

Bronchoalveolar Lavage (BAL)

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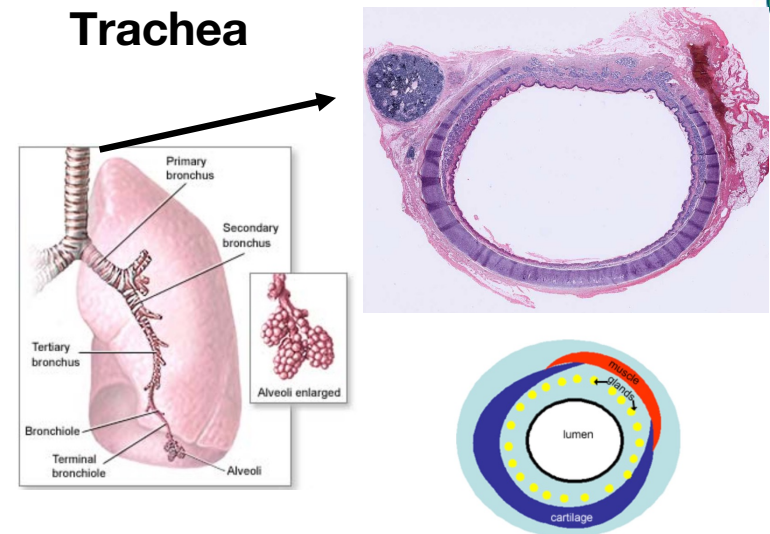
Normal Airway Histology/Ultrastructure

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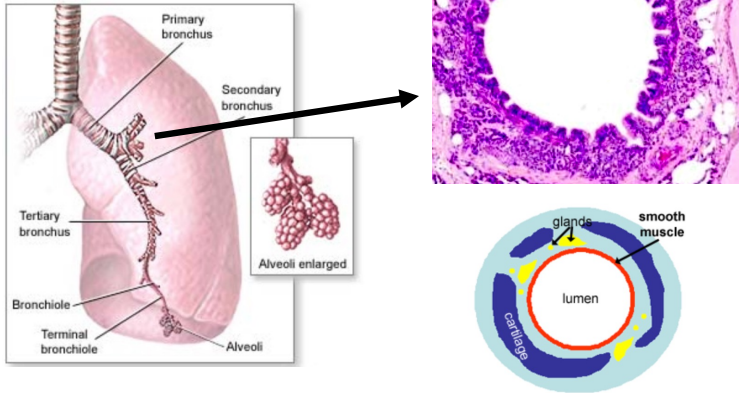
Airway Respiratory-type epithelium



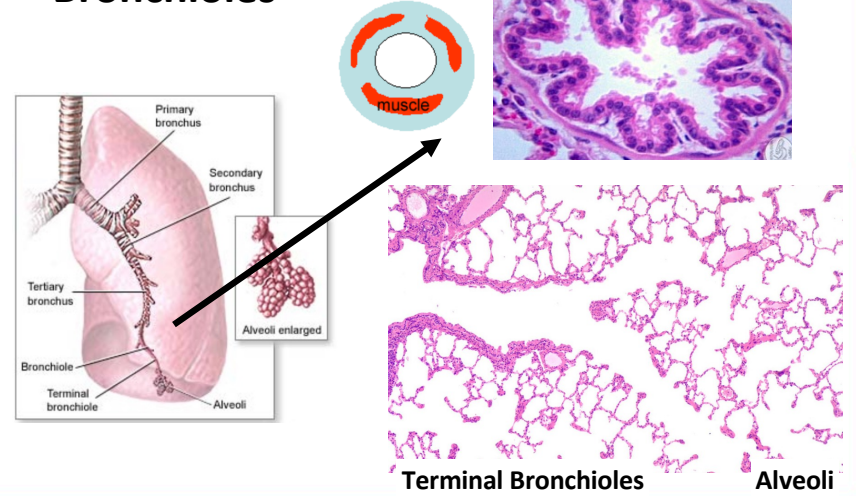
Trachea



Bronchi



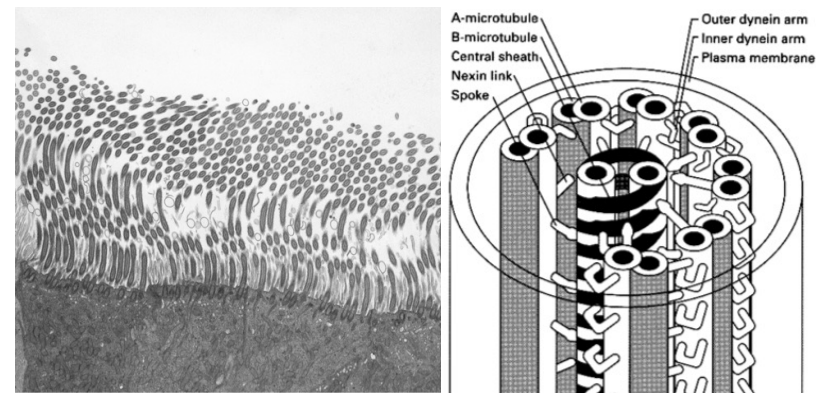
Bronchioles



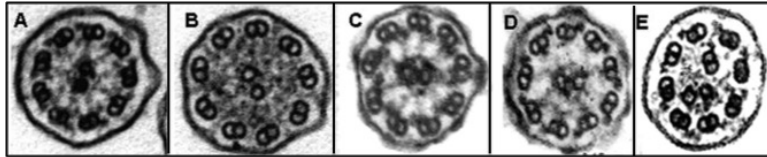
Airway Respiratory-type epithelium



Respiratory mucosa Cilia

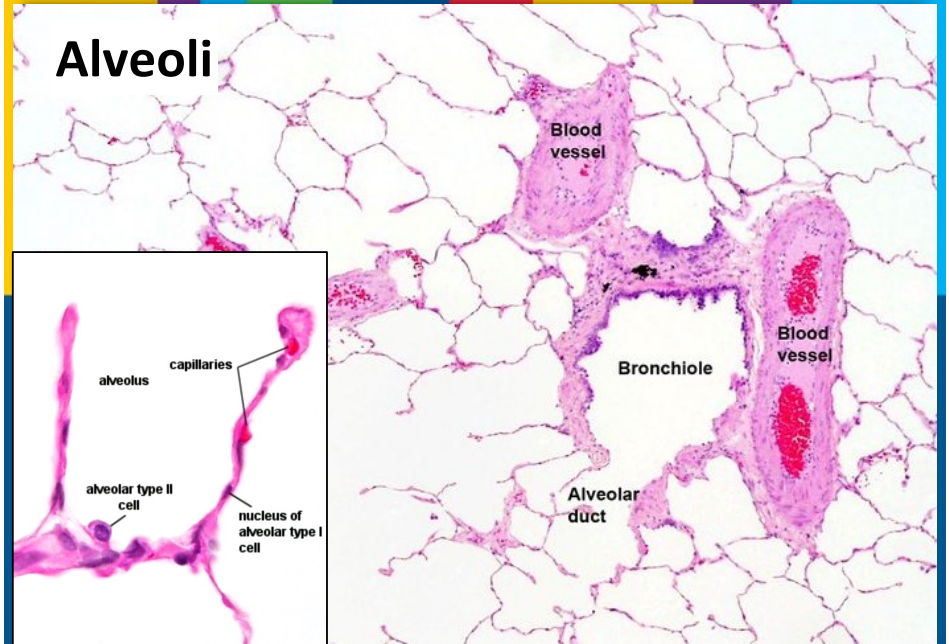


Primary Ciliary Dyskinesia

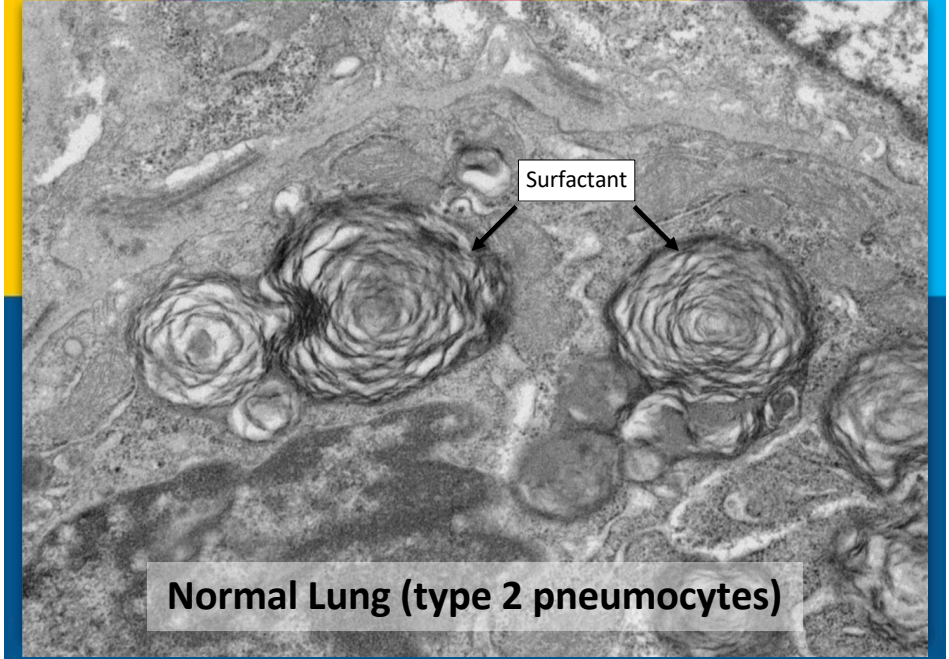
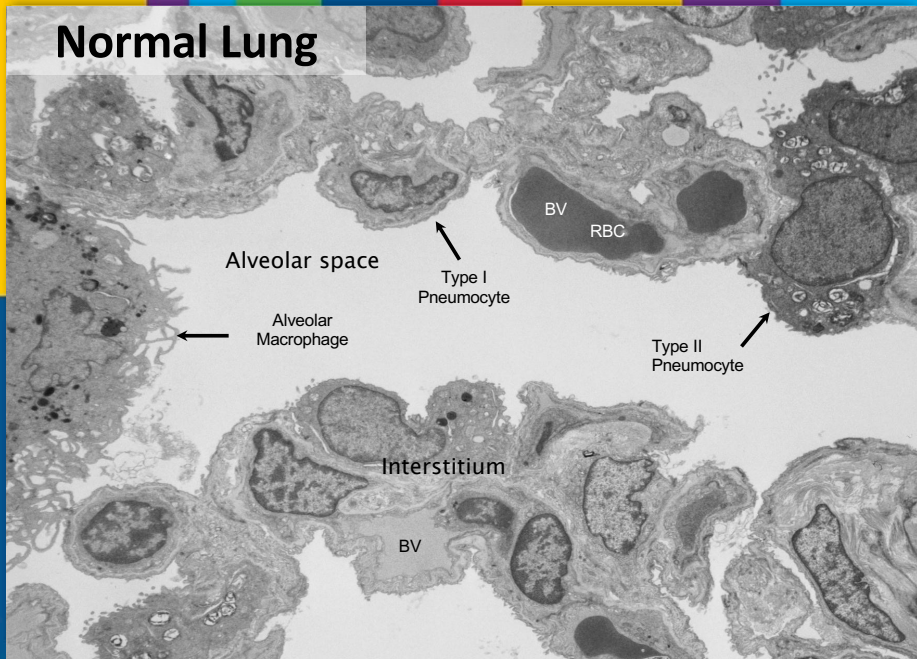


- A. Normal
- B. Outer and inner dynein arm defect
- C. Outer dynein arm defect
- D. Inner dynein arm defect alone
- E. Inner dynein arm defect with microtubule disorganization

Alveoli



Normal Lung

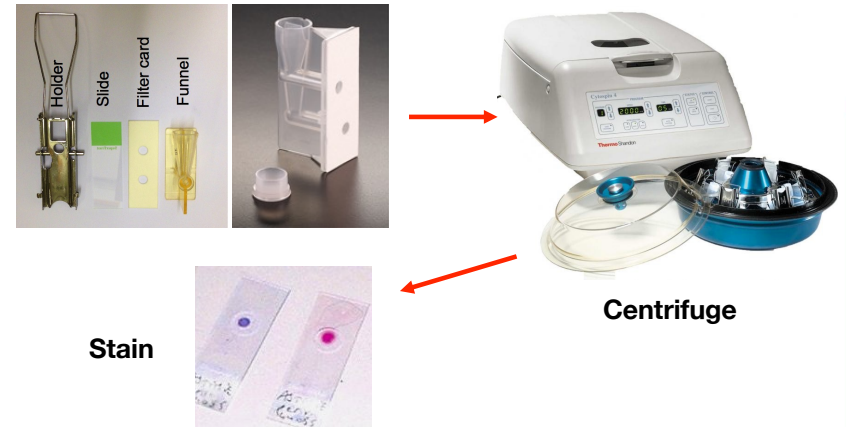




Bronchoalveolar Lavage (BAL)

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Bronchoalveolar Lavage (BAL) Preparation



Stain

Centrifuge

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Bronchoalveolar Lavage (BAL) Stains



Routine stains at CHLA:

- H&E x 2
- GMS: fungal elements
- AFB: mycobacterial organisms
- Gram: bacteria

Per order:

- Oil red O: lipid droplets
- Iron: hemosiderin deposits

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Bronchoalveolar Lavage (BAL) Adequacy



No clear criteria for adequacy but,

Not good if:

- < two million total cells [<10 cells in high-power field (HPF)]
- Severe degenerative changes

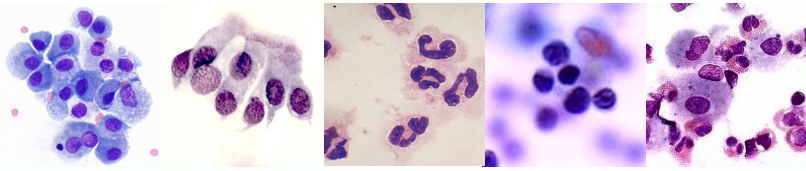
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Bronchoalveolar Lavage (BAL) Cellular Components



Components regularly seen:

- Alveolar macrophages
- Ciliated respiratory-type epithelium
- Neutrophils
- Eosinophils
- Lymphocytes
- Rare squamous cells



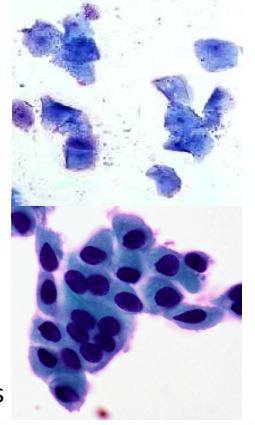
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Bronchoalveolar Lavage (BAL) Cellular Components



Abnormal findings:

- Abundant squamous cells
Oral contamination/aspiration
- Metaplastic respiratory-type epithelium
Chronic injury of the epithelium
- Microorganisms
- Increased neutrophils
- Increased eosinophils
- Increased lymphocytes
- Increased foamy macrophages
Lipid-laden macrophages
- Increased hemosiderin-laden macrophages



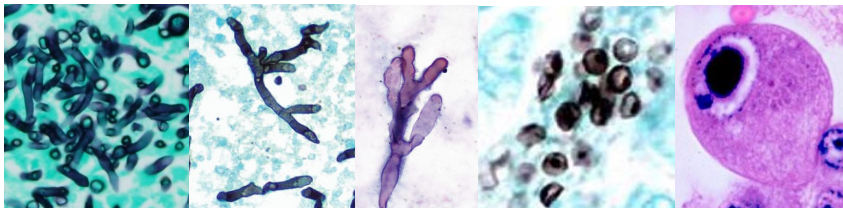
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Bronchoalveolar Lavage (BAL) Cellular Components

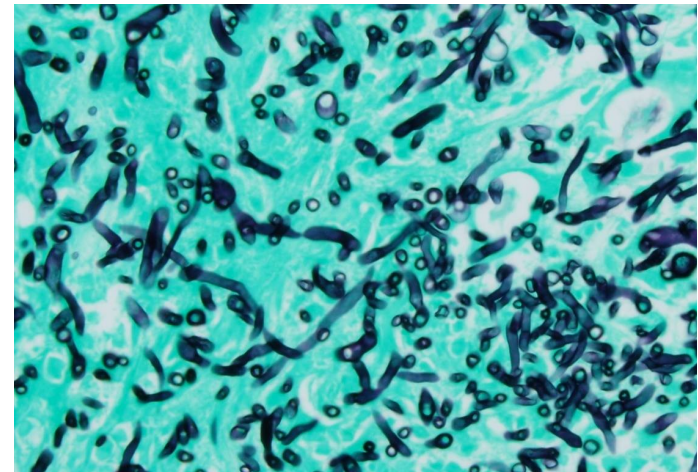


Microorganisms

- Fungal elements
- Bacteria (if a large single population)
- Viral cytopathic effects (CMV)



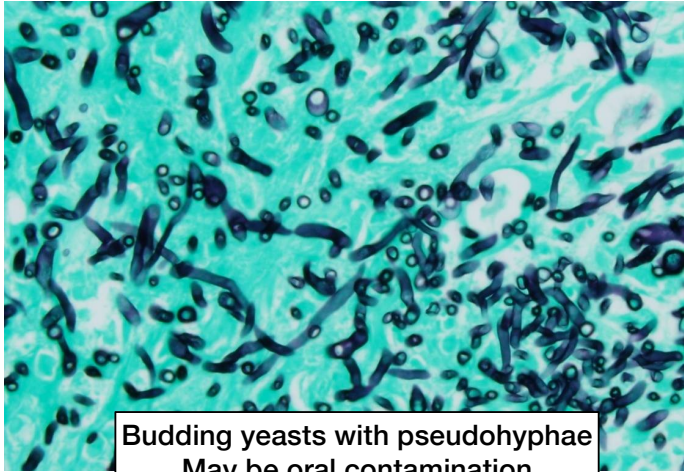
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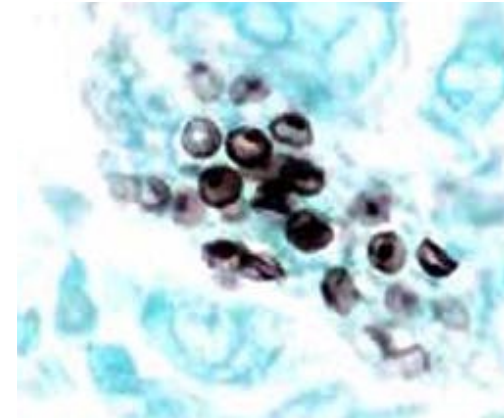
Budding yeasts with pseudohyphae



Candida spp.

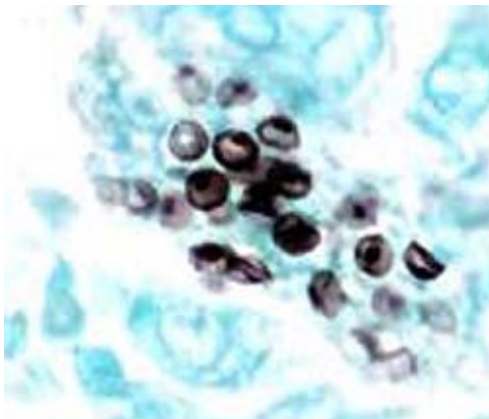


Budding yeasts with pseudohyphae
May be oral contamination



Crushed ping-pong balls

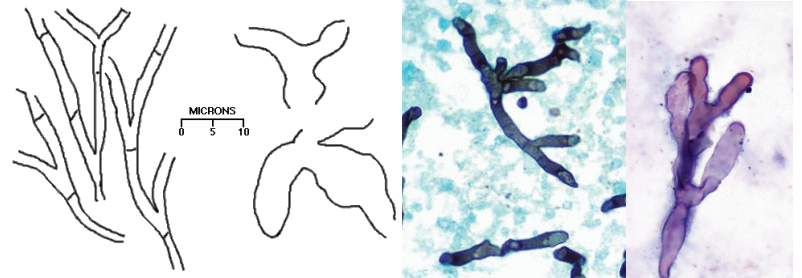
Pneumocystis



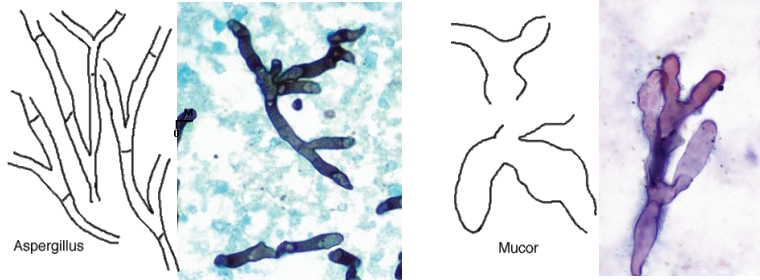
Crushed ping-pong balls



Hyphae



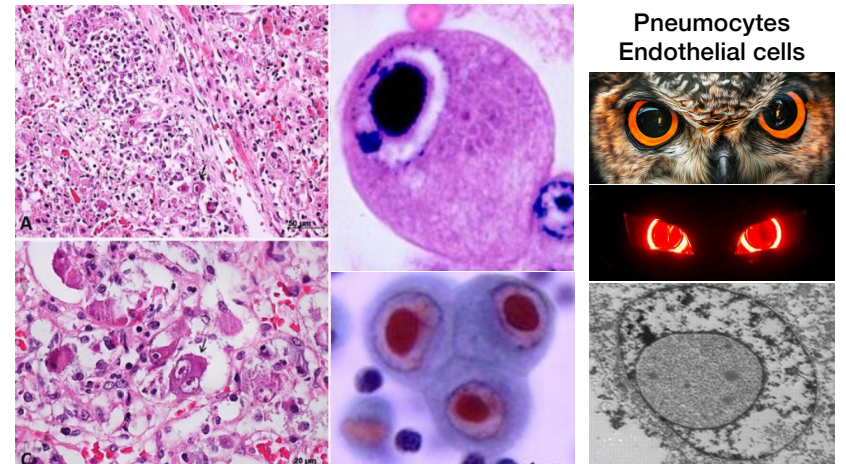
Hyphae



Narrow-angled branching septated hyphae
 Aspergillus spp., Fusarium spp.
 Scedosporium spp. etc.

Broad, ribbon-like, non-septated hyphae
 Zygomycetes (e.g. mucor, rhizopus)

Cytomegalovirus



Bronchoalveolar Lavage (BAL) Cell Differentials - Normal Children



Cell type	Mean	SD	Median	Min	Max
Macrophages %	81.2	12.7	84	34.6	94
Lymphocytes %	16.2	12.4	12.5	2	61
Granulocytes %	2.5	3.3	1.6	0.2	19
Neutrophils	1.9	2.9	0.9	0	17
Eosinophils	0.4	0.6	0.2	0	3.6
Basophils	0.3	0.5	0.1	0	2.8
Cells·kg ⁻¹ ×10 ⁴	14.6	13.3	10.7	1.5	66.8
Cells·ml ⁻¹ ×10 ⁴	10.3	11.1	7.3	0.5	57.1
Macrophages·ml ⁻¹	8.3	8.6	6.4	0.4	48
Lymphocytes·ml ⁻¹	1.8	3.4	1	0.1	23
Granulocytes·ml ⁻¹	0.3	0.4	0.1	0	2.2

Eur Respir J, 1994, 7, 1865-1870

Bronchoalveolar Lavage (BAL) Cell Differentials - Normal Adults



Normal Adults (Nonsmokers)	BAL Differential Cell Counts
Alveolar macrophages	>85%
Lymphocytes (CD4+/CD8+ = 0.9-2.5)	10-15%
Neutrophils	≤3%
Eosinophils	≤1%
Squamous epithelial*/ciliated columnar epithelial cells†	≤5%

Alveolar Macrophages



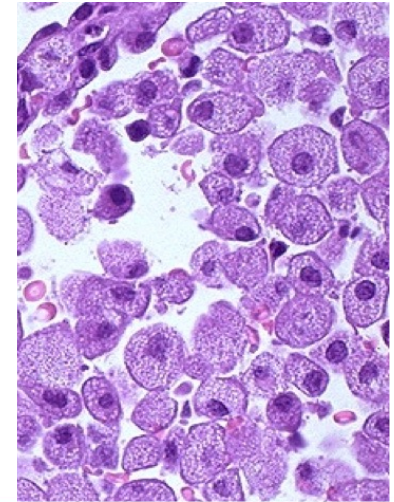
Normal: > 85-90%

Alveolar Macrophages



Foamy macrophages

- Aspiration
- Infections:
 - Mycobacteria: triacylglycerol-rich lipid bodies
- Hypersensitivity pneumonitis
- Drug-induced pneumonitis
 - Amiodarone
- Chemical inhalation
- Sarcoidosis
- Storage disorders:
 - Niemann-Pick disease
 - Gaucher disease
 - Hermansky-Pudlak disease
- Pulmonary alveolar proteinosis
- CGD

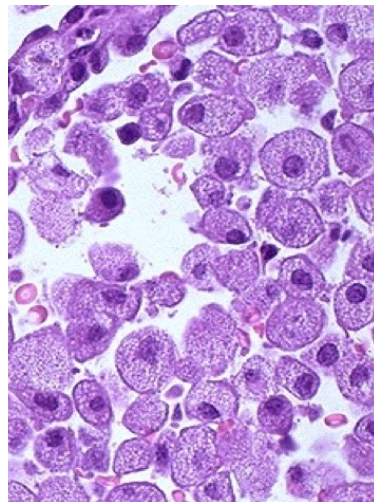


Alveolar Macrophages

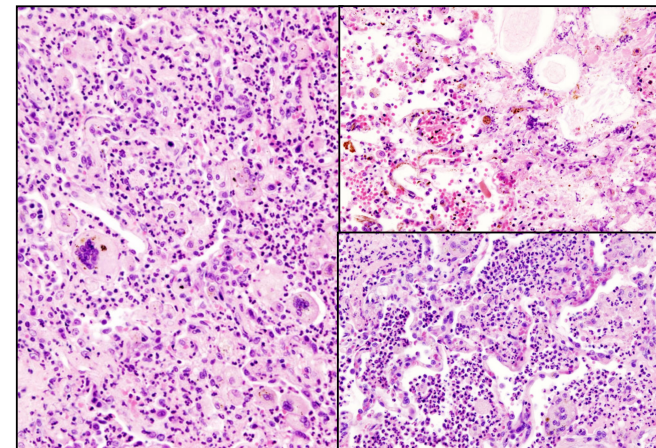


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Aspiration



Foamy macrophages (lipid-laden), neutrophils, multinucleated giant cells, vegetable/food matters -> airway-centered (peribronchiolar) interstitial inflammation -> fibrosis

Lipid Laden Macrophages - Oil red O



0: no opacification	
1: up to ¼ opacified	
2: ¼ to ½	
3: ½ to ¾	
4: totally opacified	

Lipid Laden Macrophage Index (LLMI)

Count 100 cells

$$\begin{aligned} & (\# \text{ of cells with type 4}) \times 4 \\ & (\# \text{ of cells with type 3}) \times 3 \\ & (\# \text{ of cells with type 2}) \times 2 \\ & + (\# \text{ of cells with type 1}) \times 1 \end{aligned}$$

LLMI

Lipid Laden Macrophages - Oil red O



Pediatric and Developmental Pathology 5, 551-558, 2002
DOI: 10.1007/s10024-002-0025-x
© 2002 Society for Pediatric Pathology

Limited Reliability of Lipid-laden Macrophage Index Restricts Its Use as a Test for Pulmonary Aspiration: Comparison with a Simple Semiquantitative Assay

YILING DING,^{1*} PIPPA M. SIMPSON,² DENNIS E. SCHELLHASE,² A. FRANCINE TRYKA,³ LIEMING DING,⁴ AND DAVID M. PARHAM¹

The sensitivity, specificity, and positive and negative predictive value (PPV and NPV) were **57%, 75%, 84%, and 69%**

Lipid Laden Macrophages - Oil red O



Diagnostic Cytopathology, Vol 38, No 12

Interobserver and Intraobserver Variability in the Calculation of the Lipid-Laden Macrophage Index: Implications for its Use in the Evaluation of Aspiration in Children

Michelle Reid-Nicholson, M.B.B.S.,^{1*} Renuka Kulkarni, M.B.B.S.,¹ Bamidele Adeagbo, M.B.B.S.,¹ Stephen Looney, Ph.D.,² and John Crosby, M.D.¹

Our study highlights **the lack of precision and subjectivity of the LLMI, as well as the significant inter and intra-observer bias that may occur among experienced and inexperienced pathologists.**

Lipid Laden Macrophages - Oil red O



Pulmonary Medicine
Volume 2012, Article ID 673637, 5 pages
doi:10.1155/2012/673637

Clinical Study

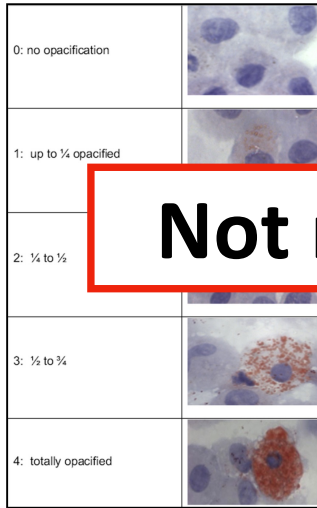
Lipid-Laden Alveolar Macrophages and pH Monitoring in Gastroesophageal Reflux-Related Respiratory Symptoms

R. Kitz, H. J. Boehles, M. Rosewich, and M. A. Rose

Pulmonology, Allergy and Cystic Fibrosis, Children's Hospital, Goethe University Frankfurt, Theodor Stern Kai 7, 60590 Frankfurt, Germany

Quantifying lipid-laden alveolar macrophages from BAL in children with GERD-related respiratory disorders does not have an acceptable specificity to prove chronic aspiration as an underlying etiology.

Lipid Laden Macrophages - Oil red O



Lipid Laden Macrophage Index (LLMI)

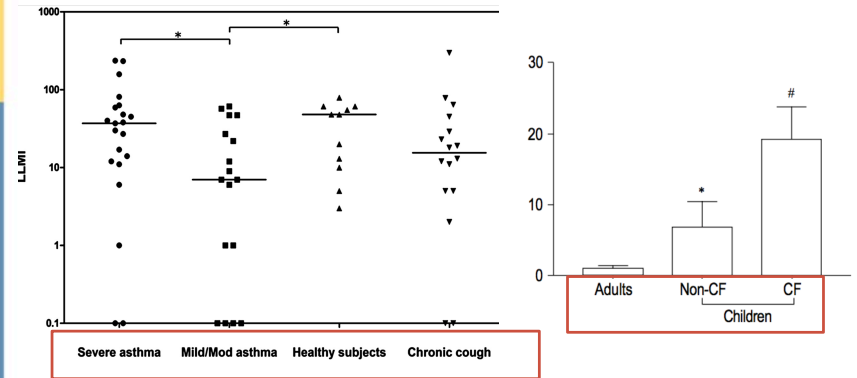
Not reliable

Count 100 cells

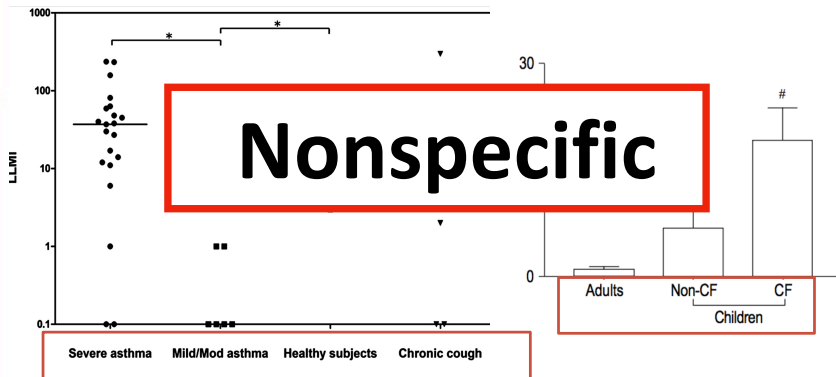
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LLMI

Lipid Laden Macrophages - Oil red O



Lipid Laden Macrophages - Oil red O



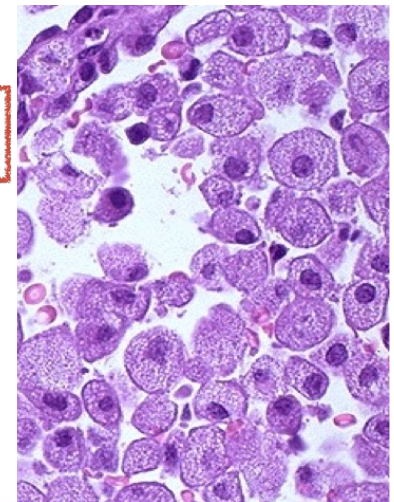
Nonspecific

Alveolar Macrophages

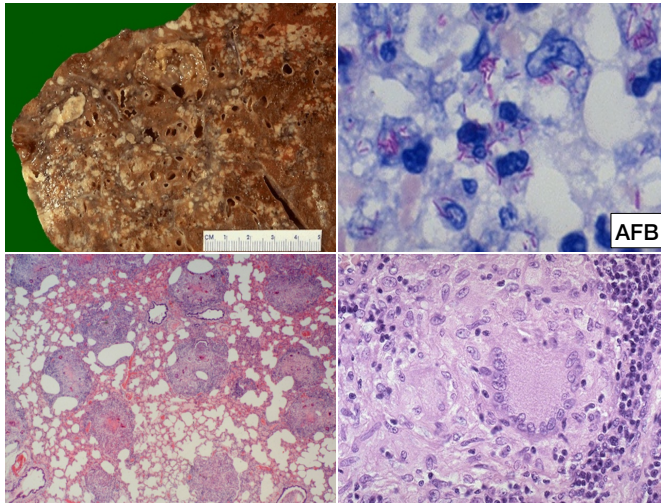


Foamy macrophages

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Mycobacterial Infection

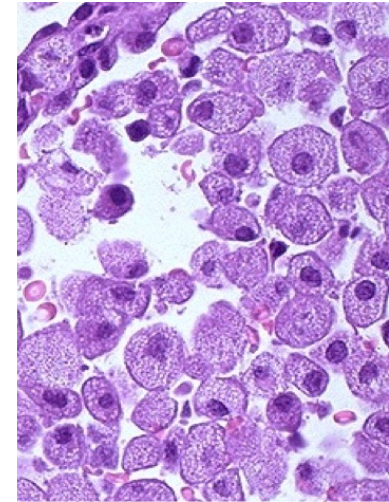


Alveolar Macrophages



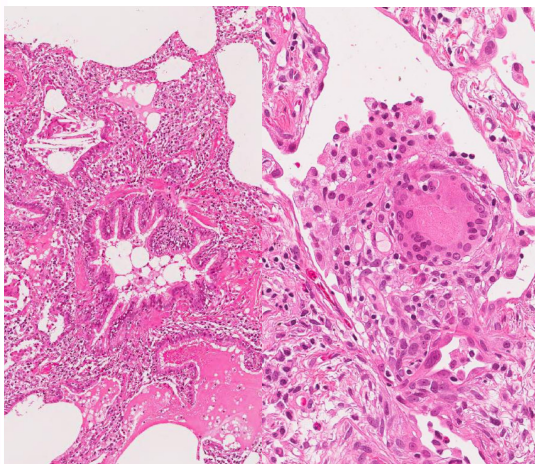
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Hypersensitivity Pneumonitis



- Foamy macrophages and lymphocytes in alveolar spaces
- Airway-centered inflammation
- Non-necrotizing granulomas
 - Giant cells with cholesterol clefts

Acute HP

- Neutrophilic
- Intra-alveolar fibrin deposition

Subacute HP

- Neutrophilic
- Lymphocytic with granulomas or giant cells
- Early fibrosis

Chronic HP

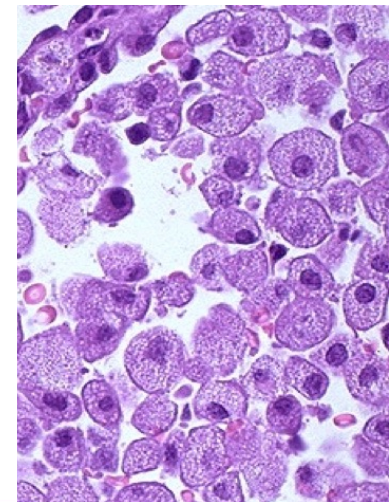
- Lymphocytic with granulomas or giant cells
- Diffuse fibrosis

Alveolar Macrophages



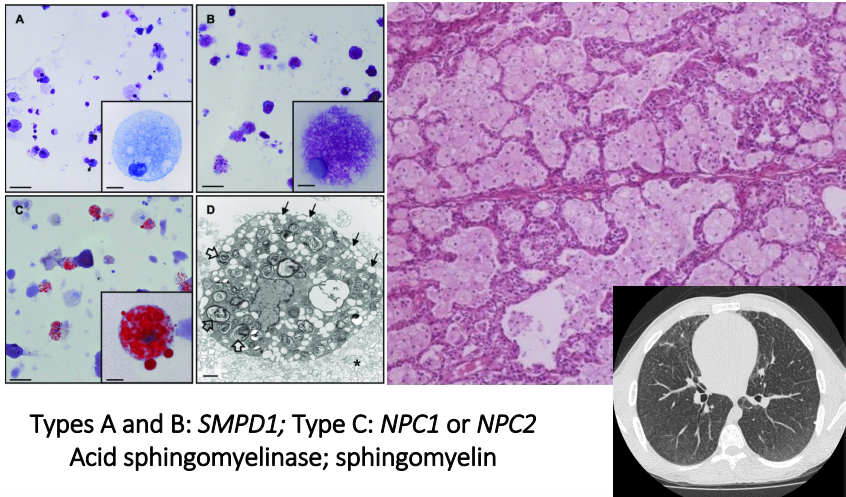
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Niemann-Pick Disease

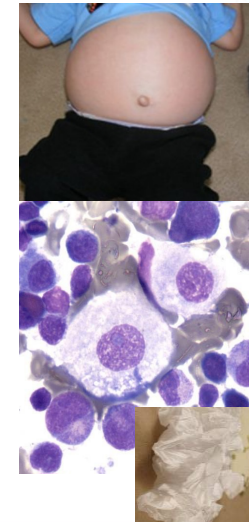


Types A and B: *SMPD1*; Type C: *NPC1* or *NPC2*
 Acid sphingomyelinase; sphingomyelin

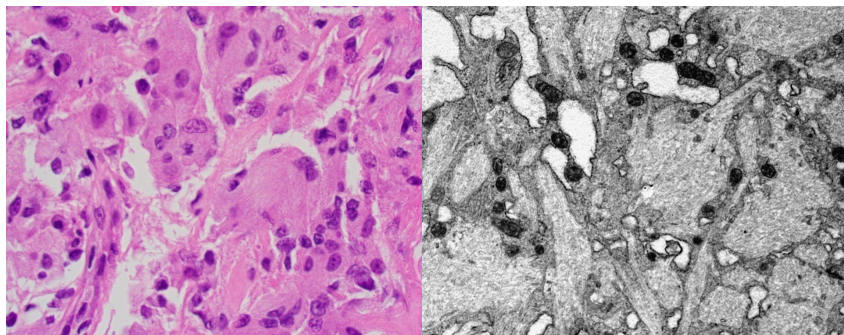
Gaucher disease



- The most common lysosomal storage disease
- *GBA*
- Beta-glucocerebrosidase
- Gaucher cells: glucosylceramide mainly in macrophages
- Three main subtypes:
 - Type 1 (non-neuronopathic form)
 - Type 2 (acute neuronopathic form)
 - Type 3 (subacute neuronopathic form)
- Common clinical manifestations:
 - Hepatomegaly, splenomegaly, pancytopenia, and bone abnormalities



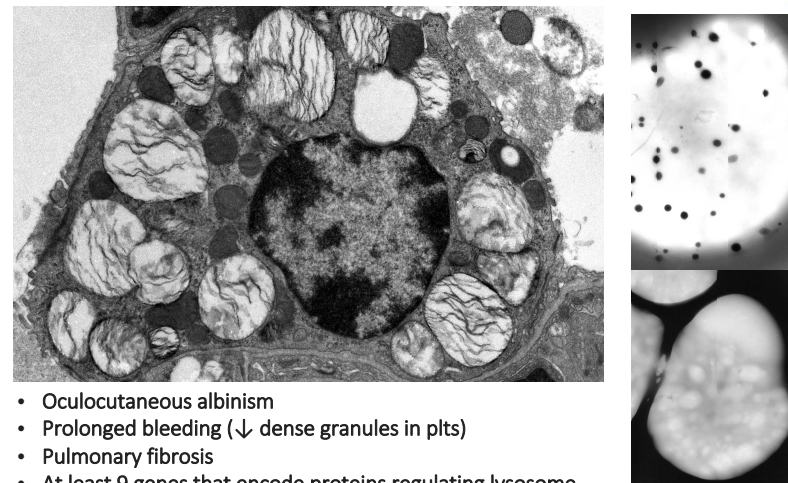
Gaucher disease



Foamy macrophages

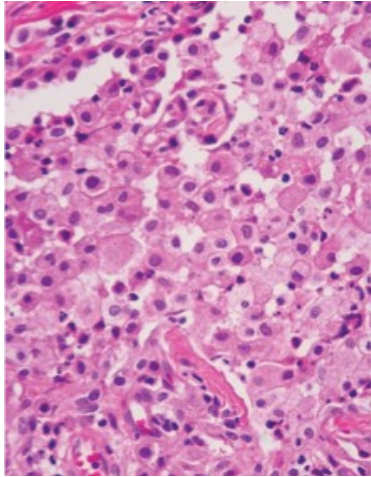
Tubular structures

Hermansky-Pudlak Syndrome



- Oculocutaneous albinism
- Prolonged bleeding (↓ dense granules in plts)
- Pulmonary fibrosis
- At least 9 genes that encode proteins regulating lysosome-related organelles

Amiodarone-Related Toxicity



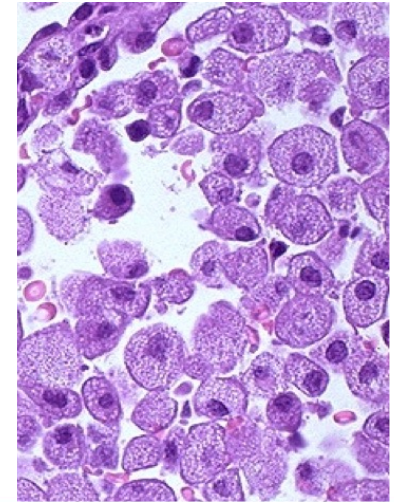
- Finely vacuolated foamy macrophages in alveoli and septa
- May have:
 - Diffuse alveolar damage/ cryptogenic organizing
 - Pneumonia-type changes
 - Nonspecific interstitial pneumonia (NIP)
 - Desquamative interstitial pneumonia (DIP)

Alveolar Macrophages



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Chronic Granulomatous Disease

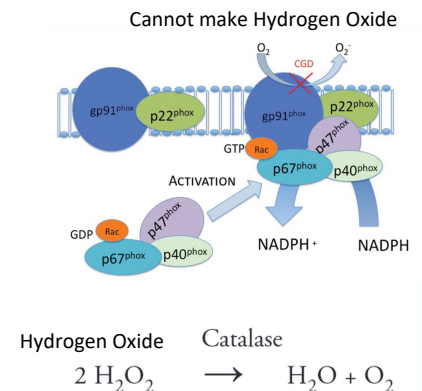
- A rare hereditary immunodeficiency
1/250,000 births
- X-linked CGD:
CYBB mutations at Xp21.1
2/3 of CGD
- Followed by mutations in *NCF1* (AR)
- 5% caused by mutations in *CYBA*, *NCF2* and *NCF4* (AR)

Chronic Granulomatous Disease

- Caused by mutations in the genes encoding subunits of the NADPH oxidase enzyme complex
- Defects lead to inability to kill intracellular pathogens.

susceptible to *catalase-positive microorganisms*:

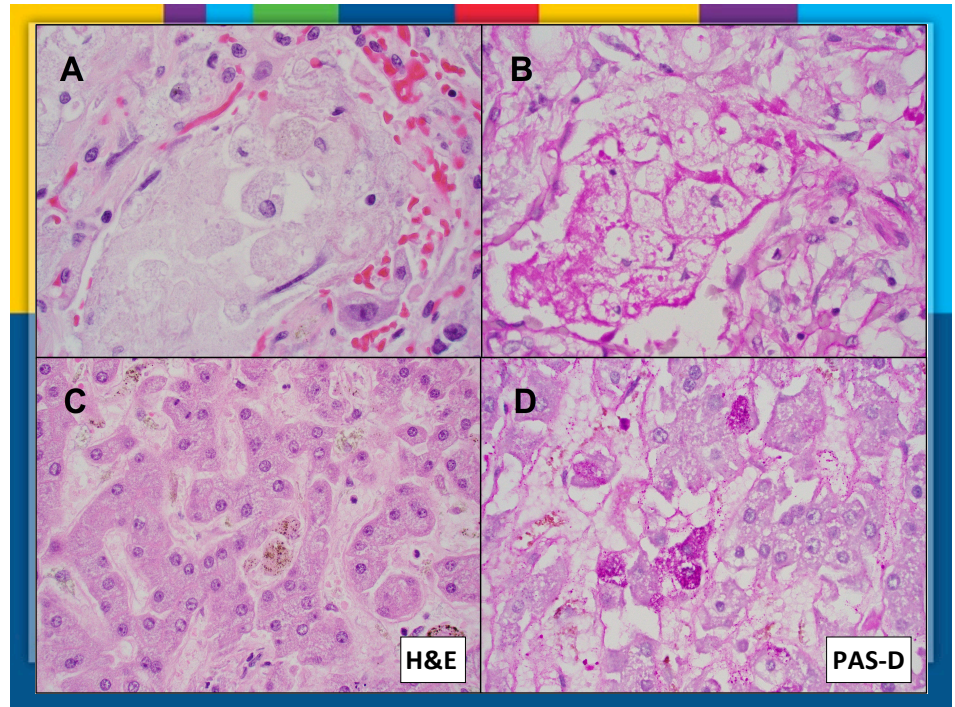
- Cannot borrow microorganisms hydrogen oxide
- *Staphylococcus aureus*, *P. aeruginosa*, *Burkholderia* spp, *Aspergillus* spp., *Nocardia* spp., *Enterobacteriaceae*, *Mycobacterium tuberculosis* (at body temp.)



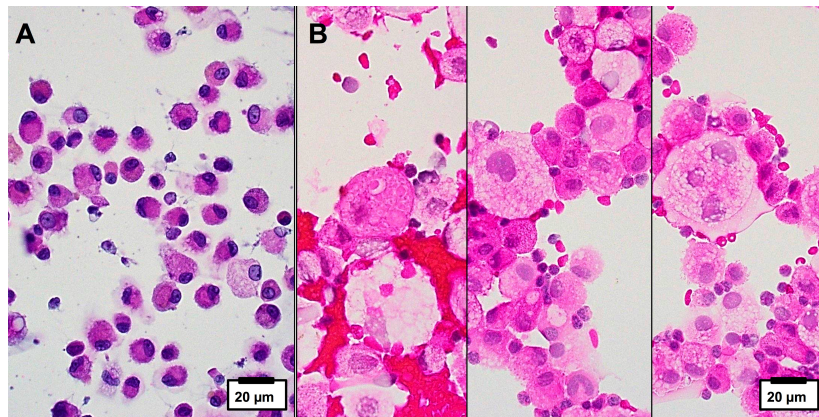
Chronic Granulomatous Disease

First described as “a syndrome of recurrent infection and infiltration of viscera by pigmented lipid histiocytosis”

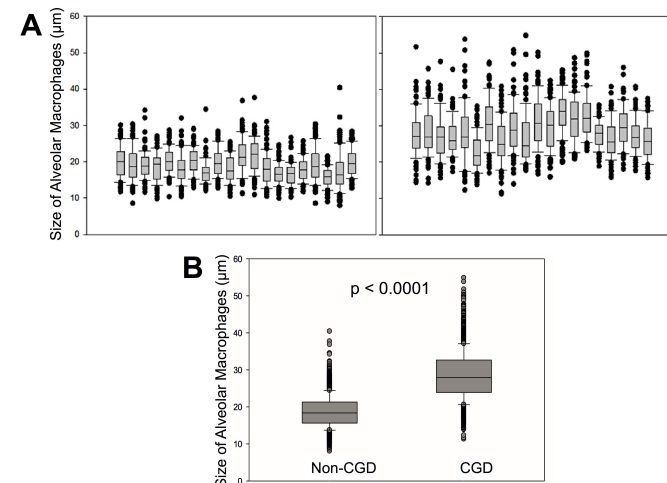
Lightly pigmented histiocytes are increased in the lymph nodes, spleen, liver, lung, skin and brain.



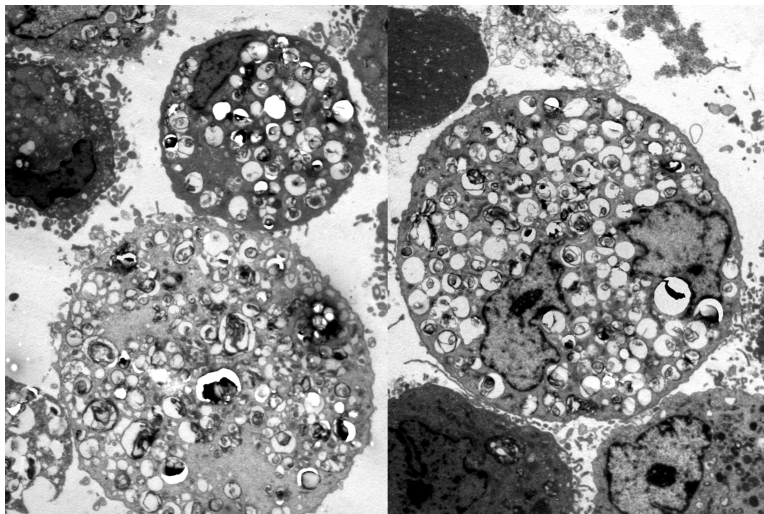
Chronic Granulomatous Disease



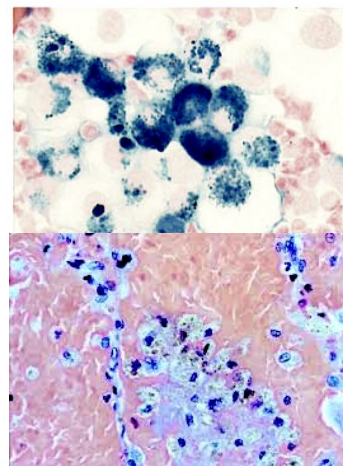
Chronic Granulomatous Disease



Chronic Granulomatous Disease



Hemosiderin-Laden Macrophages Iron Stain

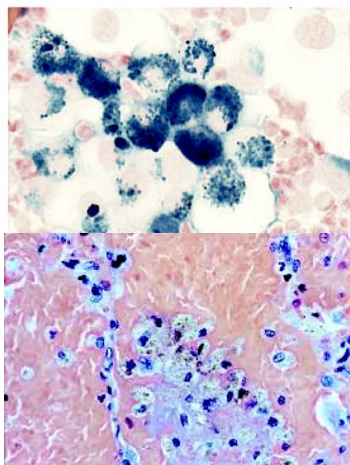


Increased hemosiderin-laden macrophages

- >20%: highly specific and sensitive for alveolar hemorrhage
- Subclinical hemorrhage is possible at as low as 5%.

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Hemosiderin-Laden Macrophages



Alveolar hemorrhage with capillaritis:

Vasculitis commonly affect the lungs:

- Granulomatosis with polyangiitis (Wegener)/microscopic polyangiitis
- Churg-Strauss vasculitis
- Goodpasture syndrome

Vasculitis uncommonly affect the lungs:

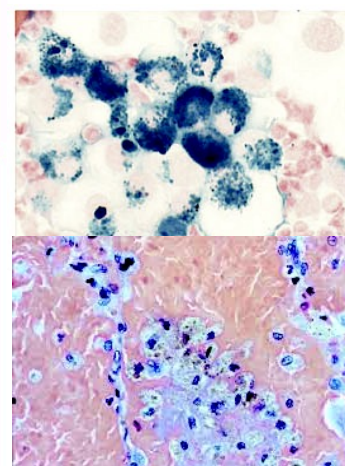
- Polyarthritid nodosa
- Takayasu arteritis
- Henoch-Shonlein purpura (HSP)
- Bechet disease

Systemic autoimmune diseases

- SLE
- Scleroderma

Transplant rejection

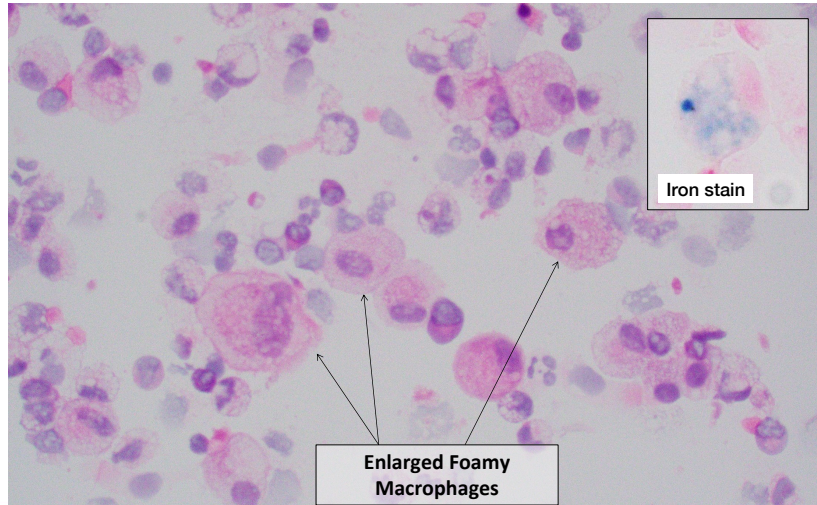
Hemosiderin-Laden Macrophages



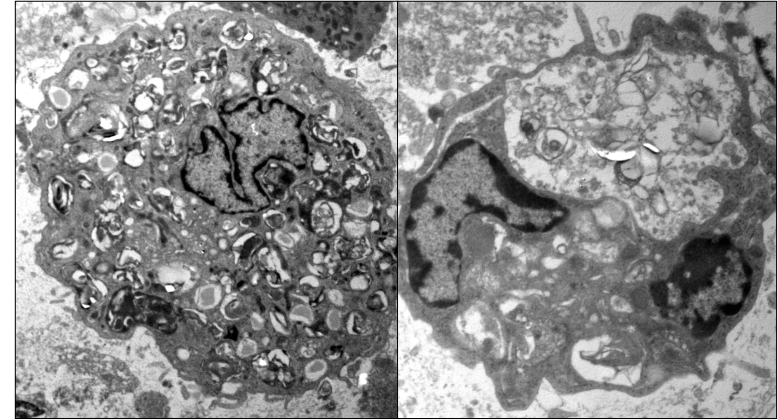
Alveolar hemorrhage without capillaritis:

- Diffuse alveolar damage (ARDS)
- Autoimmune diseases (SLE) – direct alveolar damage
- Hemorrhage secondary to other lung injuries:
 - Infections
 - Drug toxicity
 - Radiation
 - Trauma
 - Foreign body
- Cardiovascular diseases
- Malignancies
- Coagulopathies
- VOD
- Lymphangioleiomyomatosis
- Idiopathic pulmonary hemosiderosis (IPH) – endothelial damage (capillaritis?)

COPA Syndrome



COPA Syndrome



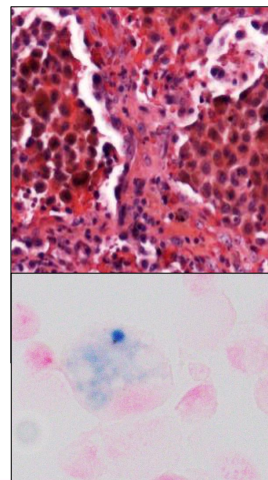
Enlarged lysosomes (organelles that digest excess materials in the cell) filled with flocculent materials

Main Histologic Features Reported in COPA Syndrome



Pulmonary hemorrhage (diffuse alveolar hemorrhage)

Capillaritis: inflammation and disruption of capillaries in the alveolar septae

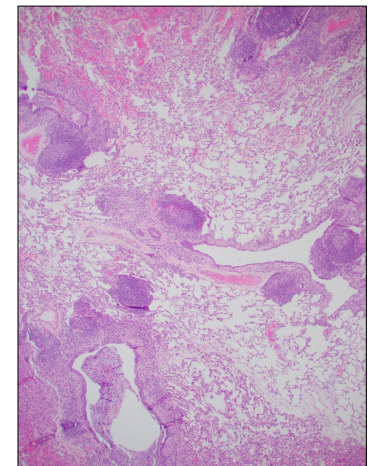


Main Histologic Features Reported in COPA Syndrome



Follicular bronchiolitis

- Aggregates of inflammatory cells (lymphocytes) adjacent to the bronchioles
- Nonspecific:
 - Can be seen in many other immune deregulatory syndromes and autoimmune diseases
 - Particular types of lymphocytes??
 - Further investigations required

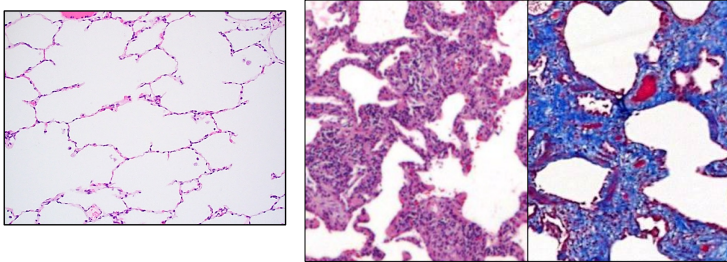


Main Histologic Features Reported in COPA Syndrome



Interstitial lung disease

Inflammation within the alveolar septae (capillaries/interstitium) -> scarring of the septae

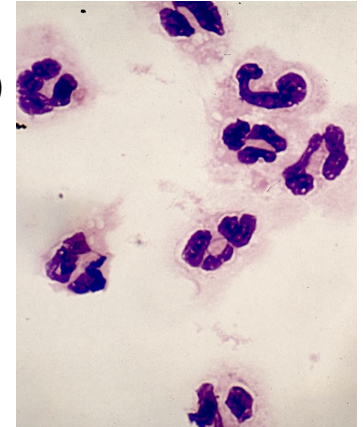


Neutrophilic Cellular Pattern



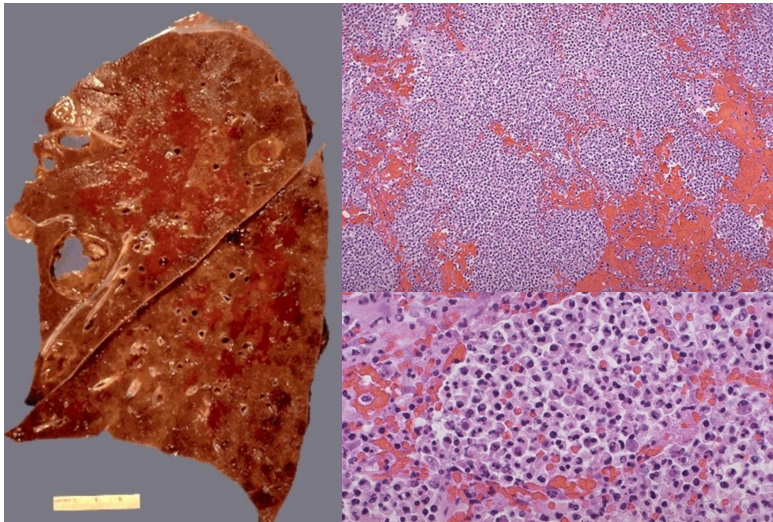
Increased neutrophils (>3-5%)

- Pneumonia, bronchitis, bronchiolitis (e.g., infections)
- Aspiration
- Diffuse alveolar damage

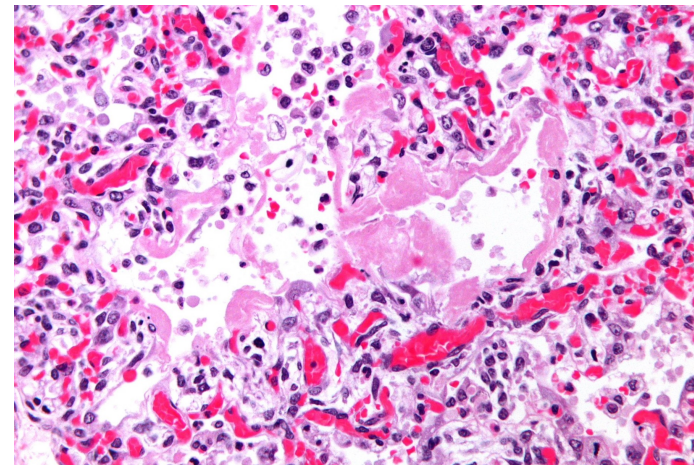


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Acute Pneumonia



Diffuse Alveolar Damage



Disruption of alveolar wall, fibrins deposits, neutrophils → organizing pneumonia

Eosinophilic Cellular Pattern



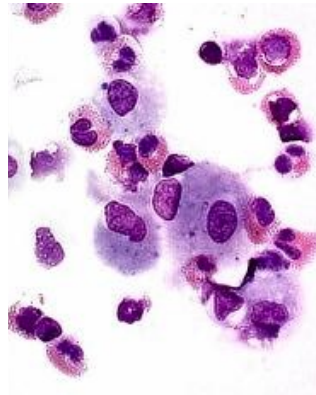
Increased eosinophils ($\geq 1\%$)

High counts (approximately >25%)

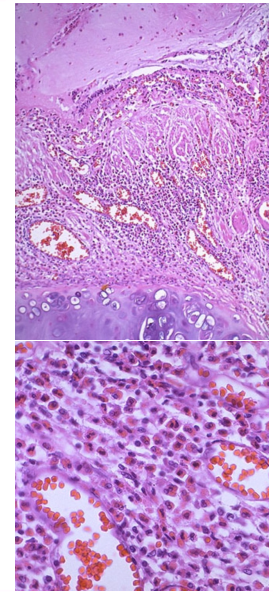
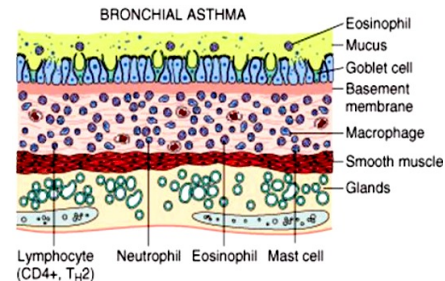
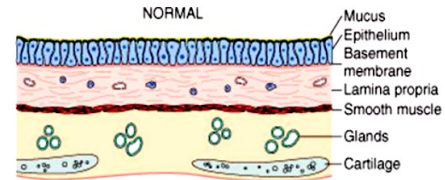
- Eosinophilic pneumonia
 - Churg-Strauss syndrome
 - Allergic bronchopulmonary aspergillosis (ABPA)
 - Hypereosinophilic syndrome
 - Tropical pulmonary eosinophilia (filarial infection)

Mild to moderate counts (approximately <25%)

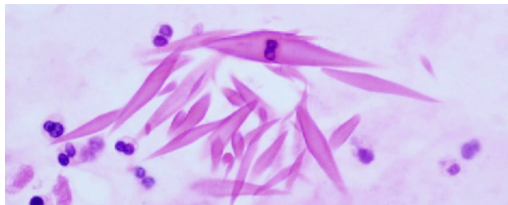
- Drug-induced pneumonitis
- Asthma
- Infections (bacterial, fungal, parasitic, pneumocystis, mycobacterial)
- Hodgkin disease
- LCH
- Interstitial pneumonitis associated with connective tissue disorders and sarcoidosis



Asthma



Asthma - Products of eosinophilic granules

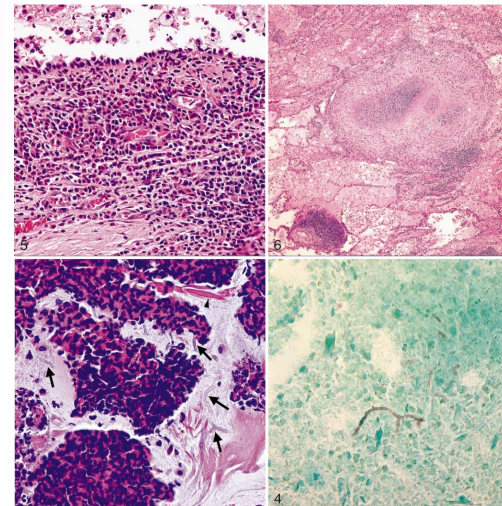


Charcot Leyden crystals



Curschmann spirals

Allergic bronchopulmonary aspergillosis

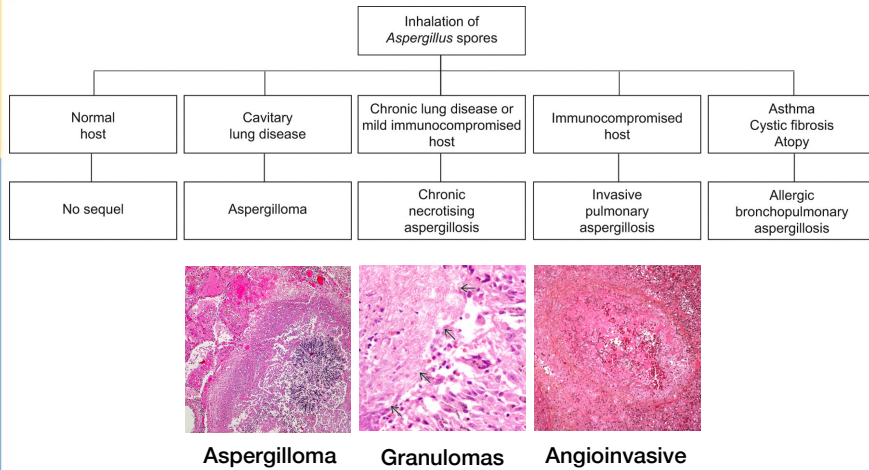


- Hypersensitivity reaction to *Aspergillus* spp.
- Higher risk in cystic fibrosis or steroid-dependent asthma
- Mucoid impaction of bronchi
- Eosinophilic pneumonia
- Bronchocentric granulomatosis

Aspergillus-Related Lung Disease



The spectrum of pulmonary aspergillosis



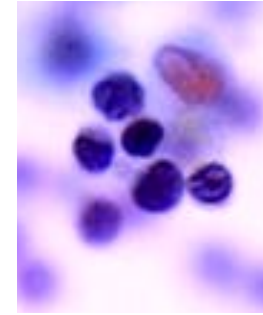
M. Kousha et al. Eur Respir Rev 2011;20:156-174

Lymphocytic Cellular Pattern



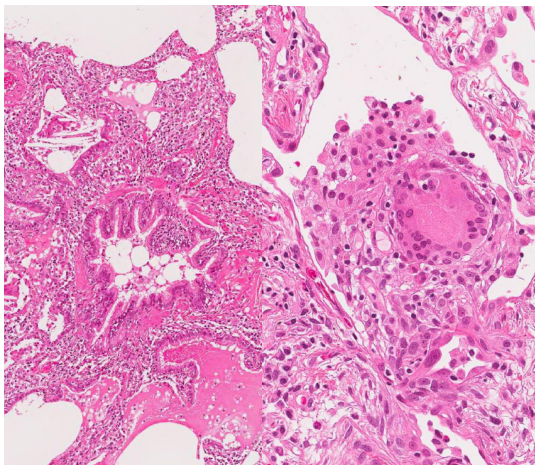
Increased lymphocytes (>15%)

- Hypersensitivity pneumonitis (60-80%)
- Sarcoidosis (acute phase: 40-60%)
- Collagen vascular disease
- Drug-induced pneumonitis
- Radiation
- Cryptogenic organizing pneumonia
- Lymphoproliferative disorders/lymphoma
- LCH
- Mycobacterial infection



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Hypersensitivity Pneumonitis



- Foamy macrophages and lymphocytes in alveolar spaces
- Airway-centered inflammation
- Non-necrotizing granulomas
 - Giant cells with cholesterol clefts

Acute HP

- Neutrophilic
- Intra-alveolar fibrin deposition

Subacute HP

- Neutrophilic
- Lymphocytic with granulomas or giant cells
- Early fibrosis

Chronic HP

- Lymphocytic with granulomas or giant cells
- Diffuse fibrosis

Lymphocytic Cellular Pattern (Adults)



CD4 : CD8 raised	CD4 : CD8 normal	CD4 : CD8 lowered
Sarcoidosis	Tuberculosis	Hypersensitivity pneumonitis
Berylliosis	Lymphangioleiomyomatosis	Silicosis
Asbestosis		Drug induced
Crohn's disease		BOOP
Rheumatoid arthritis		HIV infection

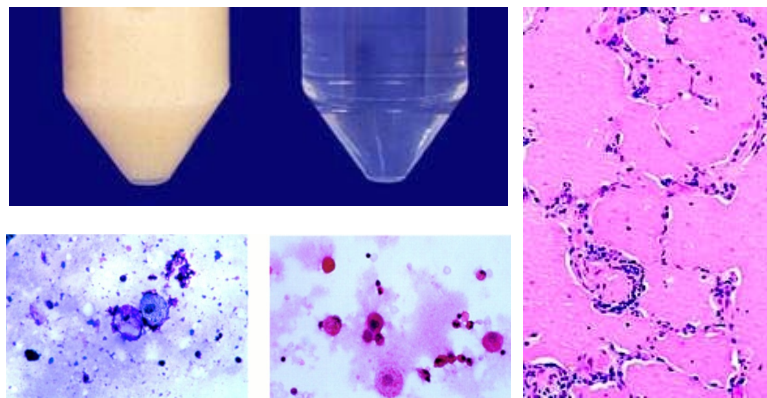
Redrawn from Poulter LW, Rossi GA, Bjerner L, et al, Eur Respir Rev 1992; 2:75.

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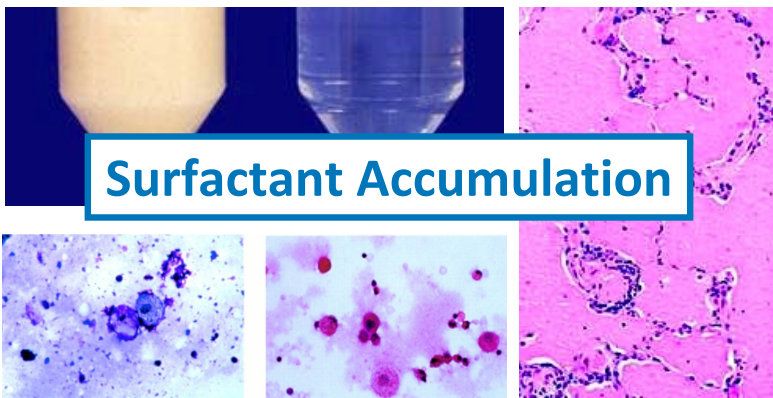


Pulmonary Alveolar Proteinosis

Pulmonary Alveolar Proteinosis

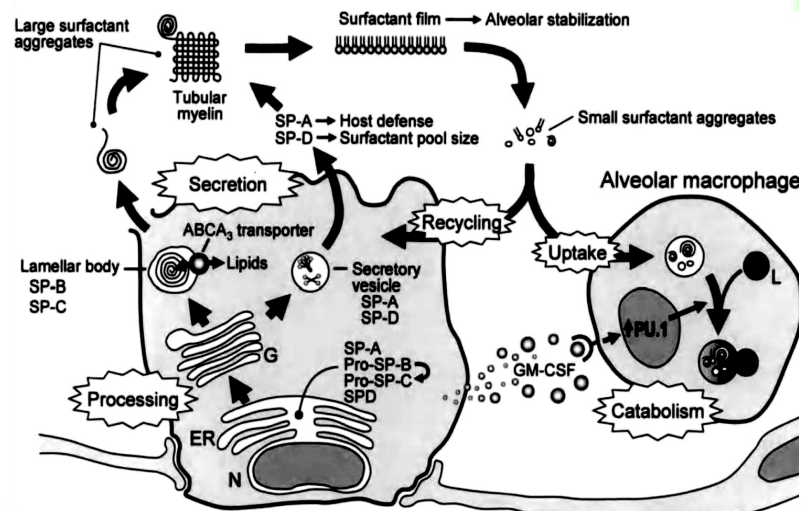


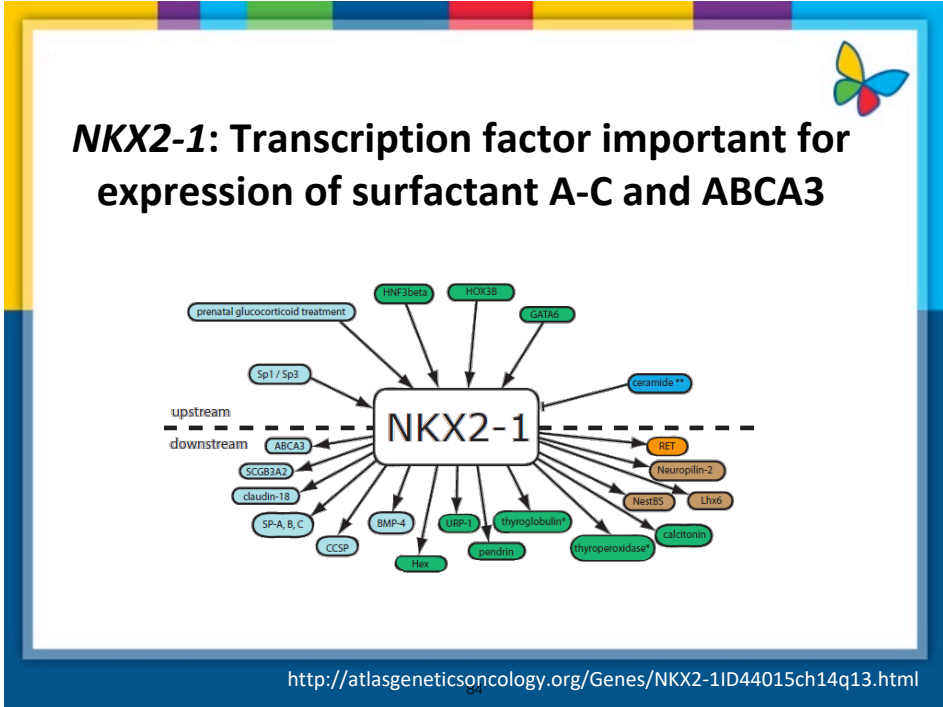
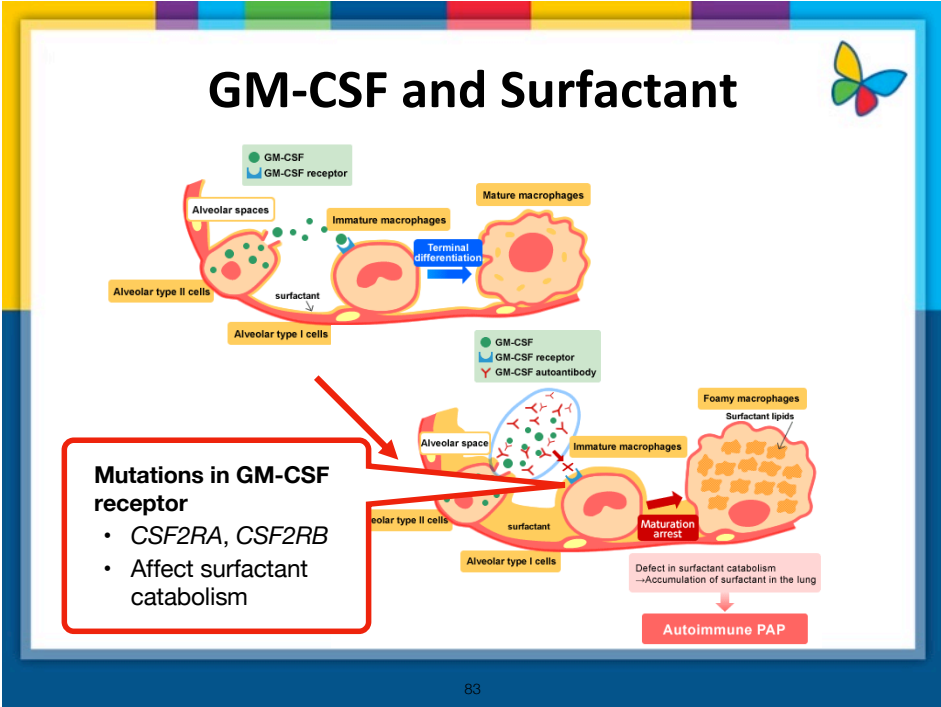
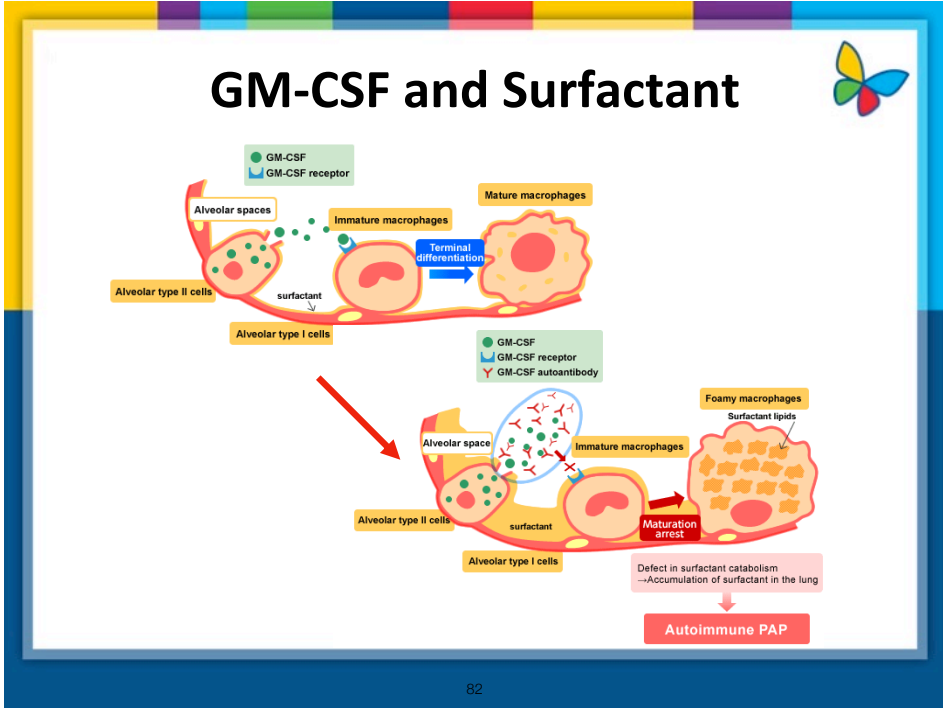
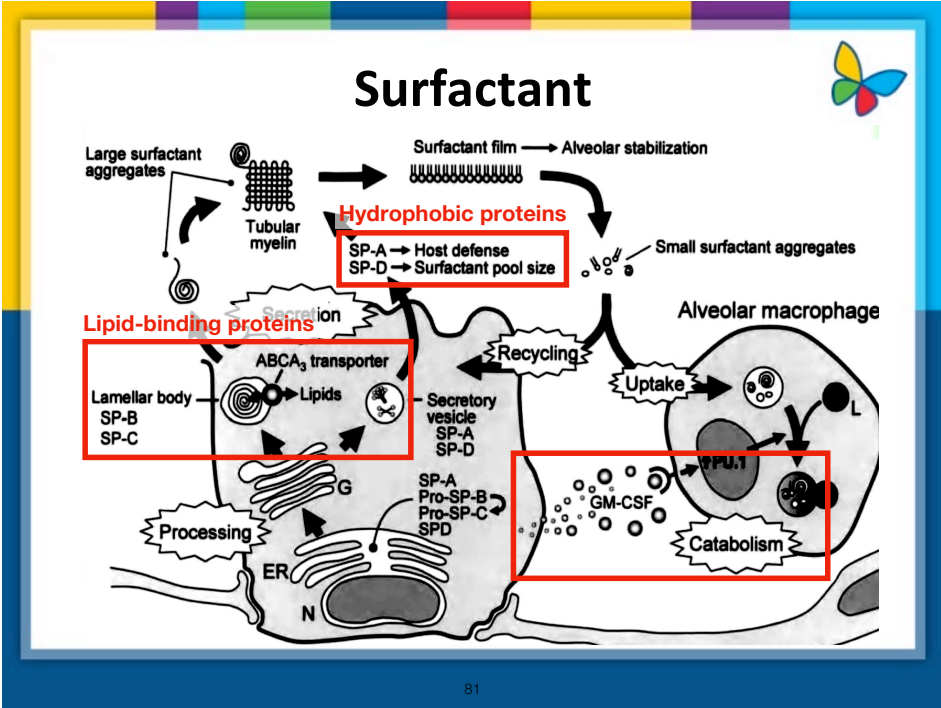
Pulmonary Alveolar Proteinosis



Surfactant Accumulation

Surfactant





Pulmonary Alveolar Proteinosis



Congenital forms

Genetic disorders of surfactant dysfunction

	SFTPB	SFTPC	ABCA3	NKX2.1
Mode of inheritance	Autosomal recessive	Autosomal dominant, <i>de novo</i>	Autosomal recessive	Autosomal dominant, <i>de novo</i>
Pulmonary presentations	Neonatal respiratory distress syndrome	Childhood interstitial lung disease Adult interstitial lung disease Neonatal respiratory distress syndrome	Neonatal respiratory distress syndrome Childhood interstitial lung disease	Neonatal respiratory distress syndrome Childhood interstitial lung disease Recurrent infection No pulmonary involvement
Course	Neonatal lethal	Highly variable Survival until 6th decade reported	Neonatal lethal Variable severity in childhood	Neonatal lethal Variable severity in childhood
Treatment options	Supportive Lung transplant ^[1,2]	Supportive ^[3] Corticosteroids ^[4,5] Hydroxychloroquine ^[4,5] Azithromycin ^[4] Lung transplant ^[2]	Supportive Corticosteroids ^[6] Hydroxychloroquine ^[6,7] Lung transplant ^[8,9]	Supportive

Mutations in GM-CSF receptor genes

- *CSF2RA*, *CSF2RB*
- Affect surfactant catabolism

UpToDate

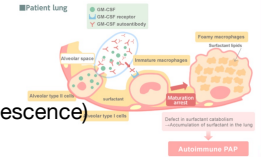
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Pulmonary Alveolar Proteinosis



Acquired form

- Antibodies to GM-CSF (autoimmune PAP)
- 90 percent of cases: adults
 - Reported in only a few children (late childhood or adolescence)



Secondary form

- Infections (*Nocardia*, mycobacteria, *Pneumocystis*, HIV)
- Hematologic malignancies
- Immunodeficiencies (HIV infection, SCID)
- Chemical inhalation [insecticides, fumes, minerals (silica, aluminum, and titanium)]

Idiopathic form

- Unclear etiology
- In about 1/3 of infant cases suggesting a surfactant defect, no mutations identified
- Most older children with PAP do not have anti-GM-CSF antibodies.

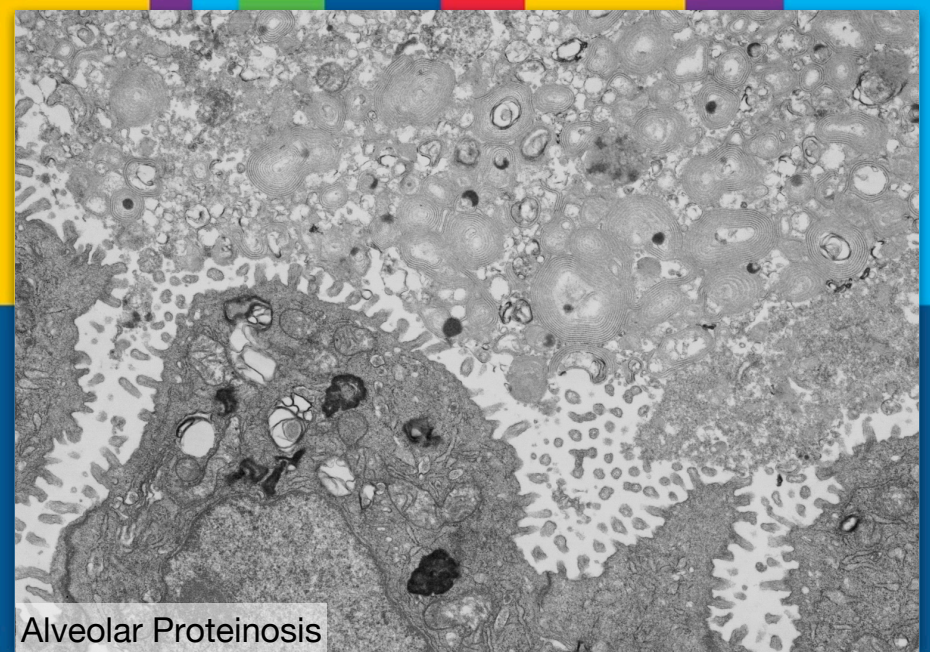
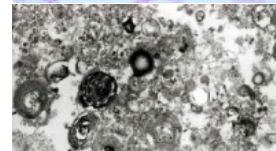
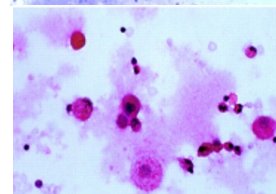
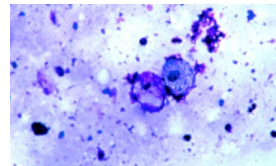
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Pulmonary Alveolar Proteinosis



BAL

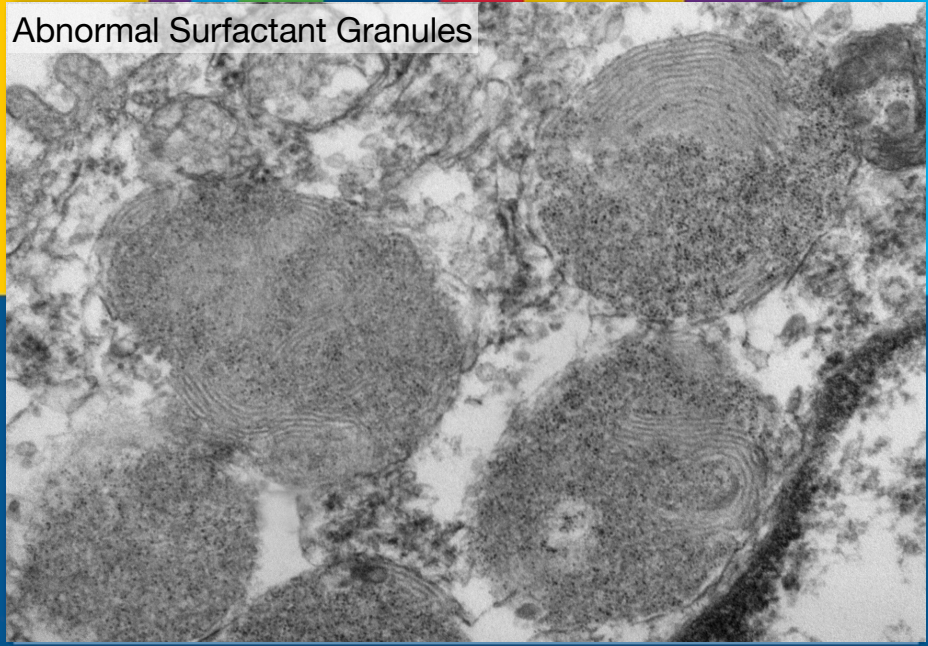
- Typically milky fluid
- Light microscopy:
 - Amorphous, lipo-proteinaceous material
 - Brightly positive with PAS stain
- Differential cell count:
 - Macrophage predominance without significant numbers of inflammatory cells
 - Large foamy macrophages are often seen
- **Electron microscopy can be helpful**



Alveolar Proteinosis

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Abnormal Surfactant Granules

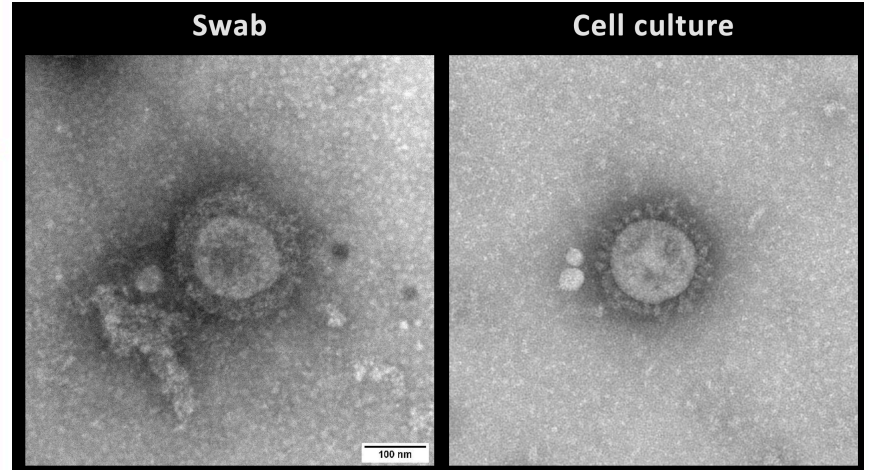


SARS-CoV-2

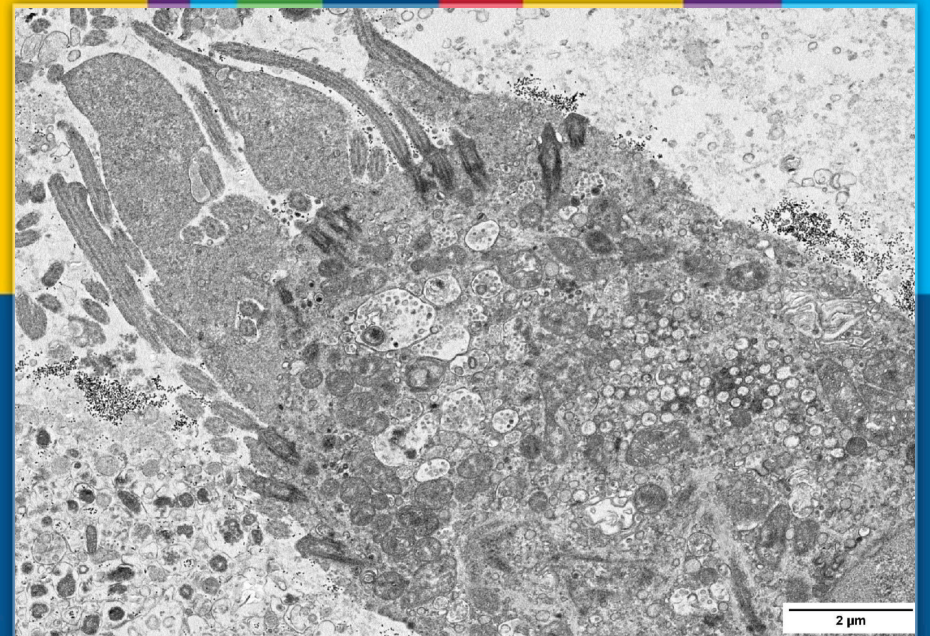
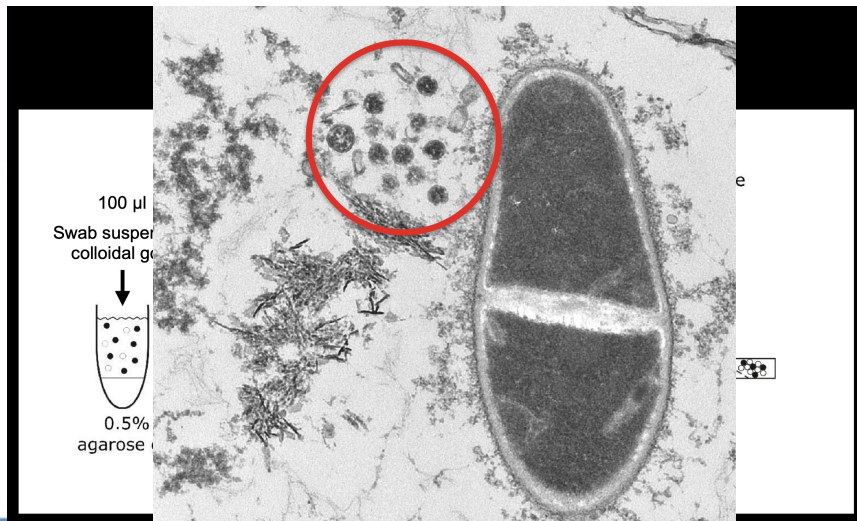


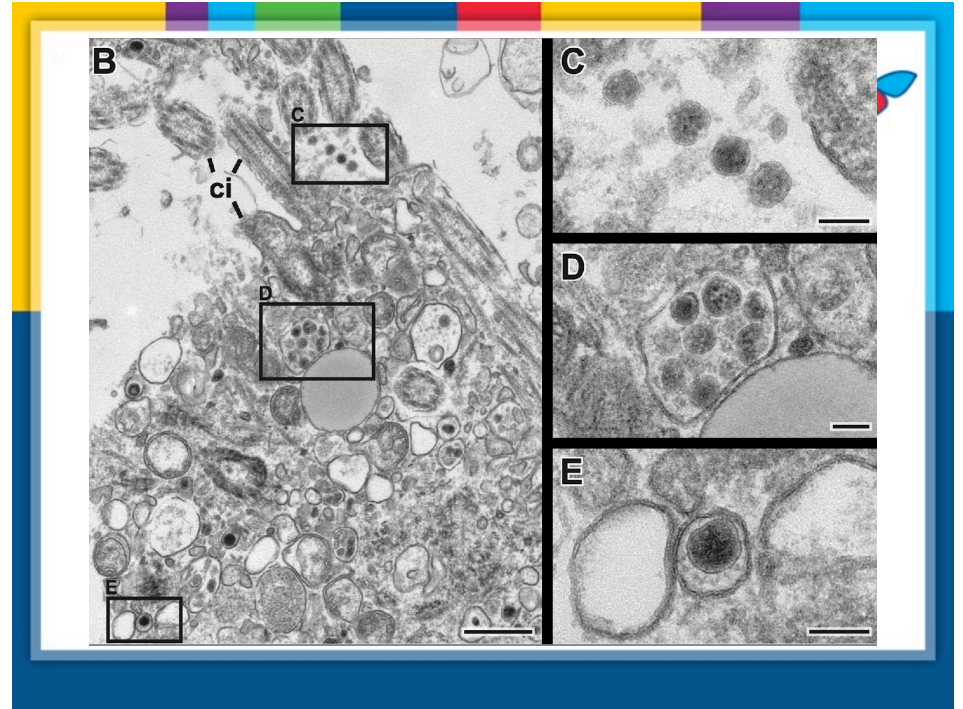
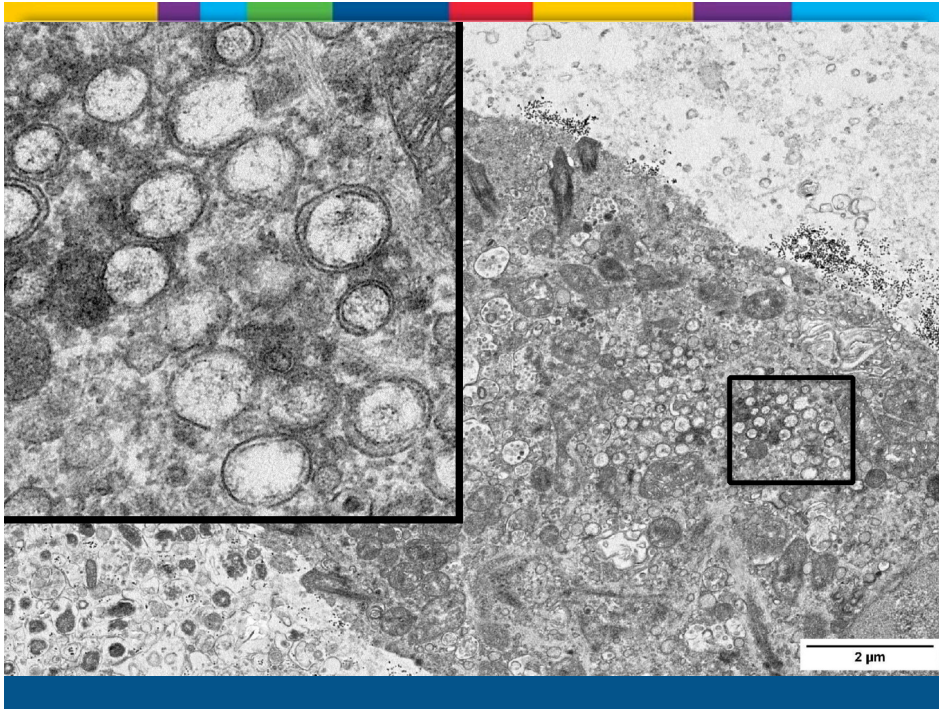
Swab

Cell culture



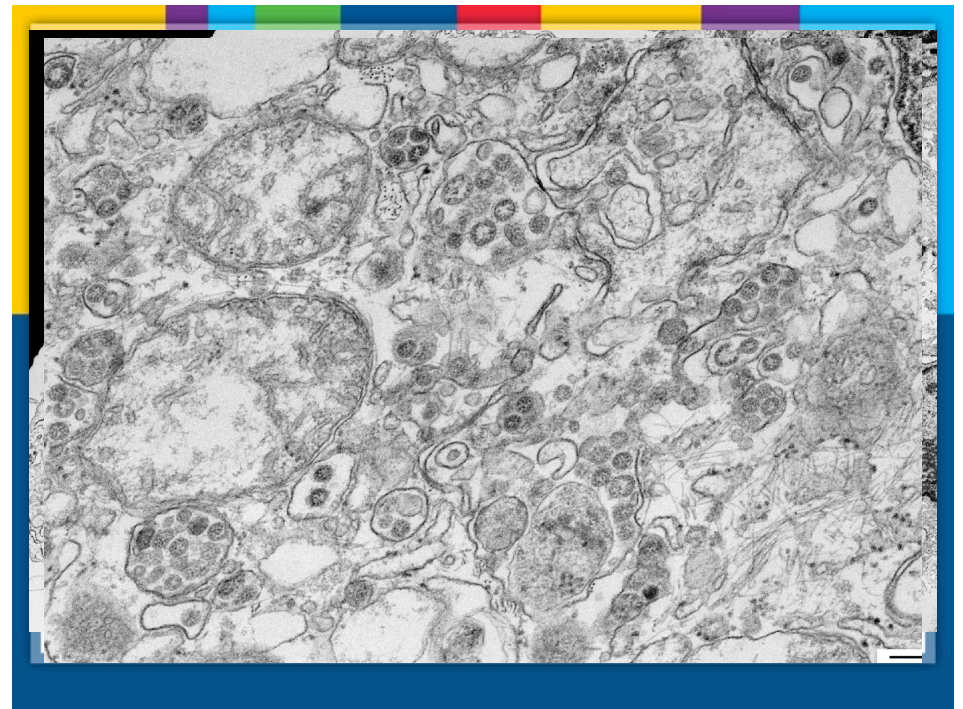
SARS-CoV-2 (Swabs)





SARS-CoV-2 (Lung Tissue)

Patient	Gender (Male/ Female)	Age	Clinical cause of death	Days from onset to death	Days from hospitalization to death	Comprehensive histologic DAD phase
CO3	M	78	Cardiogenic shock	42	35	Mid-phase
CO4	M	59	Refractory hypoxaemia	31	30	Late phase
CO5	M	79	Refractory hypoxaemia	20	16	Late phase
CO6	F	77	Haemorrhagic shock	5	4	Early phase
CO7	M	69	Refractory hypoxaemia	38	33	Mid-phase
CO8	M	47	Cardiogenic shock	14	9	Early phase

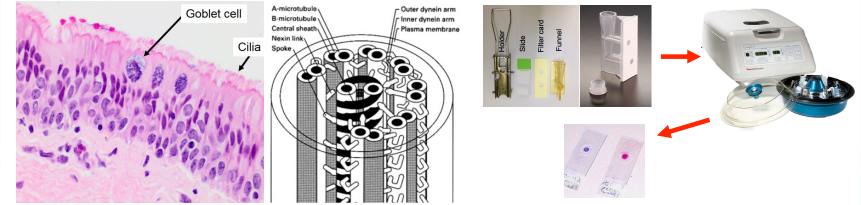


SERS-Cov-2 Infection



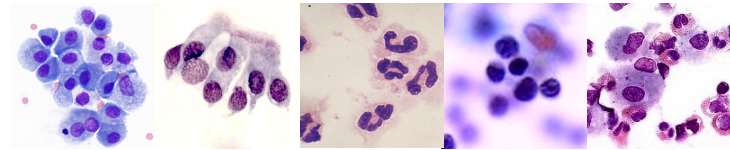
- Structural presentation is difficult (autolysis, pathologic damage, chemical fixation).
- Infection can be focal.
- Detection of viral RNA or protein does not mean that viral particles are still present.
- Alveolar damage may not be caused by viral replication.
- The alveolar damage is similar to ARDS caused by other etiologies (infections, trauma, etc.)

Summary



1. Normal airway histology/ultrastructure

2. BAL preparation



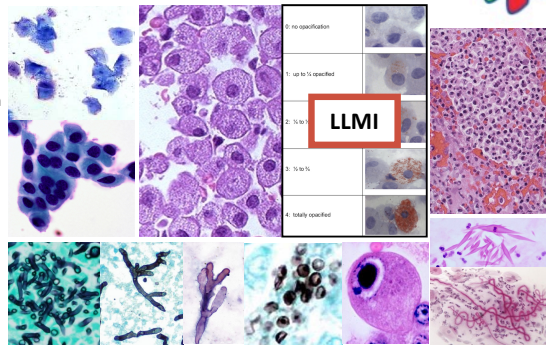
2. Normal BAL cytology

Summary



4. Abnormal findings

- Abundant squamous cells
Oral contamination/apiration
- Metaplastic respiratory-type epithelium
Chronic injury of the epithelium
- Microorganisms
- Increased neutrophils
- Increased eosinophils
- Increased lymphocytes
- Increased foamy macrophages
Lipid-laden macrophages
- Increased hemosiderin-laden macrophages



5. PAP

