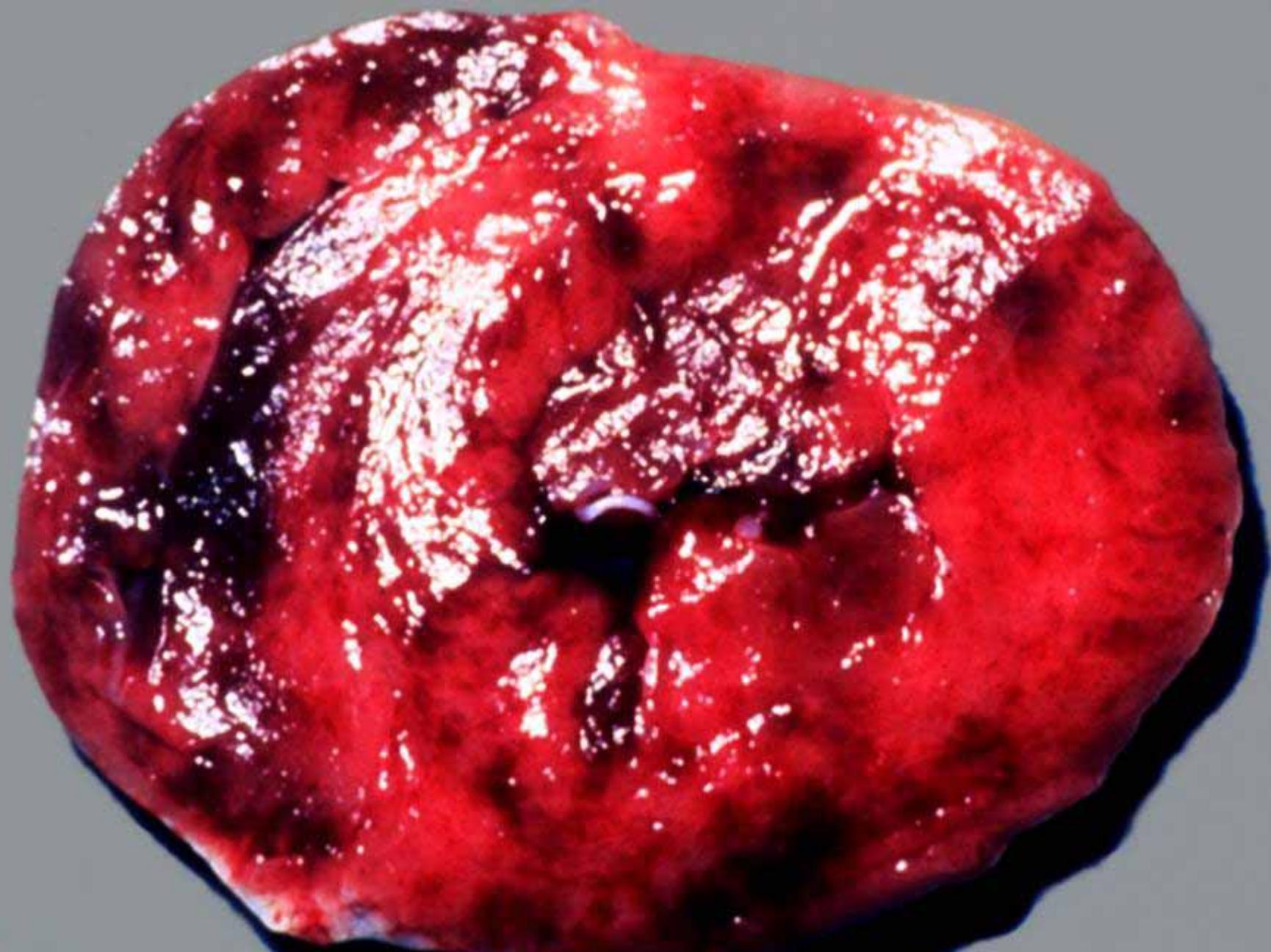




Cardiovascular System



Mulberry heart disease



Mulberry heart disease

A high-magnification light micrograph of heart tissue. The image shows a dense arrangement of pink-stained myofibers, likely cardiomyocytes, separated by white, collagenous connective tissue. A prominent, irregularly shaped, pale-staining area, characteristic of a 'mulberry' lesion, is visible in the lower right quadrant, indicating a site of myocardial necrosis or replacement fibrosis.

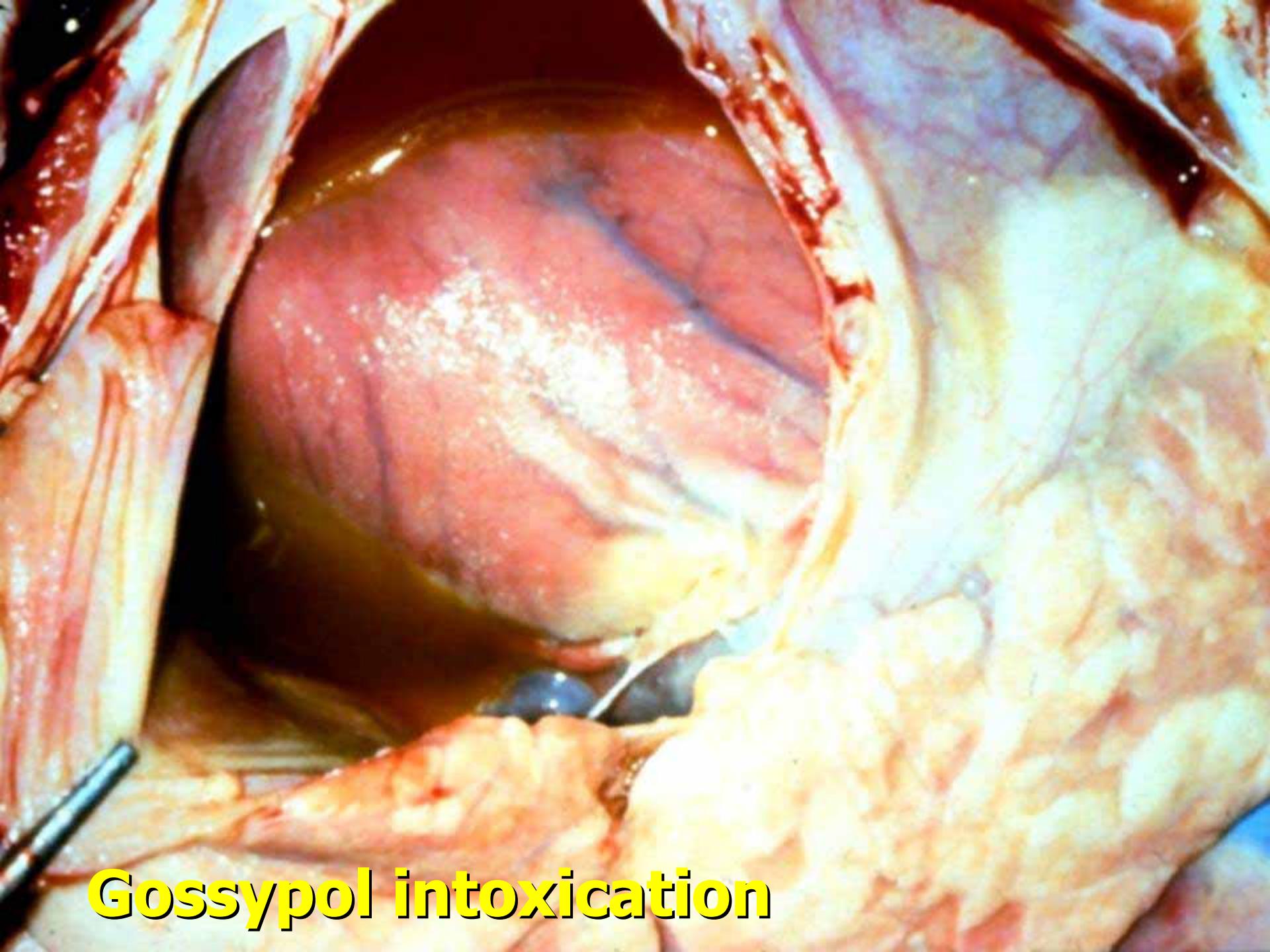
Mulberry heart disease

Heart

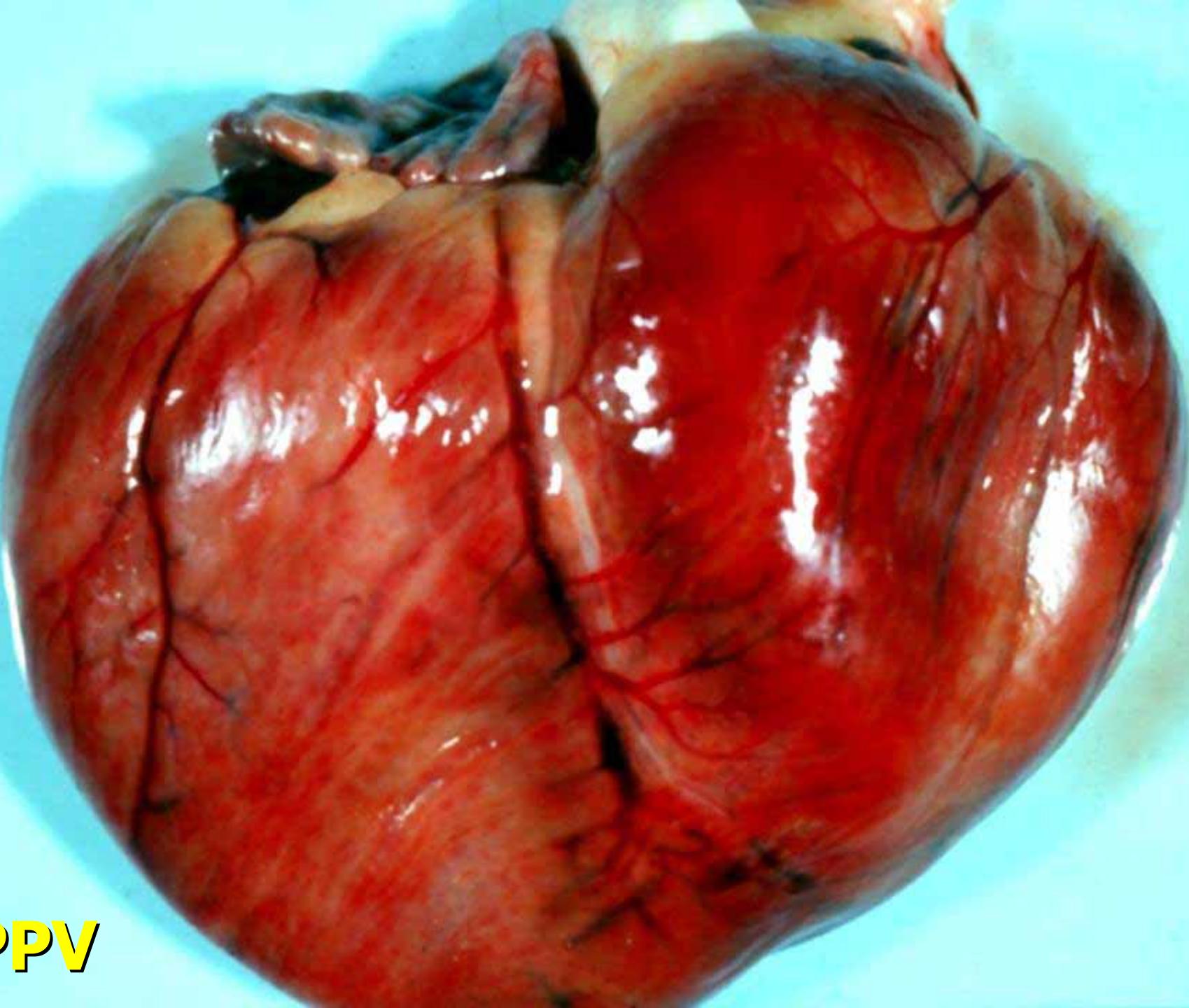


Mulberry heart disease

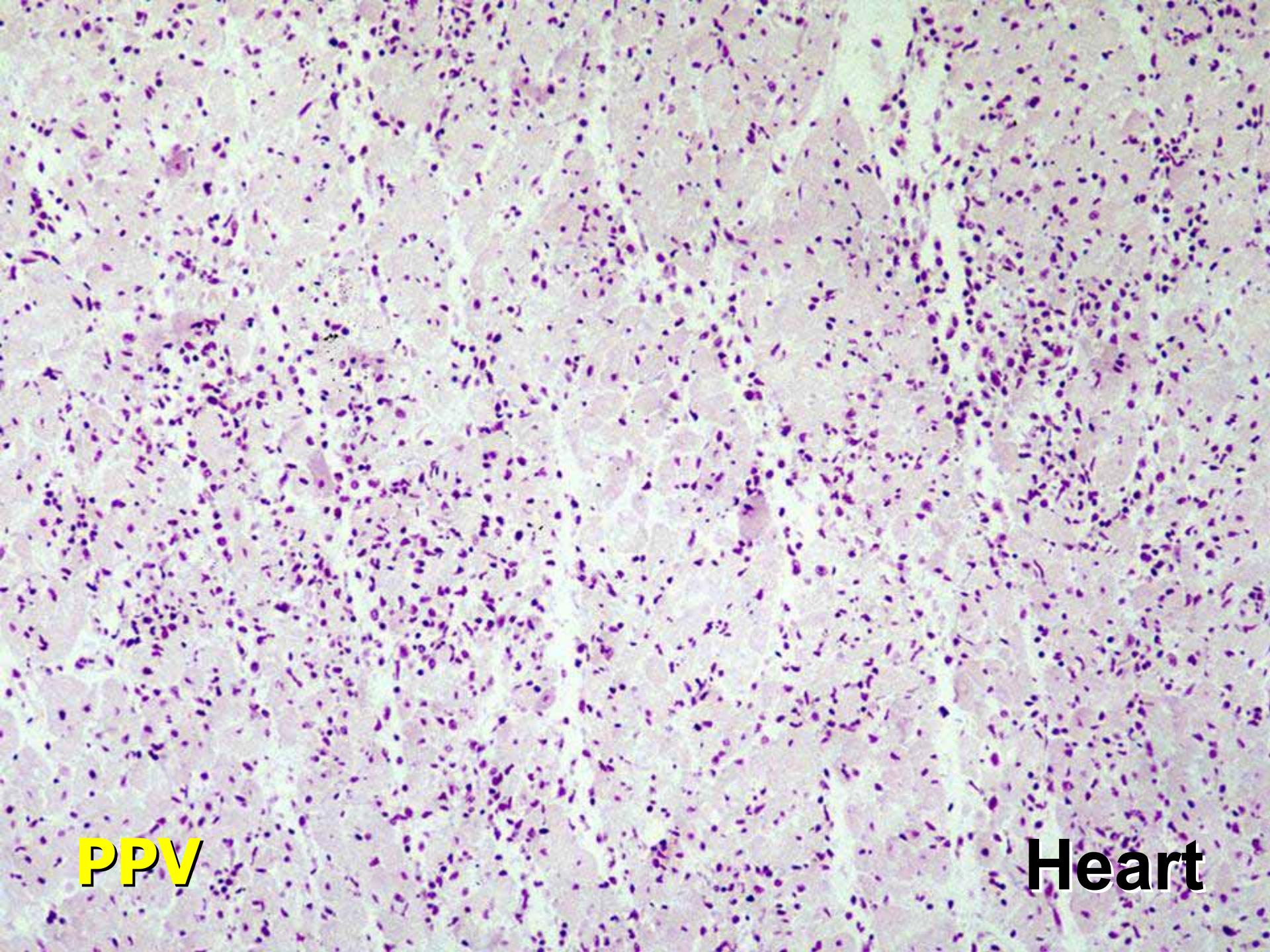
Heart



Gossypol intoxication

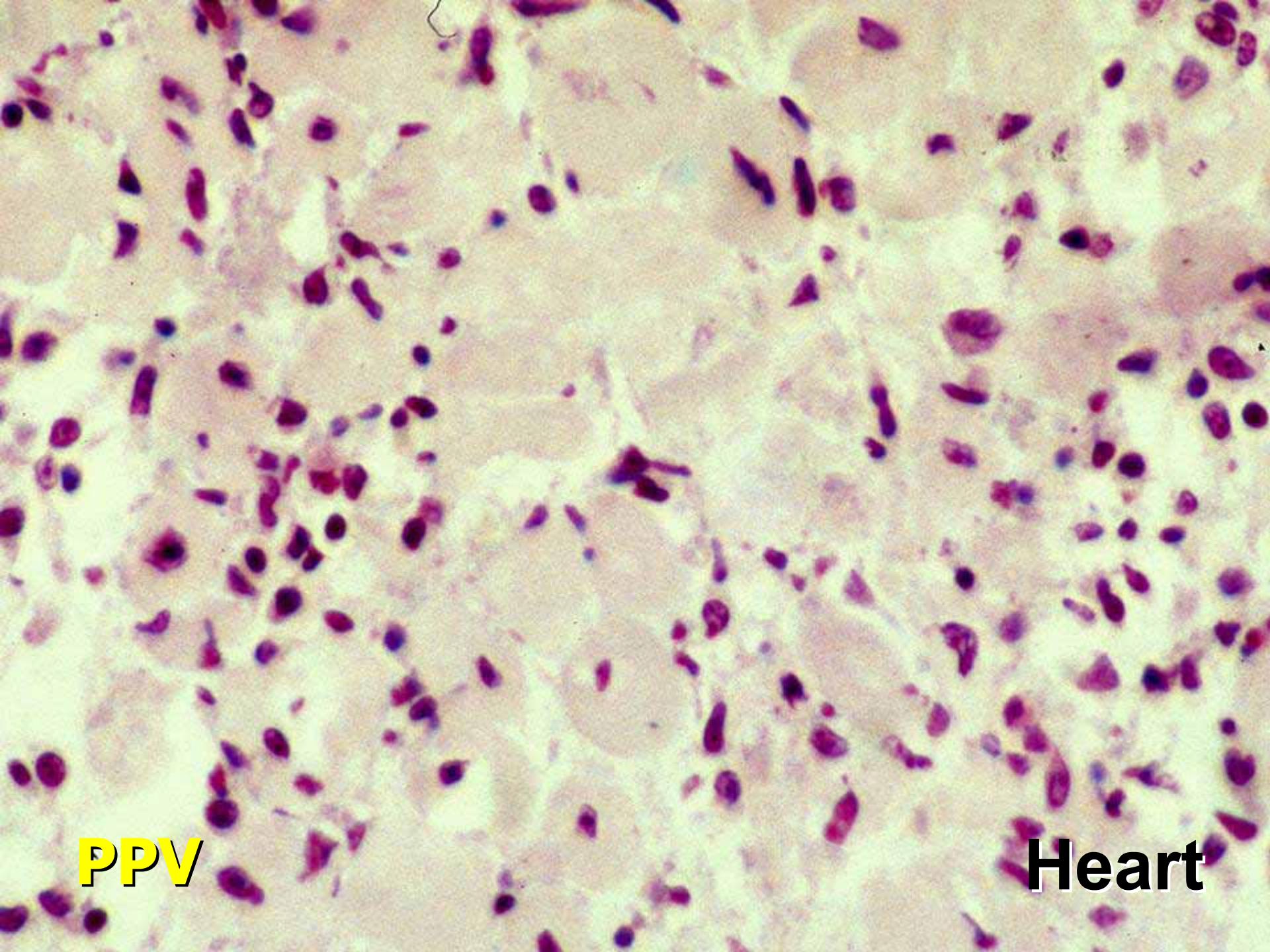


PPV

A light micrograph showing a dense cellular tissue structure, likely heart muscle. The cells are small and rounded, with some darker, more centrally located nuclei. A prominent feature is a large, irregularly shaped area in the center-right where the cellular arrangement is more disorganized and sparse.

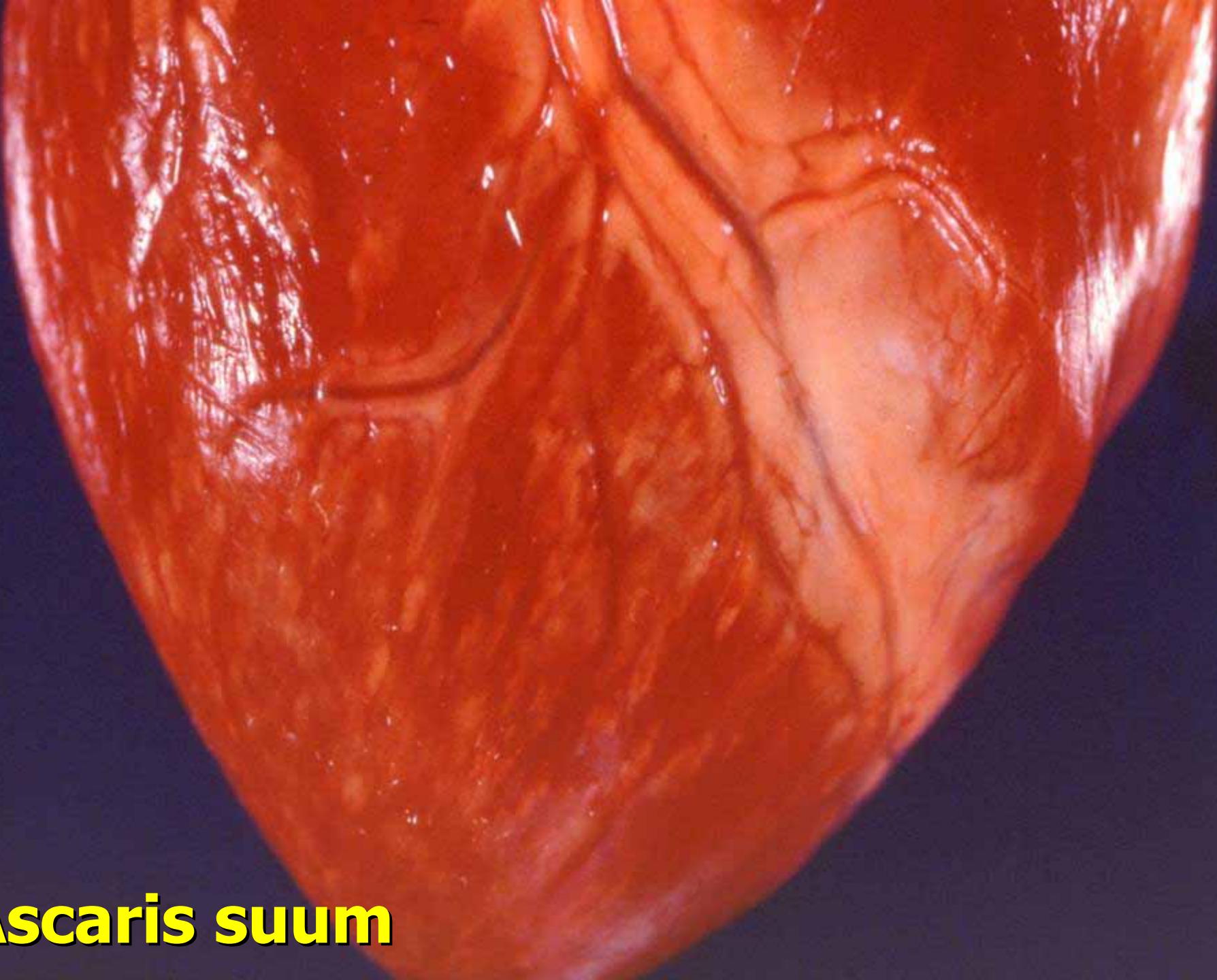
PPV

Heart



PPV

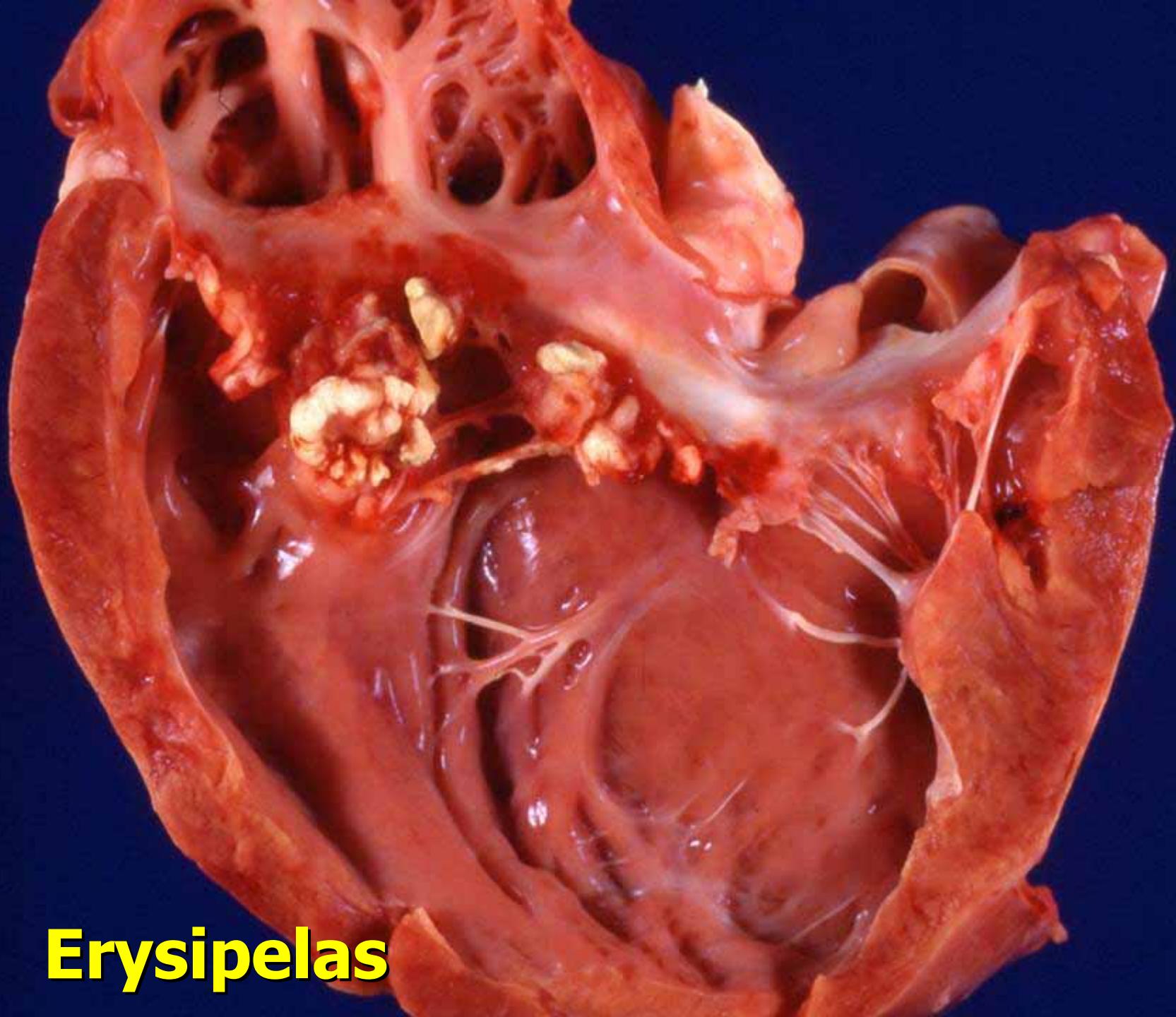
Heart



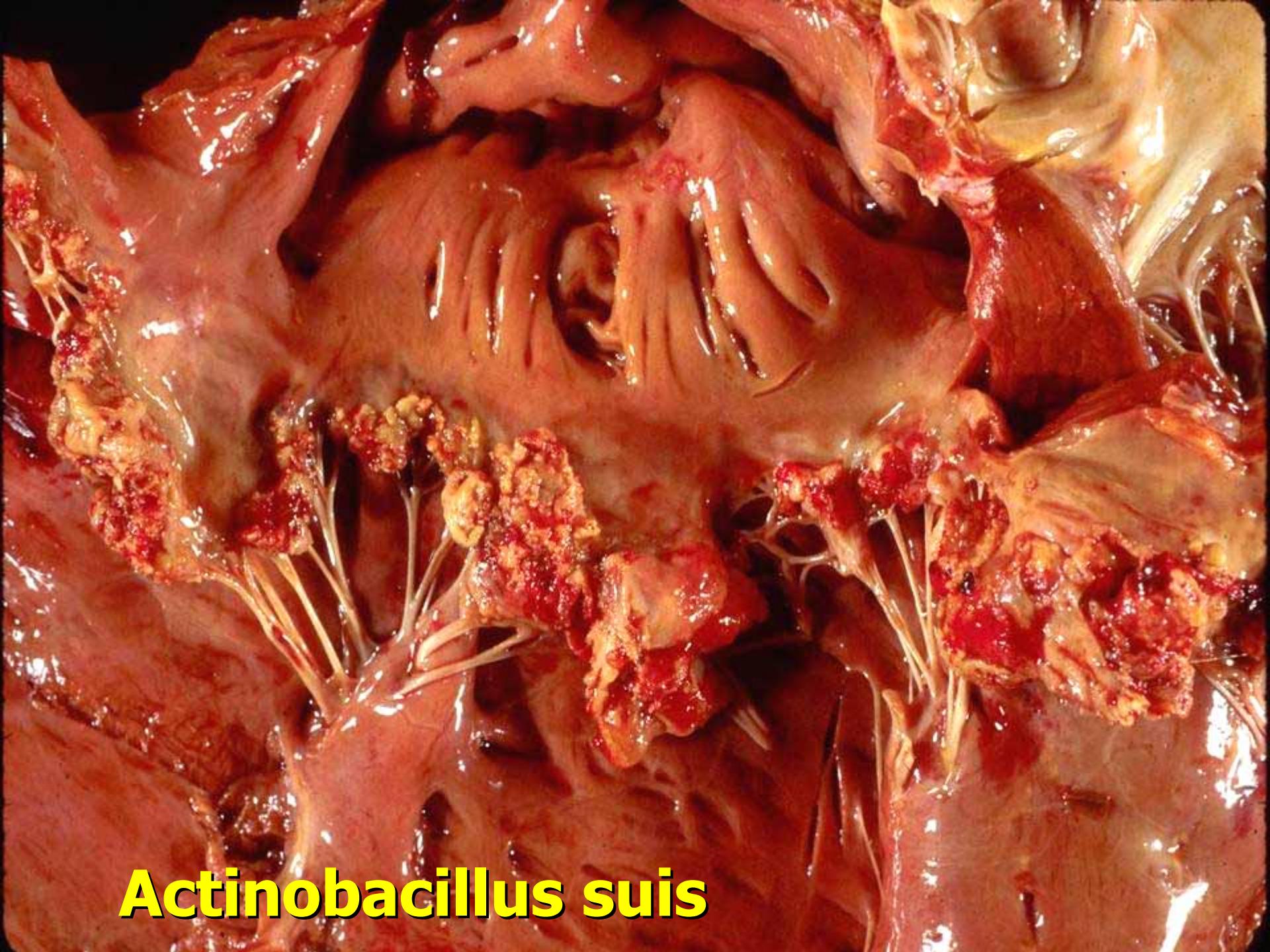
Ascaris suum



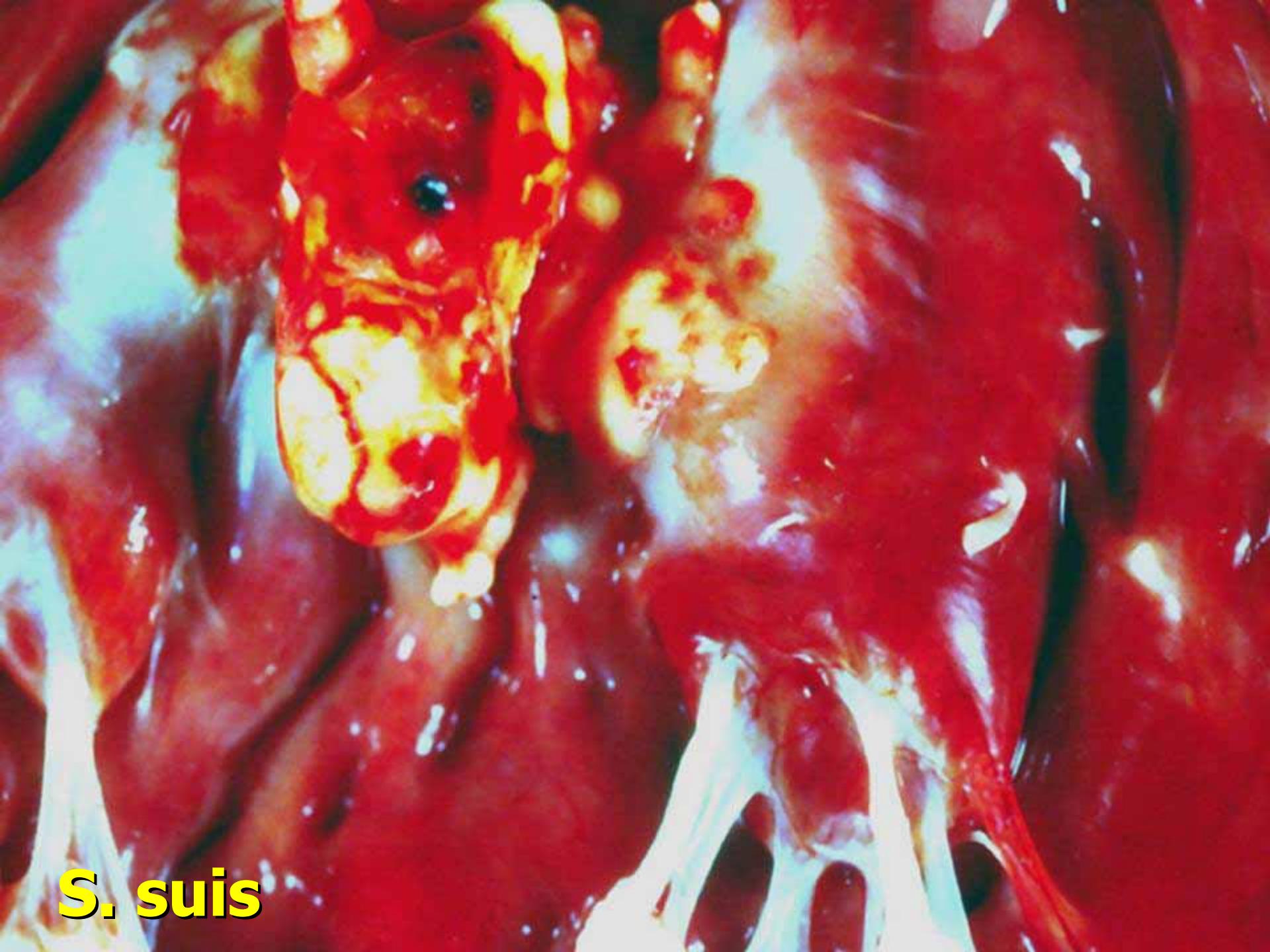
S. suis



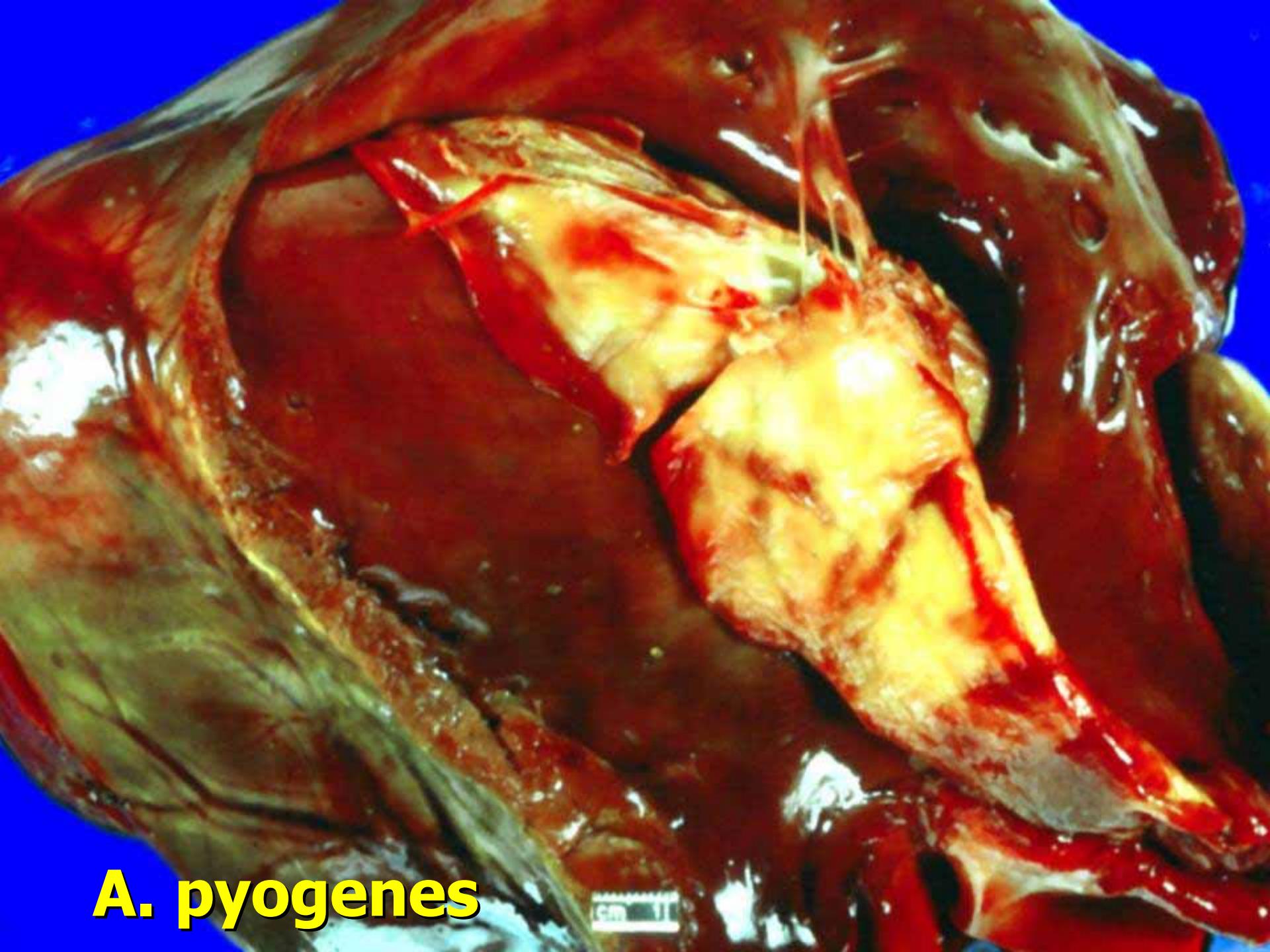
Erysipelas

A close-up, high-magnification photograph of a pig's heart. The heart shows extensive, dark red, necrotic tissue lesions, particularly in the atria and ventricles. These lesions appear as irregular, crumbly masses of tissue. The surrounding myocardium is a lighter, reddish-brown color. The overall texture is somewhat granular and friable.

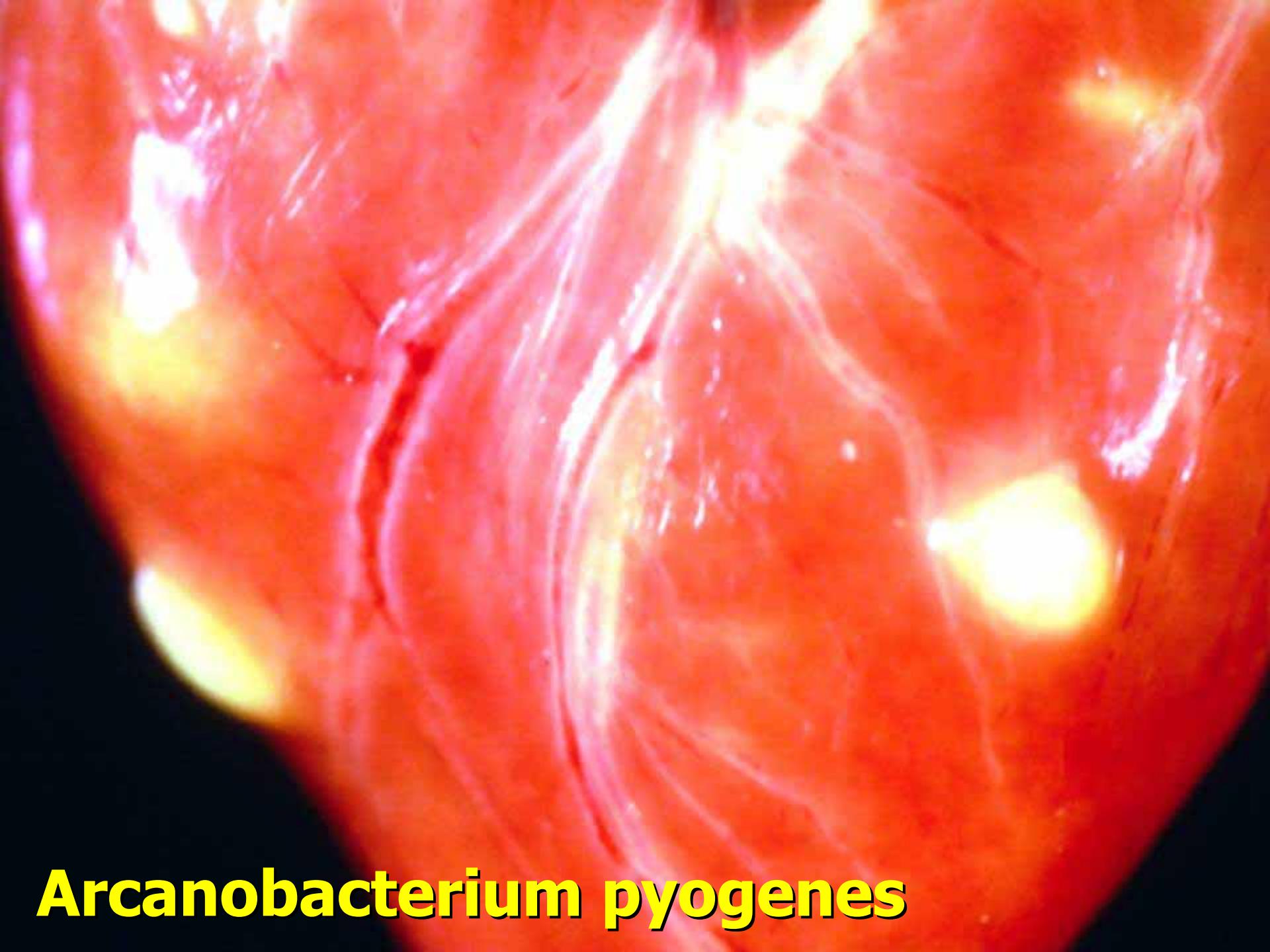
Actinobacillus suis



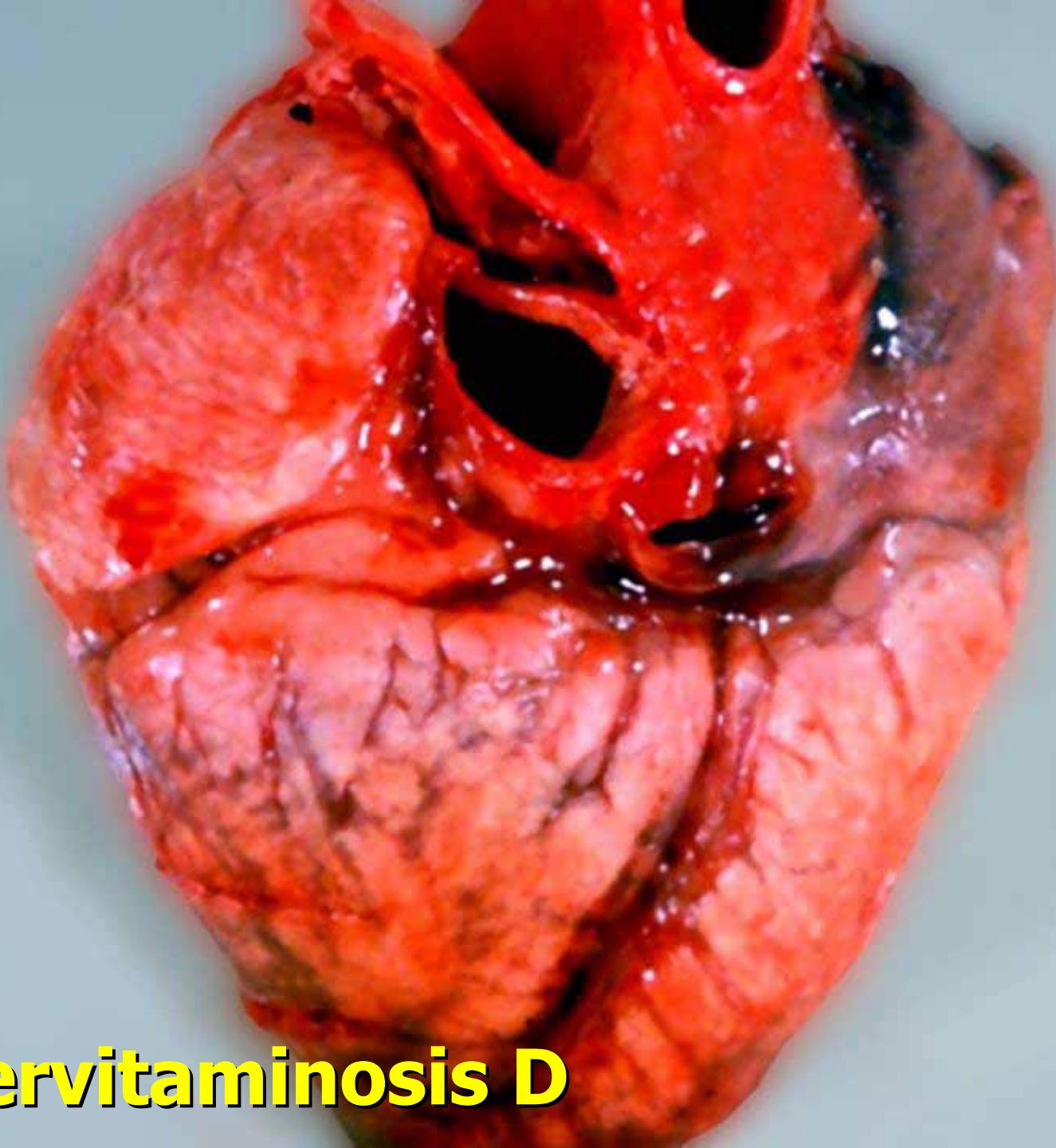
S. suis



A. pyogenes



Arcanobacterium pyogenes



Hypervitaminosis D