REKAP DAFTAR HADIR KULIAH PAKAR BLOK 7/SISTEM RESPIRASI & PENGENALAN KETERAMPILAN MEDIK
SEMESTER GASAL TAHUN AKADEMIK 2020/2021
PERIODE : 21 SEPTEMBER - 20 OKTOBER 2020

NO	NAMA DOSEN	DEPARTEMEN	JLH JAM RENCANA	BLOK 7 & PKM									REALISASI KP
				SEPTEMBER			OKTOBER						
				21	24	28	1	5	8	12	15	19	
1	dr. June L. Nainggolan, MS, SpKL	Biomedik Dasar	4	4	-	-	-	-	-	-	-	-	4
2	dr. Frisca Angreni, M.Biomed.	Anatomi	4	4	-	-	-	-	-	-	-	-	4
3	dr. Wawat Hartiaswati, MS, PHK	Anatomi	4	4	-	-	-	-	-	-	-	-	4
4	dr. Nur Nunu Prihantini, M.Si	Biokimia Kedokteran	4	-	4	-	-	-	-	-	-	-	4
5	dr. Fajar L. Gultom, SpPA	Pato. Anatomi	4	-	4	-	-	-	-	-	-	-	4
6	dr. Kurniyanto, SpPD	Ilmu Peny. Dalam	20	-	4	-	-	4	4	4	-	4	20
7	dr. Ida Bagus Eka Wija Utama, SpA	Ilmu Kes. Anak	4	-	-	4	-	-	-	-	-	-	4
8	dr. Gregorius Sepatayuda, SpRad	Radiologi	8	-	-	4	-	-	-	4	-	-	8
9	Prof. Dr. dr. Retno Wahyuningsih, MS, SpParK	Parasitologi	8	-	-	4	4	-	-	-	-	-	8
10	dr. Trimurti Parnomo, MS, SpMK	Mikrobiologi	4	-	-	-	4	-	-	-	-	-	4
11	Dra. Lusia Sri Sunarti, MS	Mikrobiologi	4	-	-	-	4	-	-	-	-	-	4
12	dr. Erica Gilda Simanjuntak, SpAn	Anestesi	4	-	-	-	-	4	-	-	-	-	4
13	dr. Belinda J. L. Latumente, Sp.KFR	Bedah	4	-	-	-	-	-	4	-	-	-	4
14	dr. Danny E. J. Luhulima, SpPK	Pato. Klinik	4	-	-	-	-	-	4	-	-	-	4
15	dr. Keswari Aji Patriawati, M.Sc., SpA	Ilmu Kes. Anak	4	-	-	-	-	-	-	4	-	-	4
16	Dr. Med. dr. Abraham Simatupang, M.Kes.	Farmakologi Terapi	4	-	-	-	-	-	-	-	4	-	4
17	dr. E. S. Diapari Pohan, SpB - M.Kes.	Bedah	4	-	-	-	-	-	-	-	4	-	4
18	dr. Jumaini Andriana Sihombing, M.Pd.Ked.	Anatomi	4	-	-	-	-	-	-	-	4	-	4
19	dr. Erida Manalu, SpPK	Pato. Klinik	4	-	-	-	-	-	-	-	-	4	4
20	Evy Suryani Arodes, M.Biomed., M.Pd.	Mikrobiologi	4	-	-	-	-	-	-	-	-	4	4
T O T A L			104										104
PERSENTASI KEHADIRAN KULIAH PAKAR BLOK 7 & KM			100%										

Jakarta, 21 Oktober 2020

Mengetahui
Manager P2SK,

Dra. Lusia Sri Sunarti, MS

Koordinator Blok 7,

dr. Danny E. J. Luhulima, SpPK

Universitas Kristen Indonesia
Fakultas Kedokteran

SURAT KEPUTUSAN
No. : 032/UKI.F5.D/HKP.3.5.6/2020
tentang

PENUGASAN TENAGA AKADEMIK DALAM MEMBERIKAN KULIAH PAKAR PIMPINAN FAKULTAS KEDOKTERAN UNIVERSITAS KRISTEN INDONESIA

MENIMBANG : Bahwa untuk kelancaran proses belajar mengajar dan meningkatkan mutu pendidikan di FKUKI diperlukan penugasan tenaga akademik FKUKI untuk memberikan Kuliah Pakar

MENGINGAT :
1. Peraturan Pemerintah No. 60 tahun 1999 tentang Pendidikan Tinggi
2. Surat Keputusan Dekan FKUKI No. 53/SK/FKUKI/11.2006 tanggal 21 November 2006 tentang Pemberlakuan Kurikulum Berbasis Kompetensi (KBK) di FKUKI
3. Surat Keputusan Rektor UKI No. 90/UKI.R/SK/SDM.8/2018 tentang pengangkatan Dekan Fakultas Kedokteran UKI
4. Surat keputusan pengangkatan sebagai tenaga akademik

MEMUTUSKAN

MENETAPKAN :
1. Penugasan dalam memberikan Kuliah Pakar :

Nama	dr. Fajar Lamhot Gultom, Sp.PA
Departemen	Patologi Anatomi
Blok	7 (Sistem Pernapasan)
Judul Materi	Gambaran Patologi Anatomi Kelainan Sistem Pernapasan
Semester	gasal 2020/2021
Kelas	A : 0,21 SKS
	B : 0,21 SKS
SKS	0,42 SKS

2. Apabila dikemudian hari ternyata terdapat kekeliruan dalam Surat Keputusan ini akan diperbaiki sebagaimana mestinya

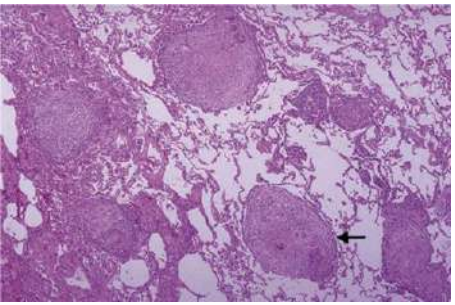
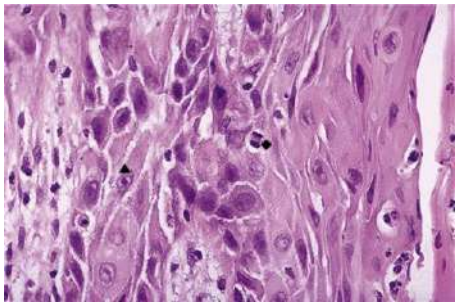
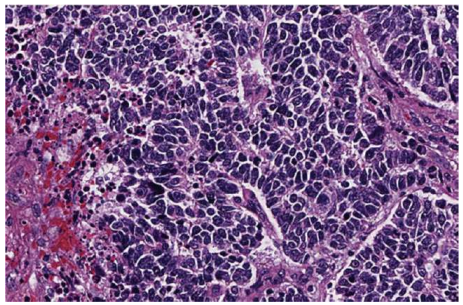
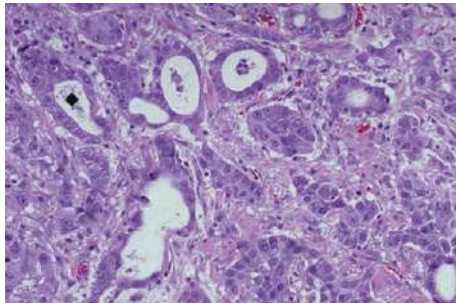
Asli Surat Keputusan ini disampaikan kepada yang bersangkutan untuk diketahui

Ditetapkan di : Jakarta
Pada tanggal : 10 September 2020
Dekan,

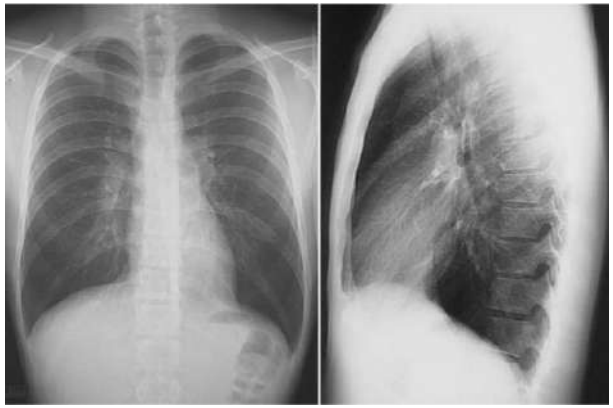
Dr. dr. Robert Hotman Sirait, Sp.An.
NIP. UKI. 031 545

Tembusan:
1. Rektor UKI
2. Wakil Dekan Bidang Akademik FKUKI

Jl. Mayjen Sutoyo no.2 Cawang - Jakarta 13630 INDONESIA Tel. 021.29362033,Tel. Langsung 021.29362038, Faks. 021.29362036 E-mail: fk-uki@uki.ac.id http://www.uki.ac.id

Respiratory Tract Pathology

Fajar L. Gultom
Departemen Patologi Anatomik
FK UKI
September 2020

4 SISTEM RESPIRASI

Paru		
18	Asma bronkial	4A
35	Atelektasis	2
36	Penyakit Paru Obstruksi Kronik (PPOK) eksaserbasi akut	3B
37	Edema paru	3B
38	Infark paru	1
39	Abses paru	3A
40	Emboli par	1
41	Kistik fibrosis	1
42	*Haematothorax*	3B
43	Tumor mediastinum	2
44	Pnemokoniasis	2
45	Penyakit paru intersisial	1
46	*Obstructive Sleep Apnea* (OSA)	1
32	Efusi pleura	2
33	Efusi pleura masif	3B
34	Emfisema paru	3A
16	Aspirasi	3B
17	Benda asing	2

COVID-19??

SKDI 2012

Tingkat Kemampuan 1: mengenali dan menjelaskan

Lulusan dokter mampu mengenali dan menjelaskan gambaran klinik penyakit, dan mengetahui cara yang paling tepat untuk mendapatkan informasi lebih lanjut mengenai penyakit tersebut, selanjutnya menentukan rujukan yang paling tepat bagi pasien. Lulusan dokter juga mampu menindaklanjuti sesudah kembali dari rujukan.

Tingkat Kemampuan 2: mendiagnosis dan merujuk

Lulusan dokter mampu membuat diagnosis klinik terhadap penyakit tersebut dan menentukan rujukan yang paling tepat bagi penanganan pasien selanjutnya. Lulusan dokter juga mampu menindaklanjuti sesudah kembali dari rujukan.

SKDI 2012

Tingkat Kemampuan 3: mendiagnosis, melakukan penatalaksanaan awal, dan merujuk

3A. Bukan gawat darurat

Lulusan dokter mampu membuat diagnosis klinik dan memberikan terapi pendahuluan pada keadaan yang bukan gawat darurat. Lulusan dokter mampu menentukan rujukan yang paling tepat bagi penanganan pasien selanjutnya. Lulusan dokter juga mampu menindaklanjuti sesudah kembali dari rujukan.

3B. Gawat darurat

Lulusan dokter mampu membuat diagnosis klinik dan memberikan terapi pendahuluan pada keadaan gawat darurat demi menyelamatkan nyawa atau mencegah keparahan dan/atau kecacatan pada pasien. Lulusan dokter mampu menentukan rujukan yang paling tepat bagi penanganan pasien selanjutnya. Lulusan dokter juga mampu menindaklanjuti sesudah kembali dari rujukan.

SKDI 2012

Tingkat Kemampuan 4: mendiagnosis, melakukan penatalaksanaan secara mandiri dan tuntas

Lulusan dokter mampu membuat diagnosis klinik dan melakukan penatalaksanaan penyakit tersebut secara mandiri dan tuntas.

4A. Kompetensi yang dicapai pada saat lulus dokter

4B. Profisiensi (kemahiran) yang dicapai setelah selesai internsip dan/atau Pendidikan Kedokteran Berkelanjutan (PKB)

Lungs

Normal anatomy & histology!!

- Paired intrathoracic organs → lobes
- Right → 3 lobes
- Left → 2 lobes

Figure 5-1 Normal lungs, gross
The external surfaces in radiologic orientation show upper, middle, and lower lobes on the right and upper and lower lobes on the left (right lung at left of *left panel*). In the *right panel* the cross-section of normal right lung shows minimal posterior and inferior congestion. There is minimal anthracotic pigment from dust in the air breathed in, scavenged by pulmonary macrophages, and transferred to pleural lymphatics to make them appear grayish black.

Robbins and Cotrans Atlas of Pathology 3rd ed, 2015

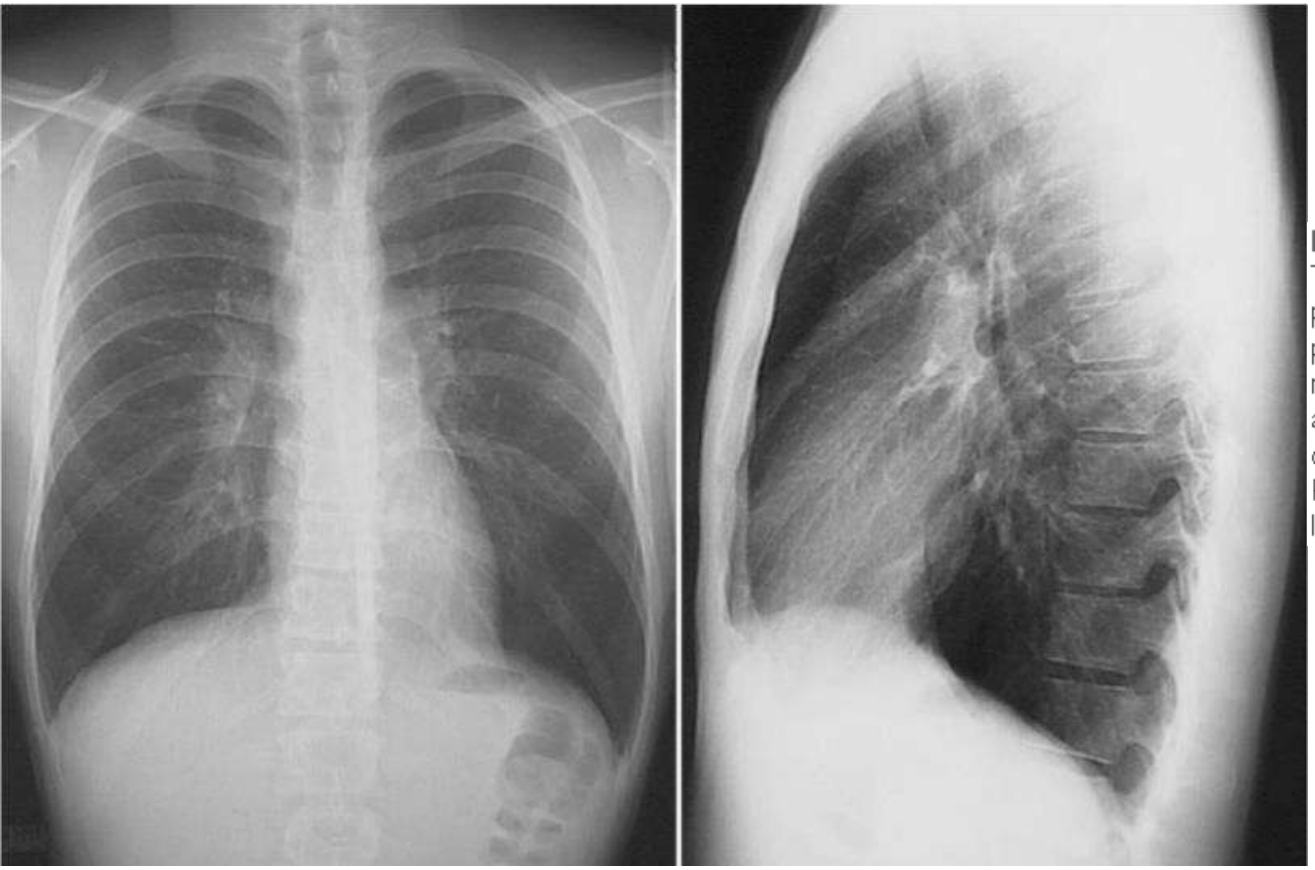


Figure 5-2 Normal lungs, radiographs
These chest radiographs reveal the normal posterior-anterior (PA) *(left)* and lateral *(right)* projection appearance of the lungs in a normal man. The darker air density represents the aerated lung parenchyma, with soft tissue and bone of the chest wall and hilum brighter. The normal PA heart shadow is about the width of the left lung.

Robbins and Cotrans Atlas of Pathology 3rd ed, 2015

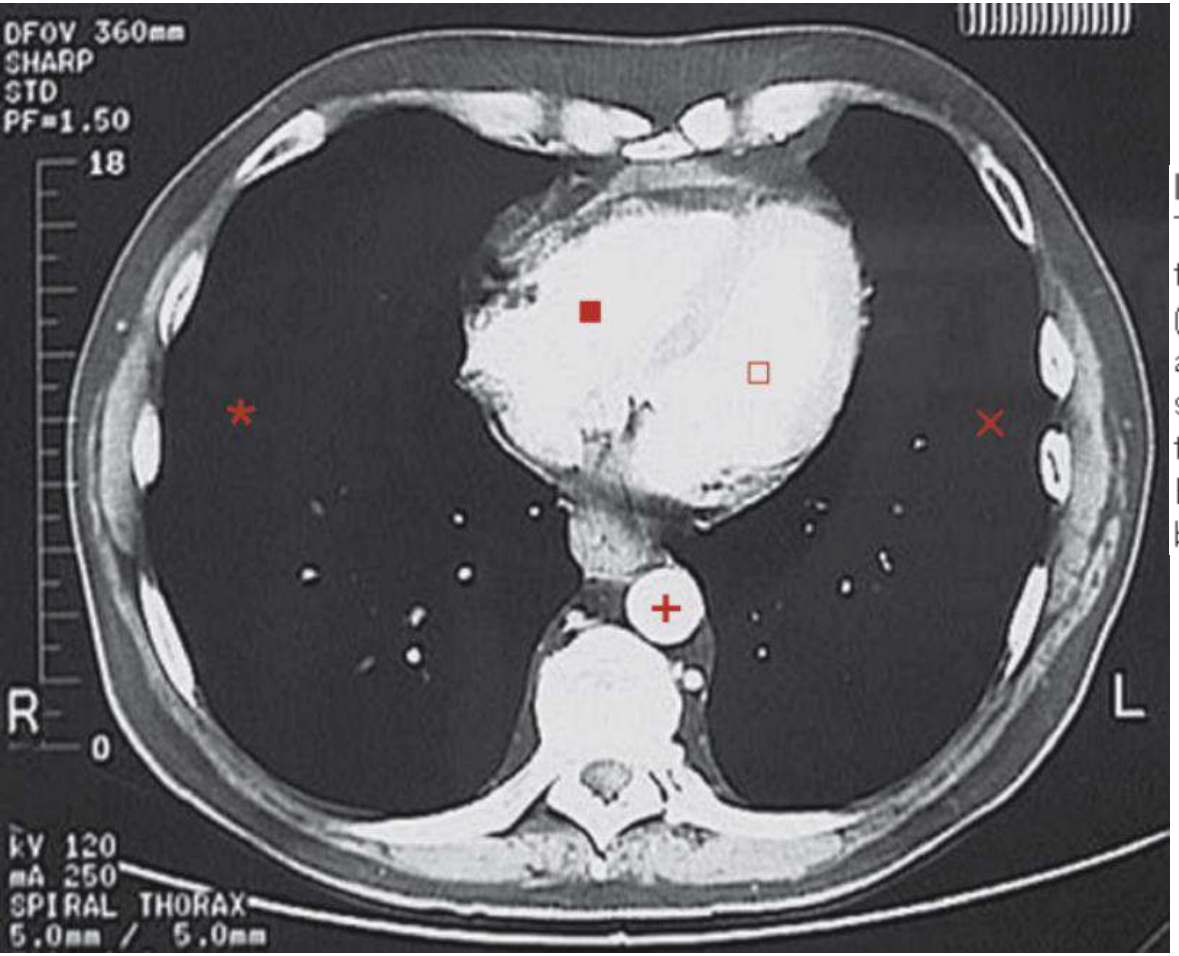


Figure 5-4 Normal lung, CT image
This chest CT scan at soft-tissue density reveals the normal appearance of the right (✱) and left (✕) lungs—essentially black from air density—in a normal man. Contrast material in the bloodstream gives the right (■) and left (□) chambers of the heart and the aorta (✚) a bright appearance. Bone of the vertebral body and ribs also appears bright. The AP diameter is normal.

Robbins and Cotrans Atlas of Pathology 3rd ed, 2015

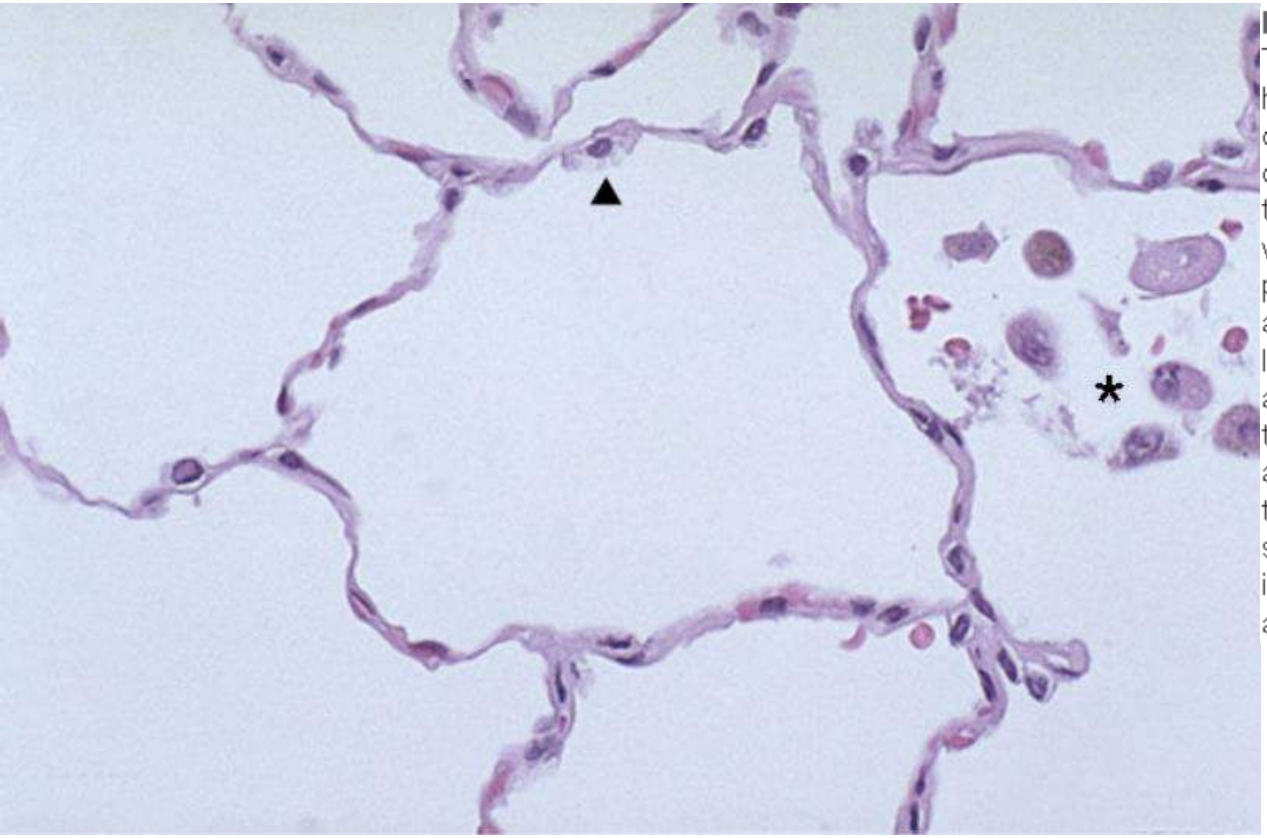
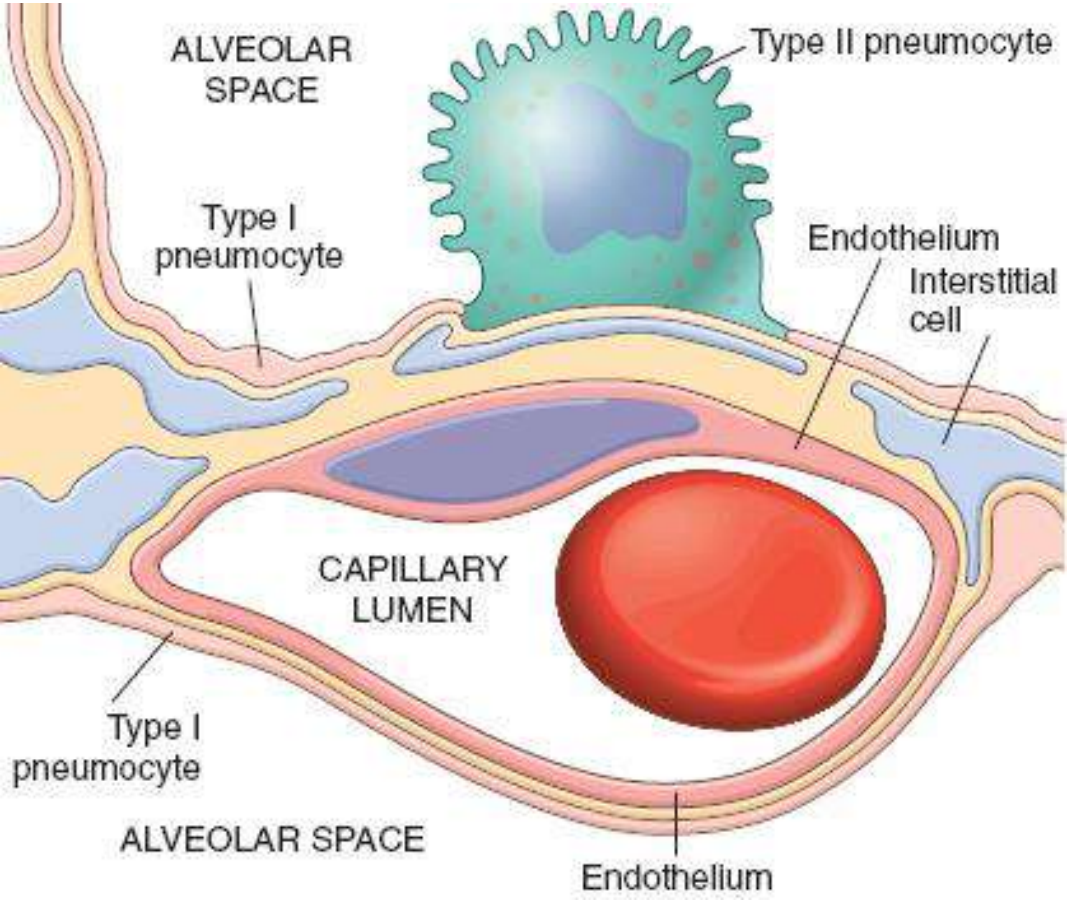


Figure 5-5 Normal adult lung, microscopic
The delicate alveolar walls of the lung are seen here at high magnification. The attenuated cytoplasm of the alveolar type I epithelial cells cannot easily be distinguished from the endothelial cells of the capillaries that are present within the alveolar walls. These thin alveolar walls provide for efficient gas exchange so that the alveolar-arterial (A-a) oxygen gradient is typically less than 15 mm Hg in young, healthy individuals, although the A-a gradient may increase to greater than 20 mm Hg in elderly individuals. Occasional alveolar macrophages (★) can be found within the alveoli. The type II pneumocytes (▲) produce surfactant that reduces surface tension to increase lung compliance and help keep the alveoli expanded.

Robbins and Cotrans Atlas of Pathology 3rd ed, 2015



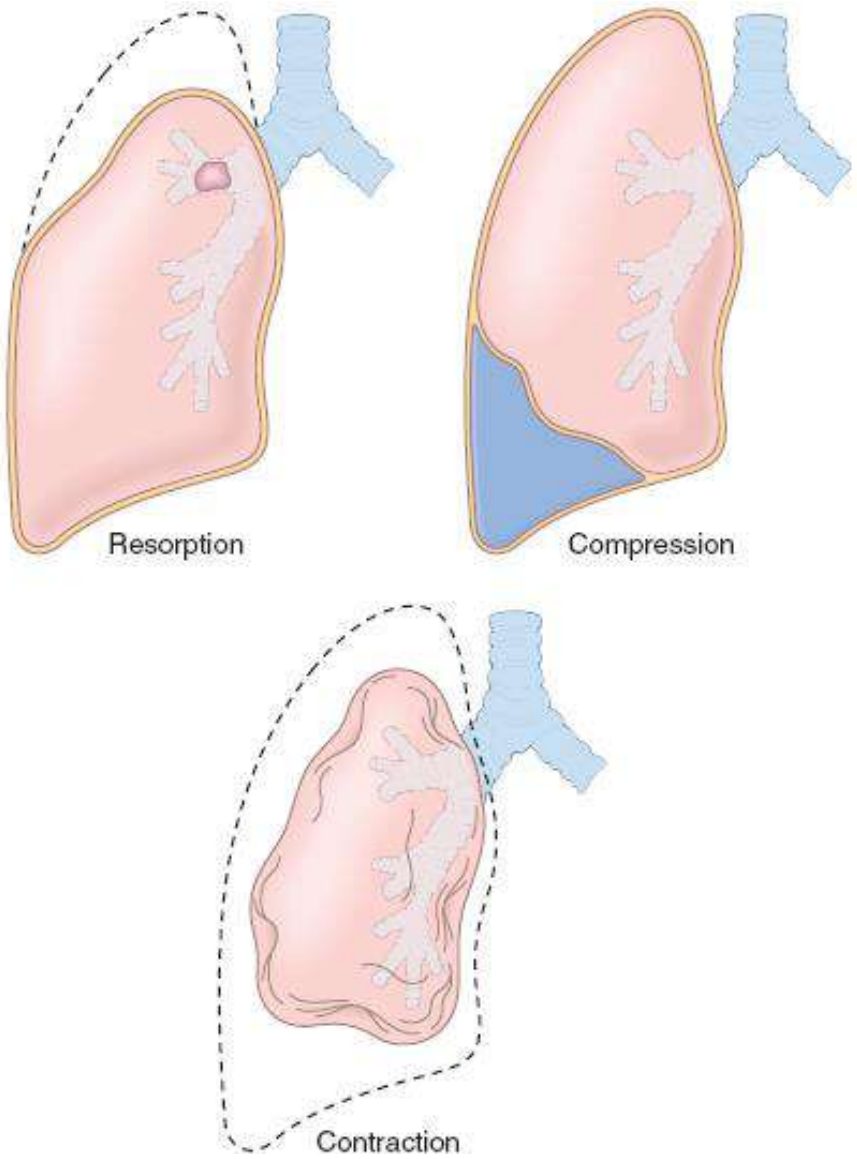
- Endothel
- Interstitium
- Alveolar epithelium:
  - Type I (respiration)
  - Type II (surfactant, stem cell)
- Macrophage alveolar (dust cell)

Robbins Basic Pathology 9th ed, 2015

Primary Lung Disease

- **Airways**
- Interstitium
- Vascular

Atelectasis (Collapse)



- Loss of lung volume → inadequate expansion
- Mechanism:
  - Resorption: obstruction → mucopurulent plug. Bronchial asthma, aspiration, chronic bronchitis
  - Compression: accumulation fluid, blood, air → pleural cavity.
  - Contraction/ cicatrization: local/ generalized fibrotic

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Acute Lung Injury (ALI) – Acute Respiratory Distress Syndrome (ARDS)

- Abrupt onset, significant hypoxemia
- Bilateral pulmonary infiltrate
- Epithelial/ pneumocytes – endothelial injury
- Severe ALI → ARDS (acute respiratory distress syndrome)
- Histopathology: Diffuse alveolar damage (DAD)

Table 12–1 Clinical Disorders Associated with the Development of Acute Lung Injury/Acute Respiratory Distress Syndrome

Direct Lung Injury	Indirect Lung Injury
Common Causes	
Pneumonia	Sepsis
Aspiration of gastric contents	Severe trauma with shock
Uncommon Causes	
Pulmonary contusion	Cardiopulmonary bypass
Fat embolism	Acute pancreatitis
Near-drowning	Drug overdose
Inhalational injury	Transfusion of blood products
Reperfusion injury after lung transplantation	Uremia

Modified from Ware LB, Matthay MA: The acute respiratory distress syndrome. N Engl J Med 342:1334, 2000.

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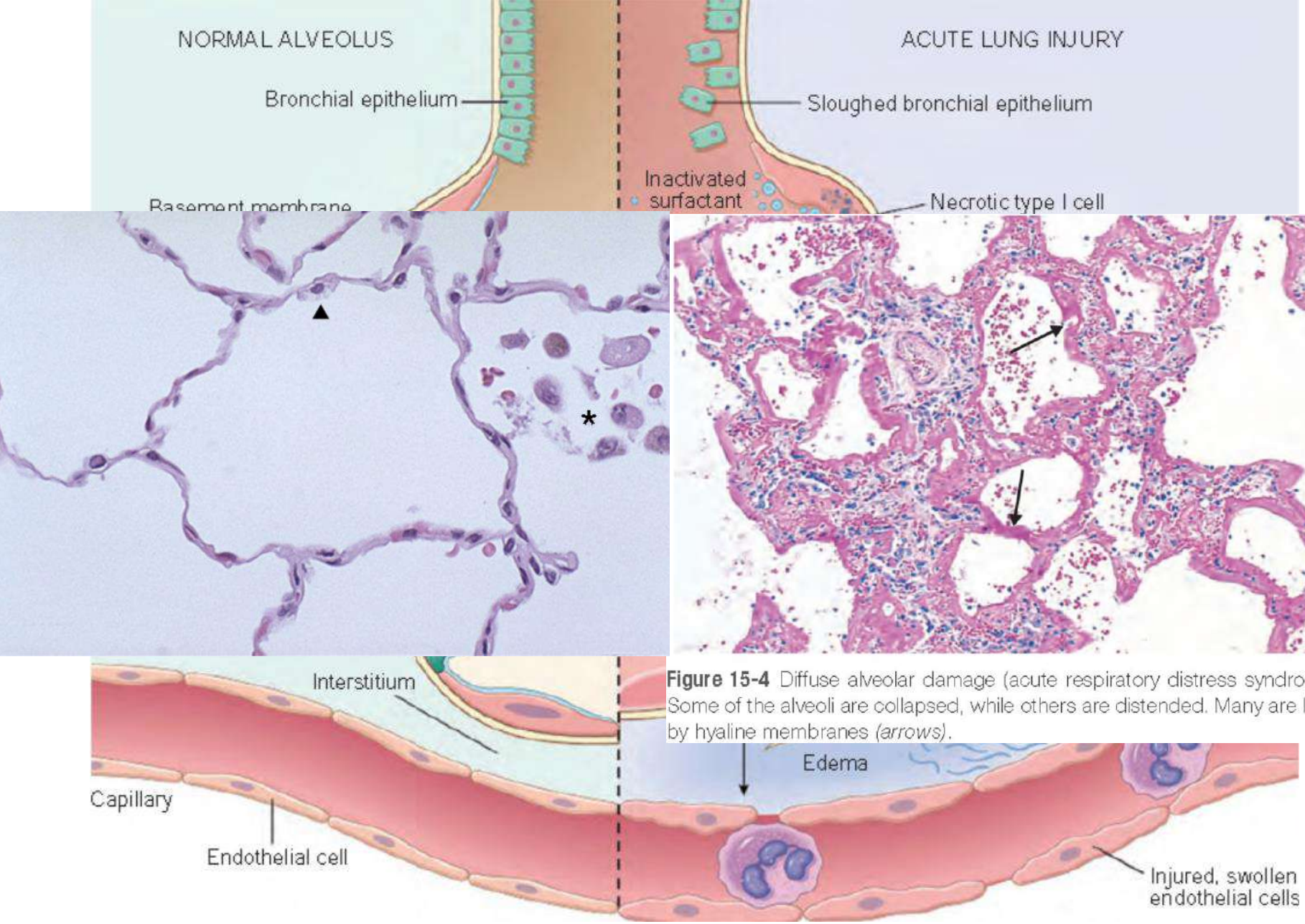

Figure 15-4 Diffuse alveolar damage (acute respiratory distress syndrome). Some of the alveoli are collapsed, while others are distended. Many are lined by hyaline membranes *(arrows)*.

Figure 15-3 The normal alveolus *(left side)* compared with the injured alveolus in the early phase of acute lung injury and acute respiratory distress syndrome. (Modified with permission from Matthay MA, Ware LB, Zimmerman GA: The acute respiratory distress syndrome. J Clin Invest 122:2731, 2012.) *IL-1,* interleukin-1; *MIF,* migration inhibitory factor; *PAF,* platelet activating factor; *TNF,* tumor necrosis factor.

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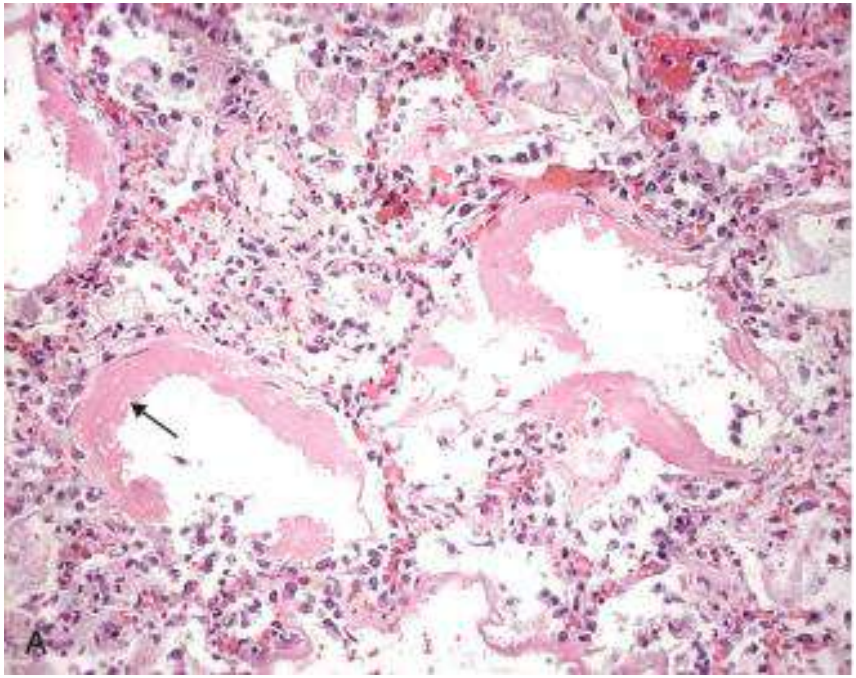

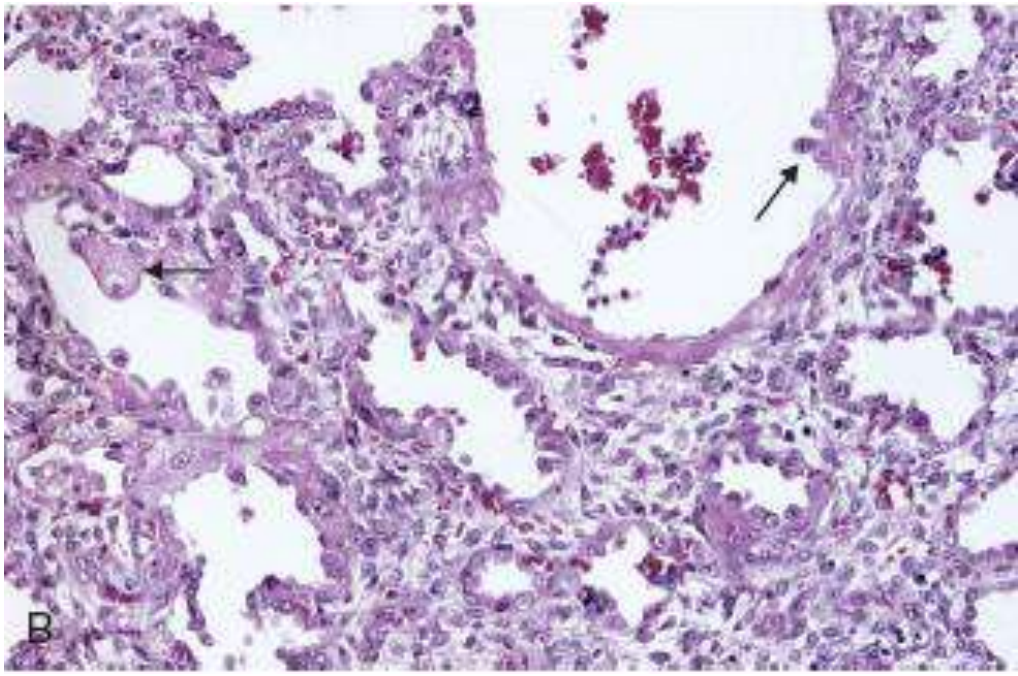

- Left: ALI & ARDS – pink hyaline membrane
- Right: healing stage – type II pneumocyte

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Obstructive??
VS
Restrictive??

Obstructive lung disease

- **Emphysema → COPD**
- **Chronic bronchitis → COPD**
- Asthma
- Bronchiectasis

Table 15-3 Disorders Associated with Airflow Obstruction: The Spectrum of Chronic Obstructive Pulmonary Disease

Clinical Term	Anatomic Site	Major Pathologic Changes	Etiology	Signs/Symptoms
Chronic bronchitis	Bronchus	Mucous gland hyperplasia, hypersecretion	Tobacco smoke, air pollutants	Cough, sputum production
Bronchiectasis	Bronchus	Airway dilation and scarring	Persistent or severe infections	Cough, purulent sputum, fever
Asthma	Bronchus	Smooth muscle hyperplasia, excess mucus, inflammation	Immunologic or undefined causes	Episodic wheezing, cough, dyspnea
Emphysema	Acinus	Airspace enlargement; wall destruction	Tobacco smoke	Dyspnea
Small-airway disease, bronchiolitis*	Bronchiole	Inflammatory scarring/obliteration	Tobacco smoke, air pollutants, miscellaneous	Cough, dyspnea

*Can be seen with any form of obstructive lung disease or as an isolated finding.

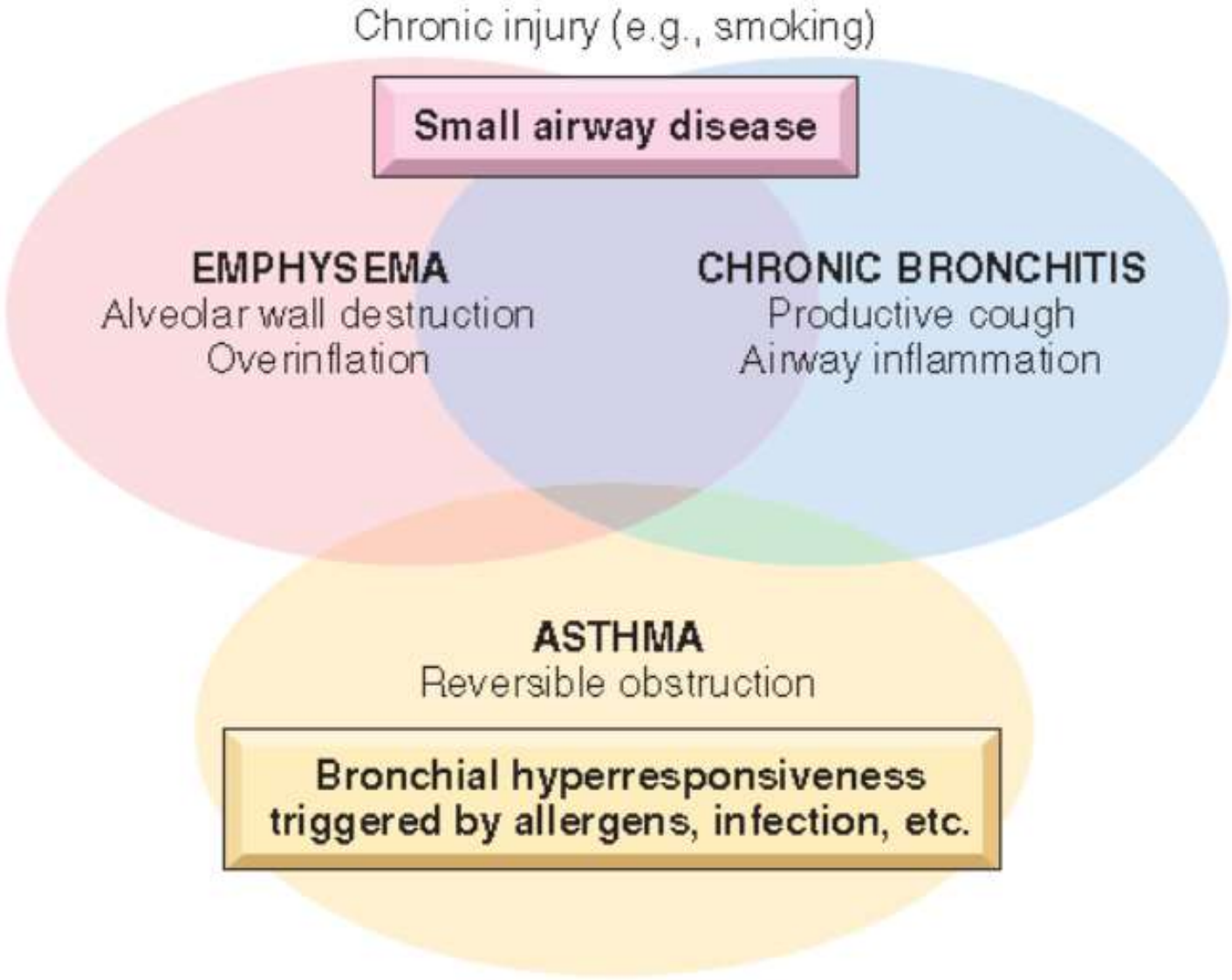

Figure 12–5 Schematic representation of overlap between chronic obstructive lung diseases.

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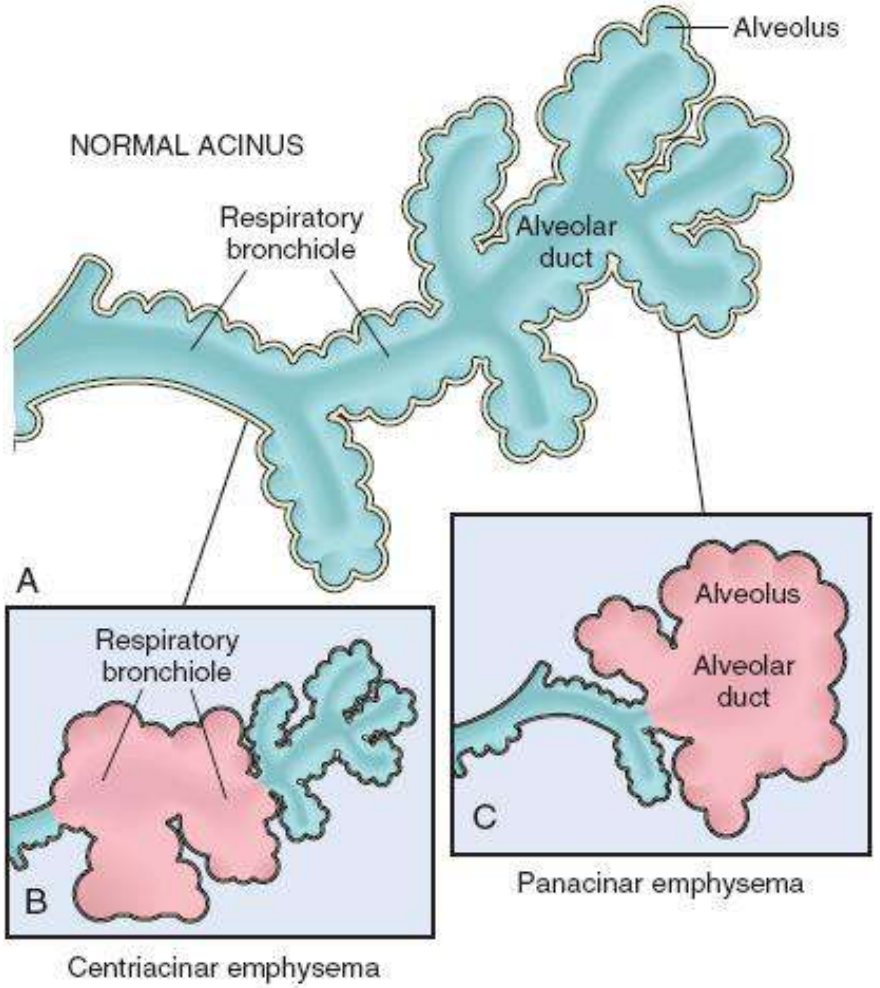
Emphysema

- Irreversible/ permanent enlargement
- Walls destruction, fibrosis (-)
- Distal terminal bronchiole
- Subtypes:
  - **Centriacinar** (smoking related)
  - **Panacinar** (α1-antitrypsin def)
  - Distal acinar
  - Irregular



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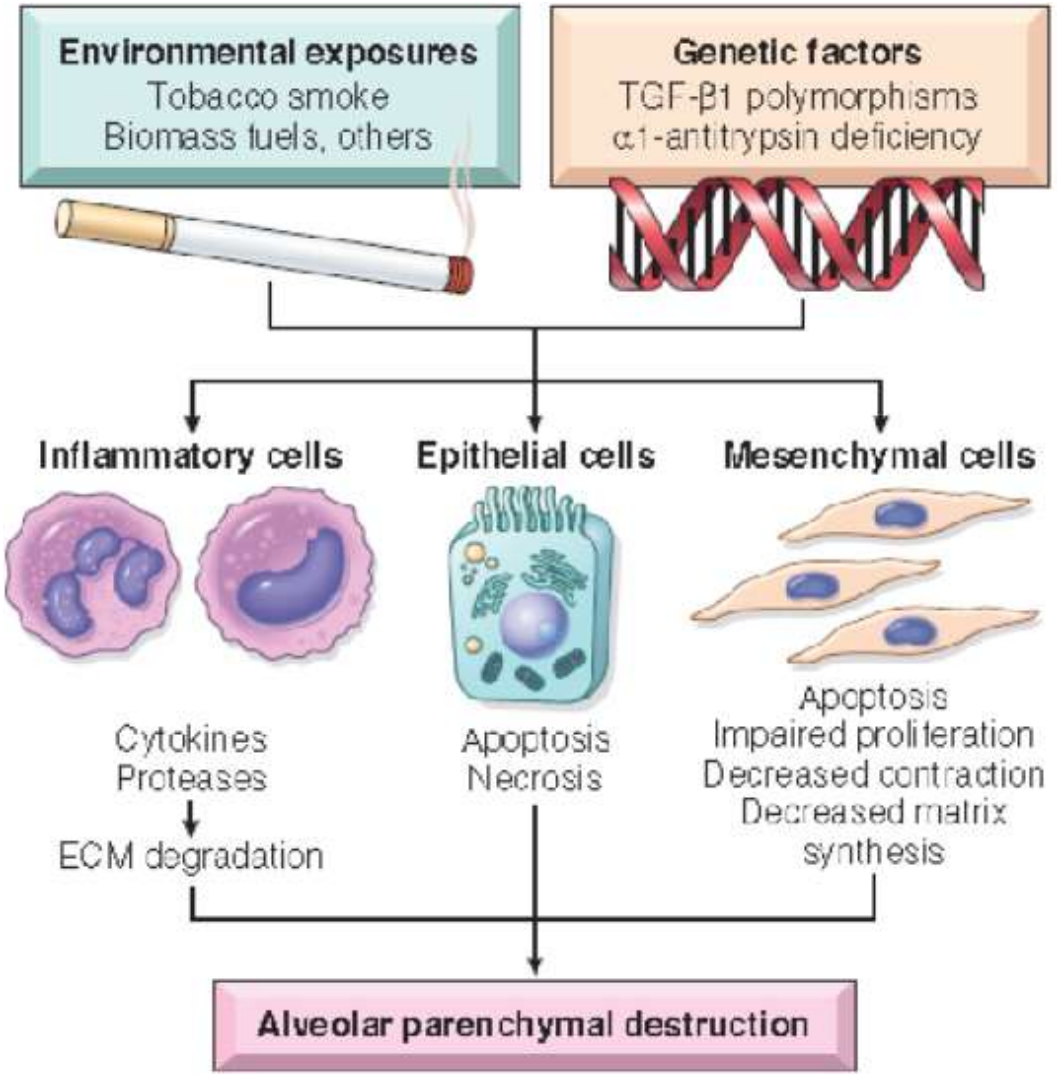

Figure 12–7 Loss of cellular homeostasis in emphysema pathogenesis. Exposure to inhaled toxins (such as cigarette smoke) leads to epithelial cell death, inflammation, and extracellular matrix proteolysis. In susceptible persons, mesenchymal cell survival and reparative functions are impaired by direct effects of inhaled toxic substances and inflammatory mediators and by the loss of the peri- and extracellular matrix. The result is loss of structural cells of the alveolar wall and the associated matrix components.

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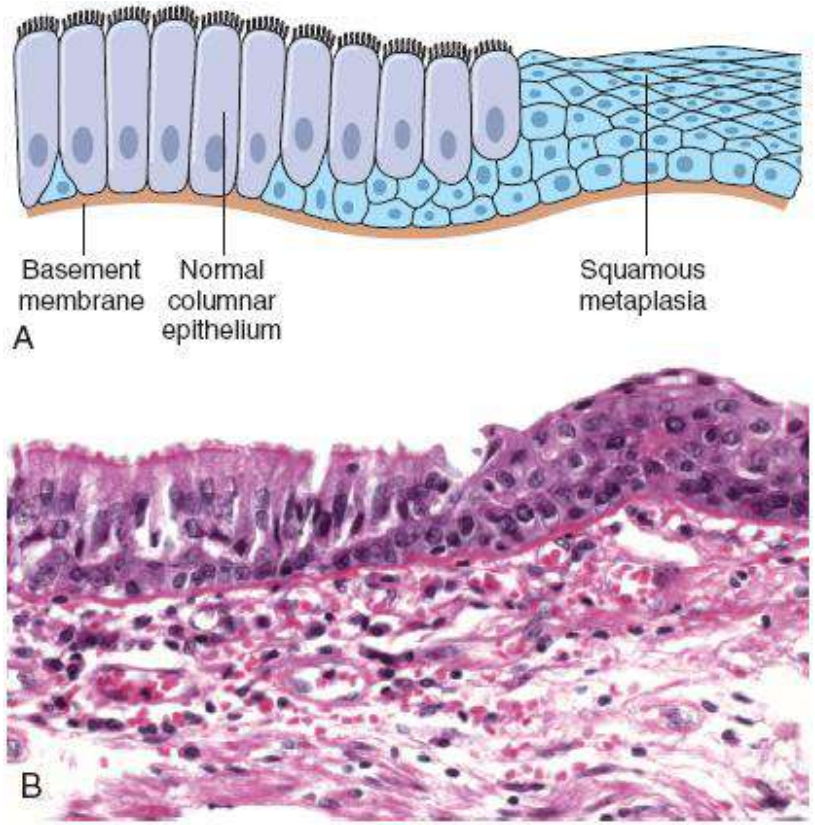
Chronic bronchitis

- **Persistent & Productive** cough **3 months, 2 consecutive years**
- Cigarette smoking, air pollutant



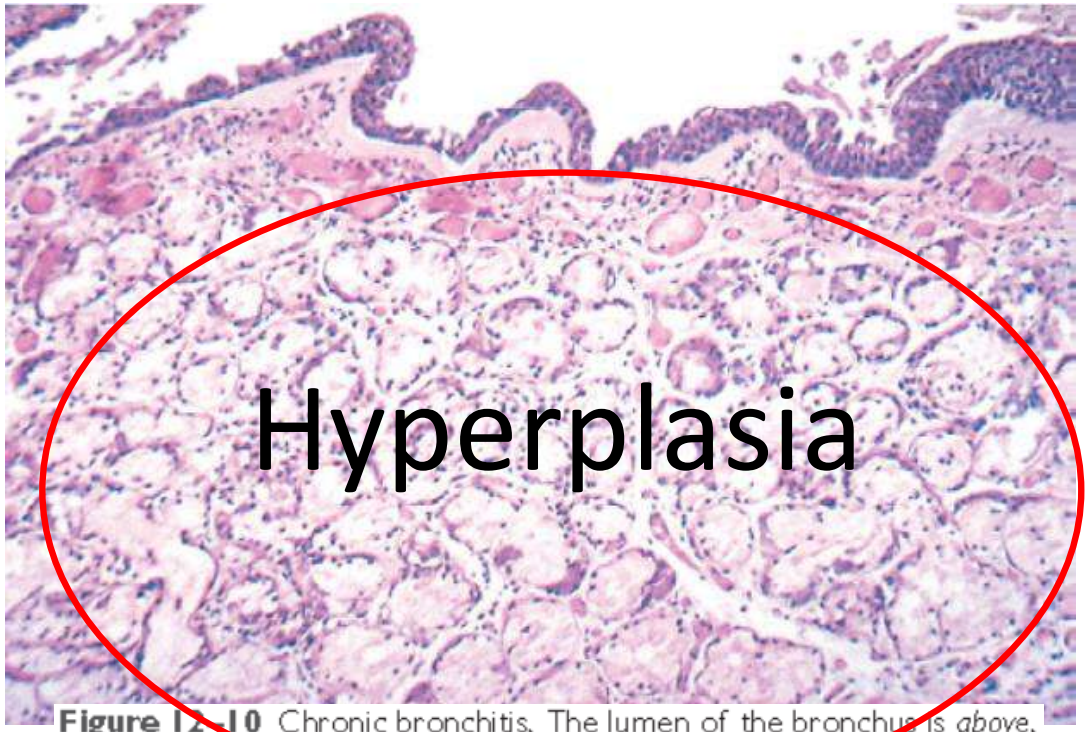


Figure 12-10 Chronic bronchitis. The lumen of the bronchus is *above*. Note the marked thickening of the mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium.

(From the Teaching Collection of the Department of Pathology, University of Texas, Southwestern Medical School, Dallas, Texas.)

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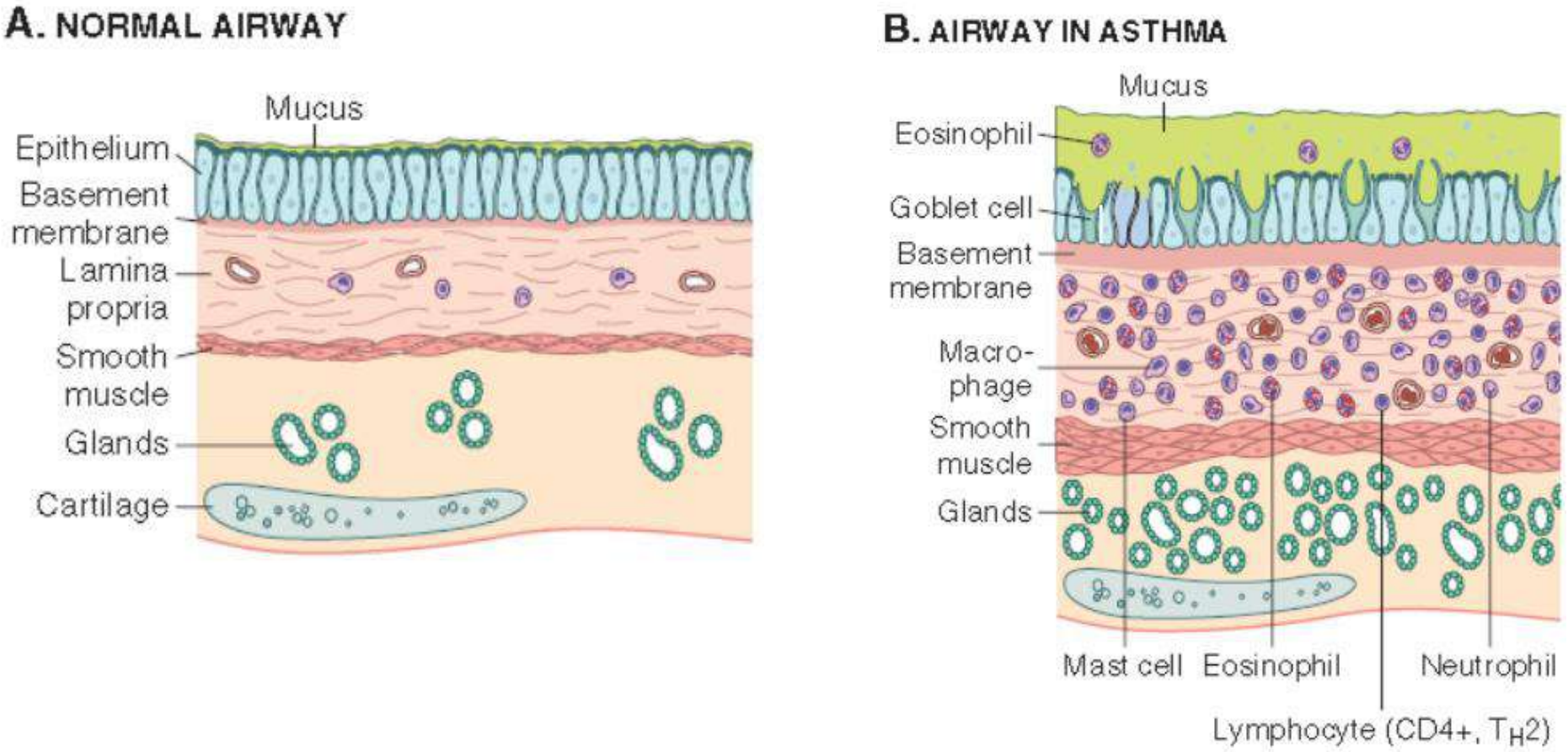
Asthma

Hallmarks:

- Intermitten and reversible airway obstruction
- Chronic inflammation with eosinophils
- Smooth muscle hypertrophy and hyperreactivity
- Increased mucus secretion
- Significant incidence increase in western world → “Hygiene Hypothesis”

Asthma



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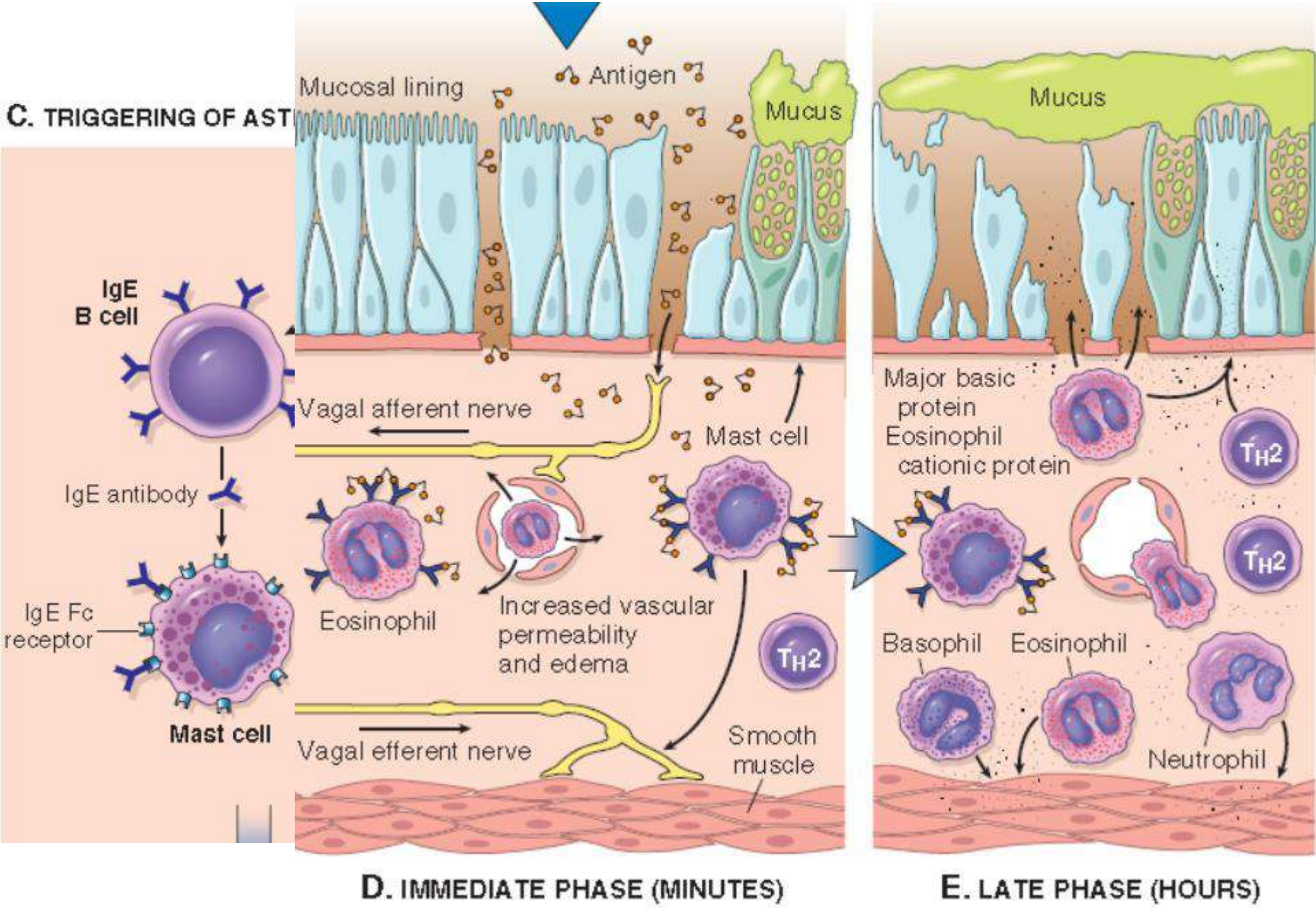

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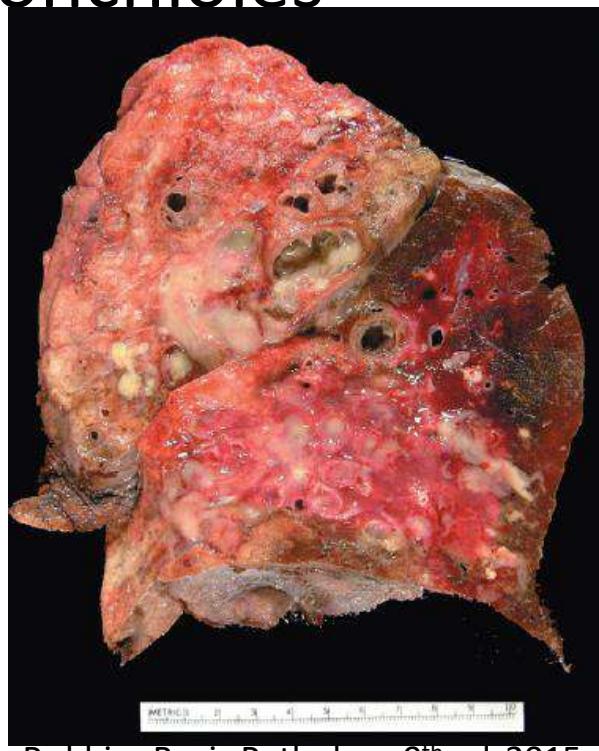
Bronchiectasis

- Obstruction and chronic persistent infection
- Destruction: muscle – elastic tissue
- Permanent dilation: bronchi – bronchioles
- Secondary: post pneumonia, **TB**



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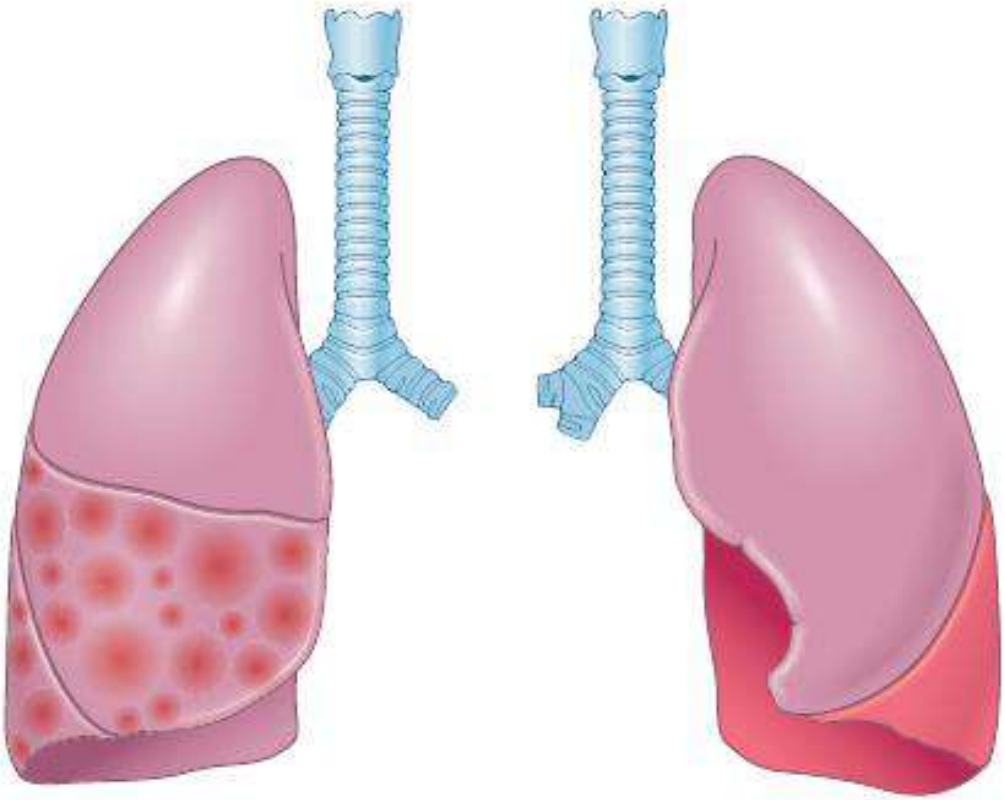
Pulmonary Infection

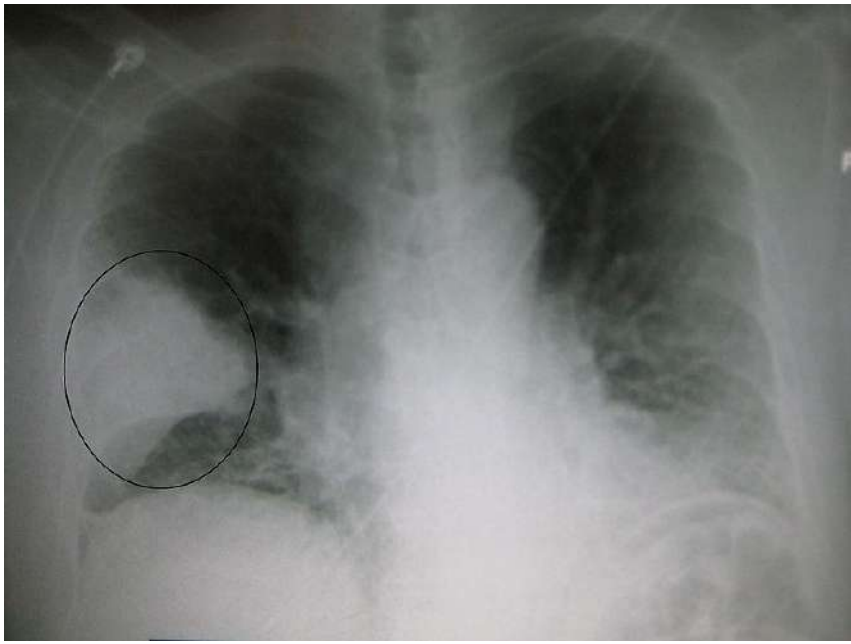
PNEUMONIA

- 1/6 all deaths in US, Indonesia?
- Local defense compromised:
  - Loss/ suppression cough reflex (coma, anesthesia, drug) → aspirated gastric content
  - Injury mucociliary (cigarette smoke, viral disease)
  - Accumulation of secretion (bronchial obstruction)
  - Interference phagocytic/ bactericidal action of alveolar macrophages (alcohol, tobacco smoke)
  - Pulmonary congestion and edema
- Streptococcus pneumonia > 90%
- Bronchopneumonia – lobar pneumonia
- Anatomic n radiologic



Bronchopneumonia

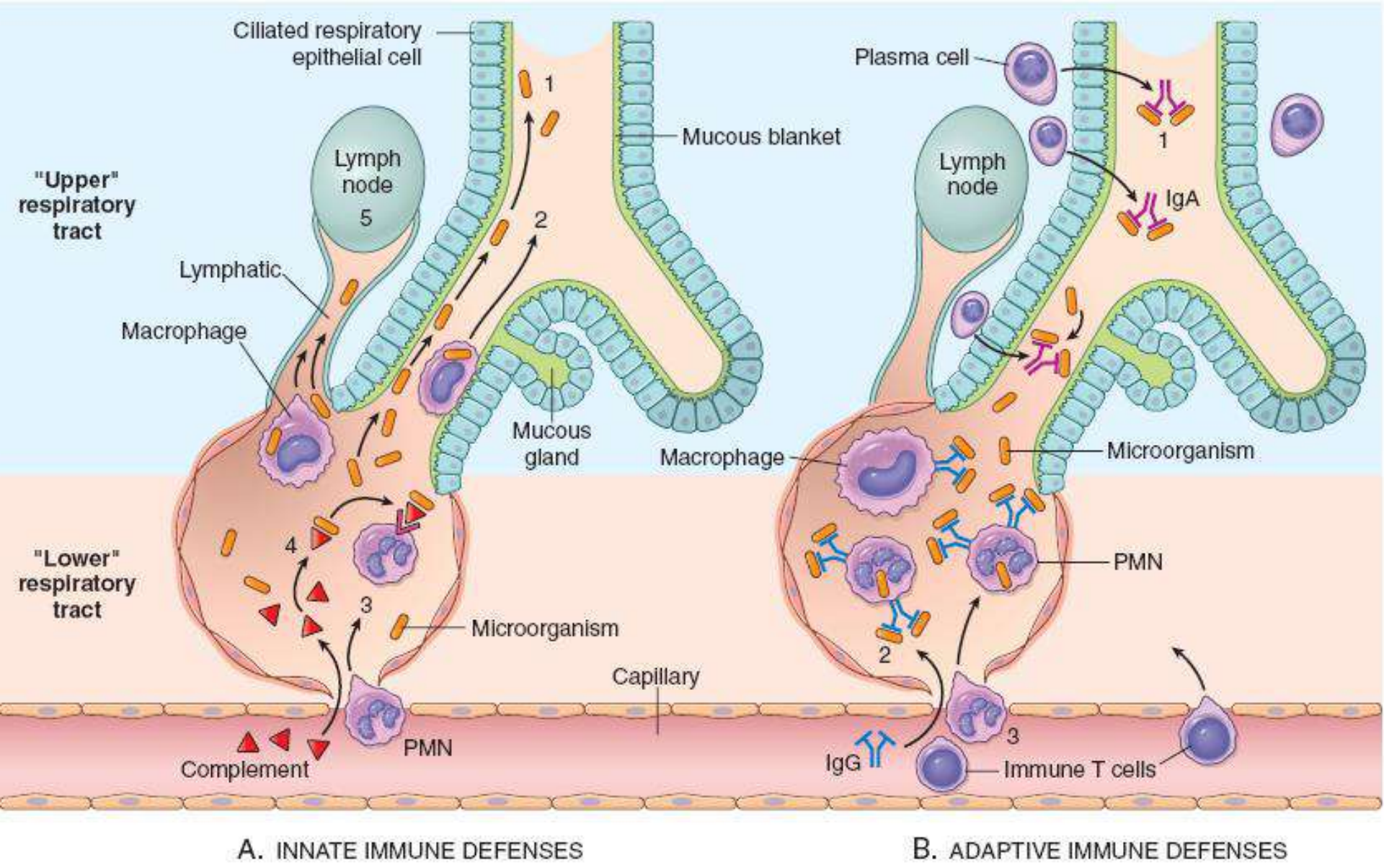
Lobar pneumonia



BLURRY…..

Classify – etiology or clinical settings

Robbins Basic Pathology 9th ed, 2015



Robbins Basic Pathology 9th ed, 2015

CAP - HAP

- Community acquired pneumonia

  S. pneumonia, H. influenzae,….

- Hospital acquired pneumonia

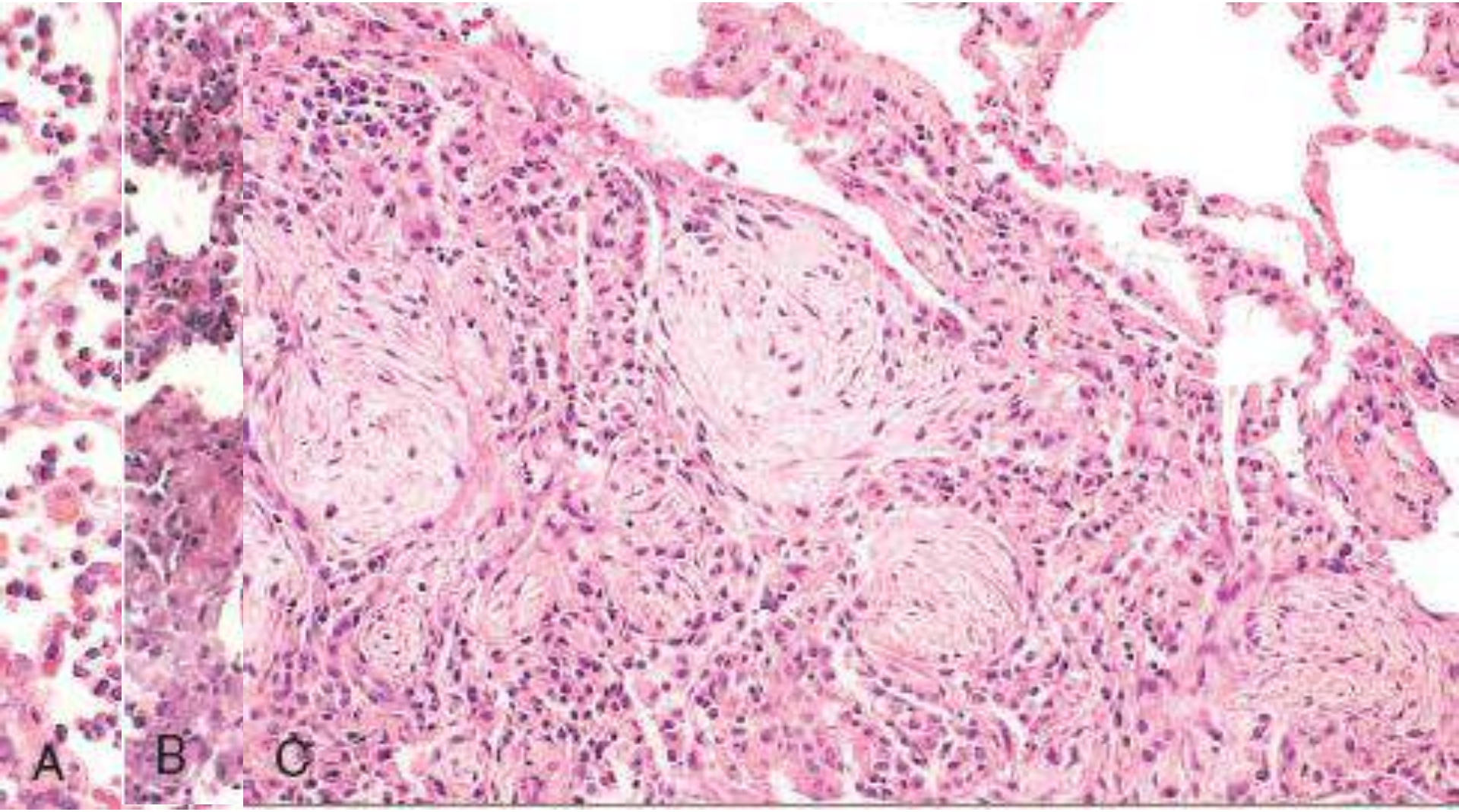
  P. aeruginosa, K. pneumonia,….
- Community acquired atypical pneumonia
- Aspiration pneumonia

CAP - HAP

Era before antibiotics

Morphology: congestion – red hepatization – gray hepatization – resolution

Pneumonia



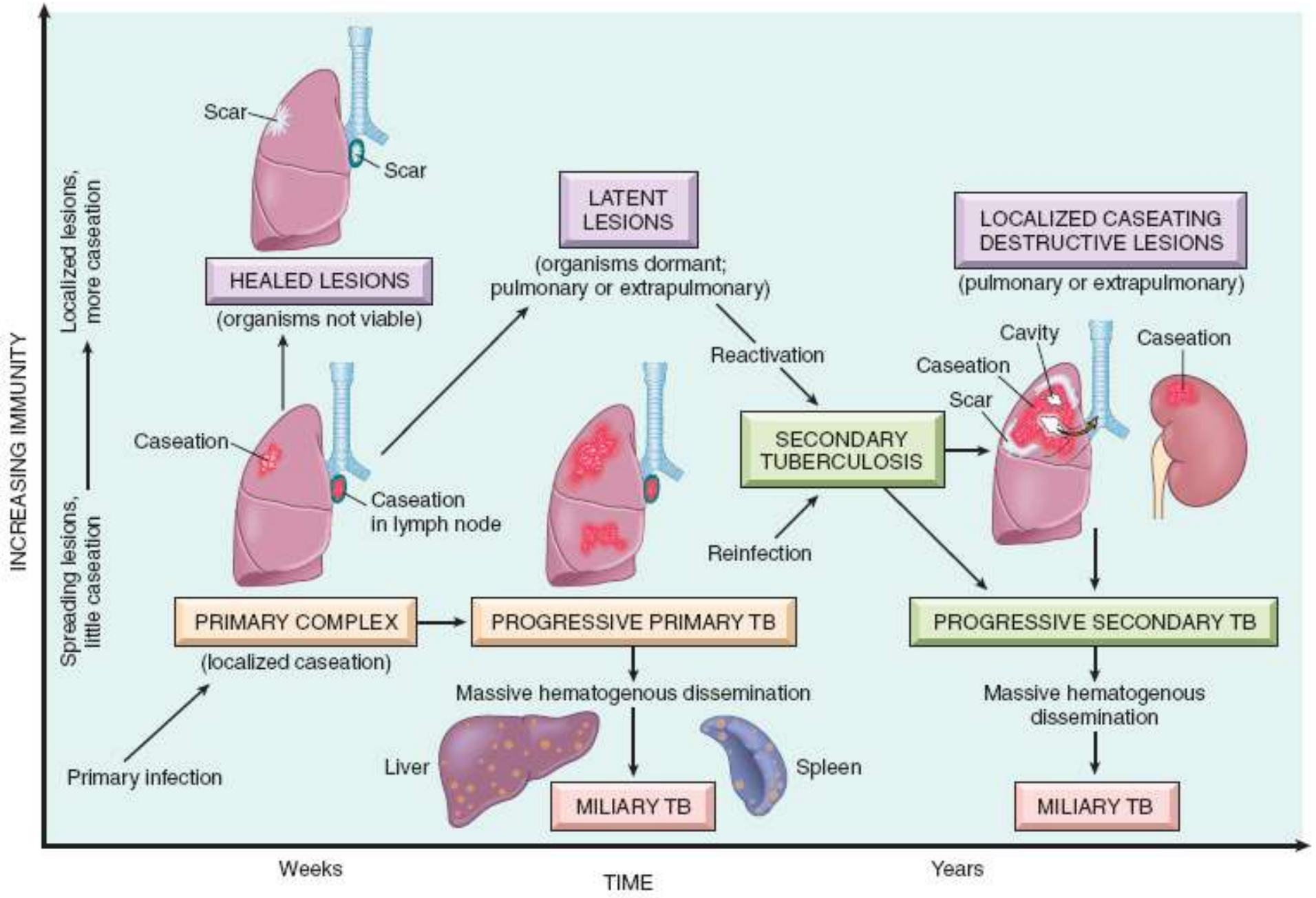
Robbins Basic Pathology 9th ed, 2015

Chronic pneumonia

- Tuberculosis (TB)
- Histoplasmosis
- Blastomycosis
- Coccidioidomycosis

Tuberculosis

- M. Tbc
- Chronic granulomatous disease
- Lung – extrapulmonal



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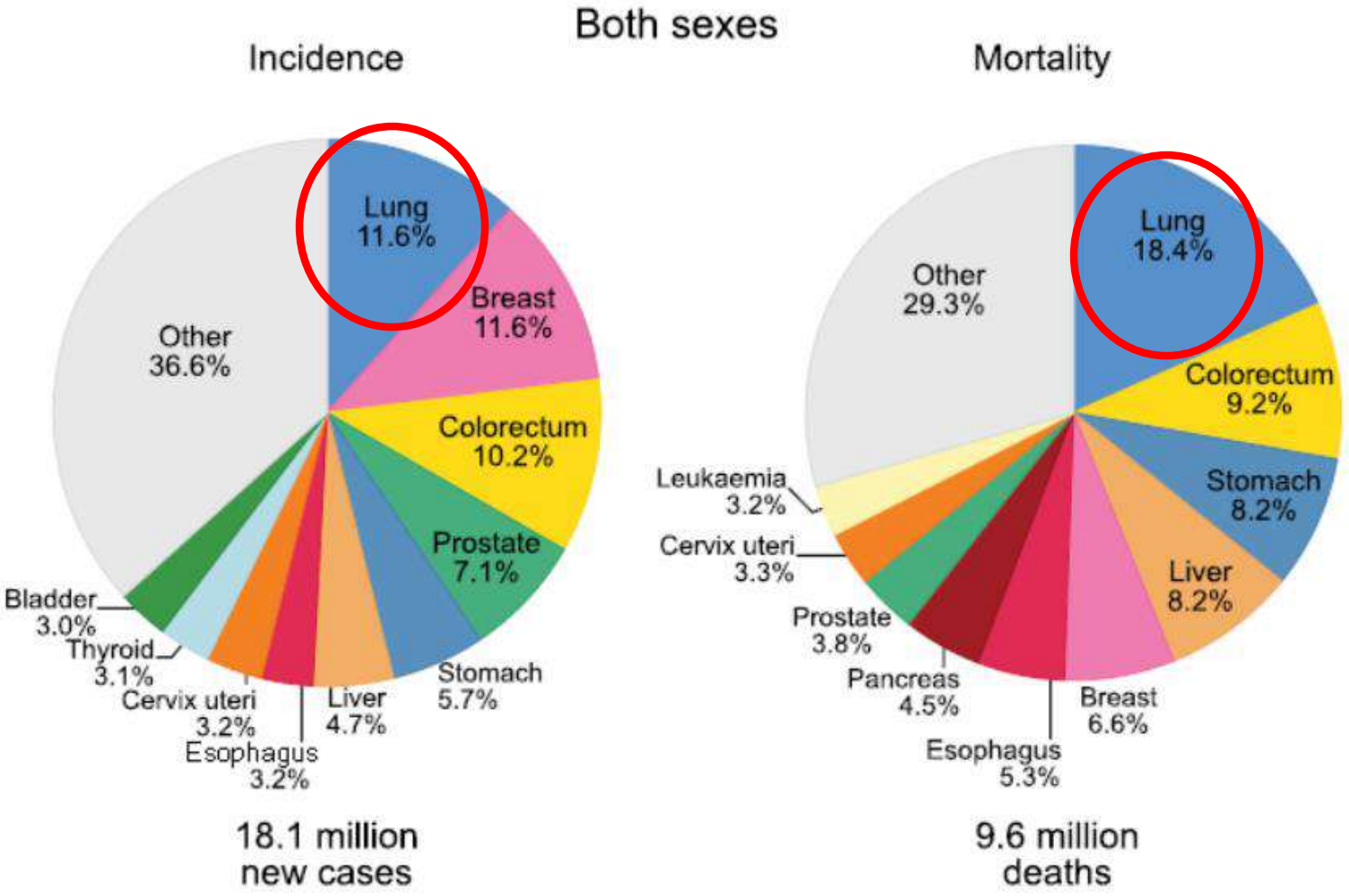
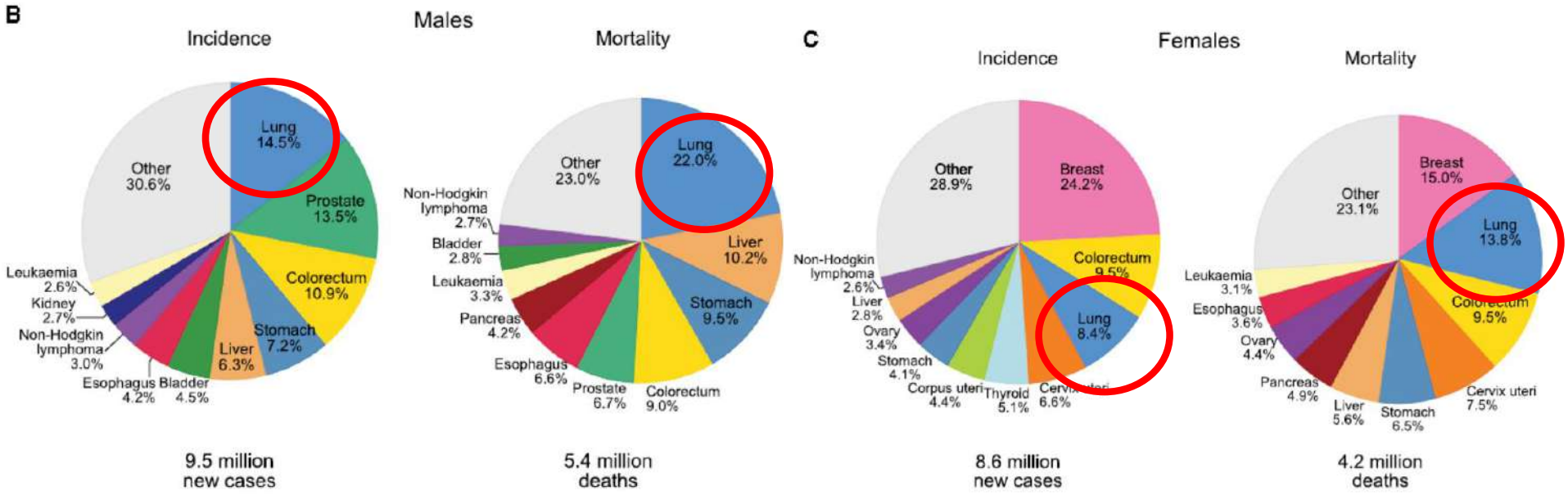
Lung Tumors



Lung cancer: 1st most frequently diagnosed and 1st leading cause of death

GLOBOCAN, 2018

Lung Tumors



GLOBOCAN, 2018

WHO, 2004

Table 1.06
Stage of lung carcinoma at presentation by histologic subtype. SEER data 1983-1987.
Modified, from reference {192}.

Stage	Squamous	Adenocarcinoma	Small cell	Large cell
Localized	21.5%	22.2%	8.2%	15.2%
Regional	38.5%	33.1%	26.1%	31.5%
Distant	25.2%	35.9%	52.8%	40.3%
Unstaged	14.8%	8.8%	12.8%	12.9%

60 – 70%

Poor prognosis…

Lung tumors

- Benign - malignant, epithelial – mesenchymal
- 90-95 % → carcinoma
- 5-10%: carcinoids, lymphoma
- **SMOKING RELATED…TOBACCO EPIDEMIC !!! polycyclic aromatic hydrocarbons, such as benzopyrene, anthracenes, radioactive isotopes**
- EGFR – KRAS mutation → personalized therapy

Lung tumors

TOBACCO EPIDEMIC !!!

Lung cancer is one of the most preventable cancers. Most lung cancers could be avoided by eliminating smoking initiation and increasing smoking cessation among current smokers. This requires a comprehensive tobacco control program that includes raising the price of tobacco products through excise taxes, banning smoking in public places and tobacco sales to minors, restricting tobacco advertising and promotion, counteradvertising, and providing treatment and counseling for tobacco dependence. In the United States, comprehensive tobacco control programs in many states, including California and New York, have markedly decreased smoking rates and accelerated the reduction in

Prior WHO 2004 classification

- Tumors other than small cell carcinoma (SCLC) lumped together → non-small cell carcinoma (NSCLC)
- No theurapetic implication for subtyping
- Light microscopy → only H&E stained

WHO Lung, Thymus and Heart (2015)

Non small cell lung cancer:

- **Adenocarcinoma**
- **Squamous cell carcinoma**
- Large cell neuroendocrine (NE) carcinoma
- Neuroendocrine/ Carcinoid tumor: Typical and Atypical
- Adenosquamous carcinoma
- Pleomorphic carcinoma
- Spindle cell carcinoma
- ……..

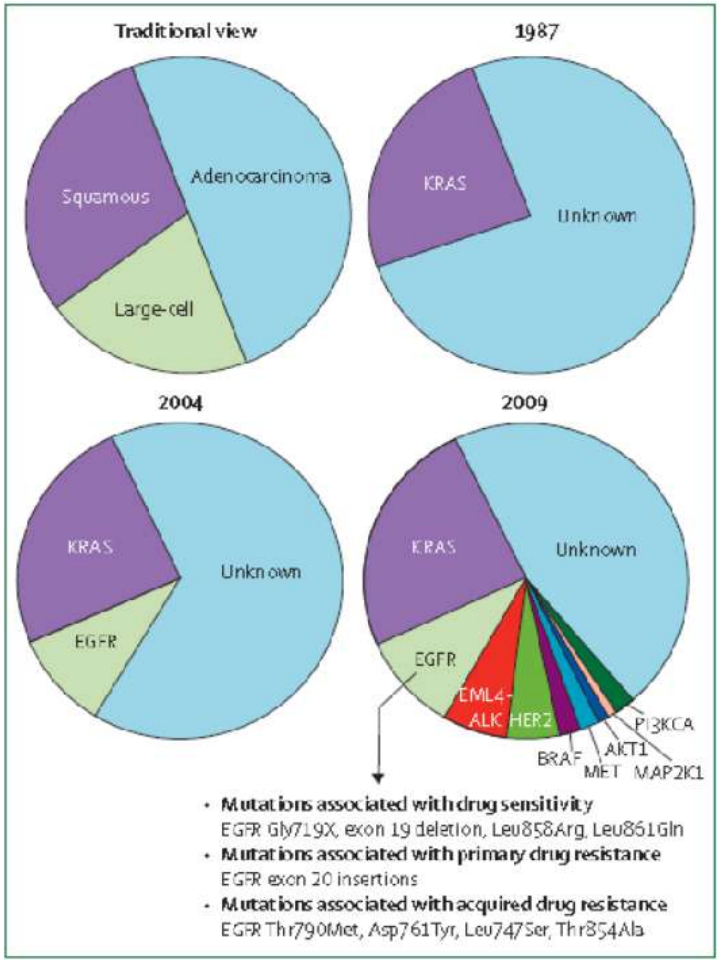
WHO Lung, Thymus and Heart (2015)

- Molecular biology and patient treatment
- EGFR gene mutation in lung adenocarcinoma → targeted therapy (gefitinib, erlotinib) → Significant response
- ALK inhibitor → crizotinib → adenocarcinoma with ALK fusion
- Molecular testing for treatment selection in lung cancer

- Bell DW et al. Epidermal growth factor receptor mutations and gene amplification innsclc. Molecular analysis of the IDEAL/INTACT gefitinib trials. J Clin Oncol. 2005
- Eberhard DA. Mutations in the egfr and in kras are predictive and prognostic indicators in nsclc treated with chemotherapy alone and in combination with erlotinib. J Clin Oncol 2005
- Kwak LE et al. Anaplastic lymphoma kinase inhibition innsclc. NEJM. 2010

Major Genetic Changes in Lung Cancer
"Oncogene Addiction"

Alterations	Small cell carcinoma (%)	Adenocarcinoma (%)	Squamous cell carcinoma (%)
Mutation			
BRAF	0	< 5	0
EGFR Caucasian	< 1	10–20	< 1
Asian	< 5	35–45	< 5
ERBB2/HER2	0	< 5	0
KRAS Caucasian	< 1	15–35	< 5
Asian	< 1	5–10	< 5
PIK3CA	< 5	< 5	5–15
RB	> 90	5–15	5–15
TP53	> 90	30–40	50–80
Amplification			
EGFR	< 1	5–10	10
ERBB2/HER2	< 1	< 5	< 1
MET	< 1	< 5	< 5
MYC	20–30	5–10	5–10
FGFR1	< 1	< 5	15–25
Gene rearrangement			
ALK	0	5	< 1
RET	0	1–2	0
ROS1	0	1–2	0
NTRK1	0	< 1	0
NRG1	0	< 1	0



- Travis et al. WHO Lung, thymus and heart. 2015.
- Pao W. New driver mutations in non-small lung cancer. Lancet Oncol. 2011

Adenocarcinoma

- Def: malignant epithelial tumor with glandular differentiation, mucin production or pneumocyte marker expression.
- Incidence → Lung cancer trends by histologic type
- Changes in design and characteristic of manufactured cigarretes
- Clinical features: variety of symptoms/ no symptoms at all. Progressive shortness of breath, cough, chest pain/ pressure, hoarseness, loss of voice, hemoptysis.
- Localization: **lung periphery**
- Adeno markers: TTF1, Napsin A

- Lewis et al. U.S. Lung cancer trends by histologic type. Cancer.2014

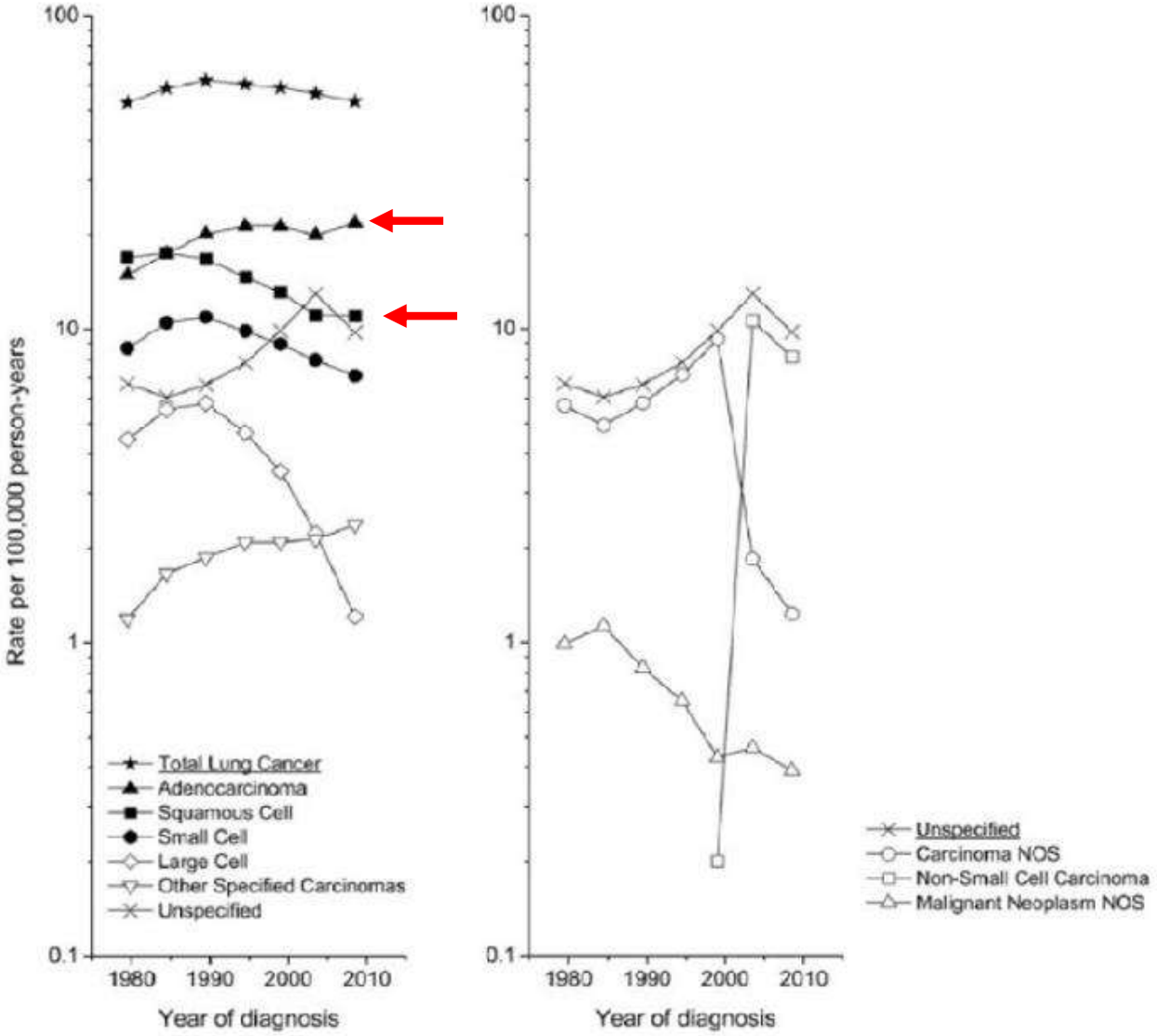
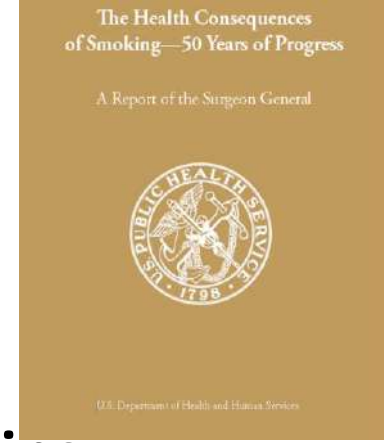


Figure 1.

Trends in lung cancer incidence rates (age-adjusted 2000 US standard) from 1977 to 1981 through 2006 to 2010 in the SEER 9 registries by histologic type.

- Lewis et al. U.S. Lung cancer trends by histologic type. Cancer.2014



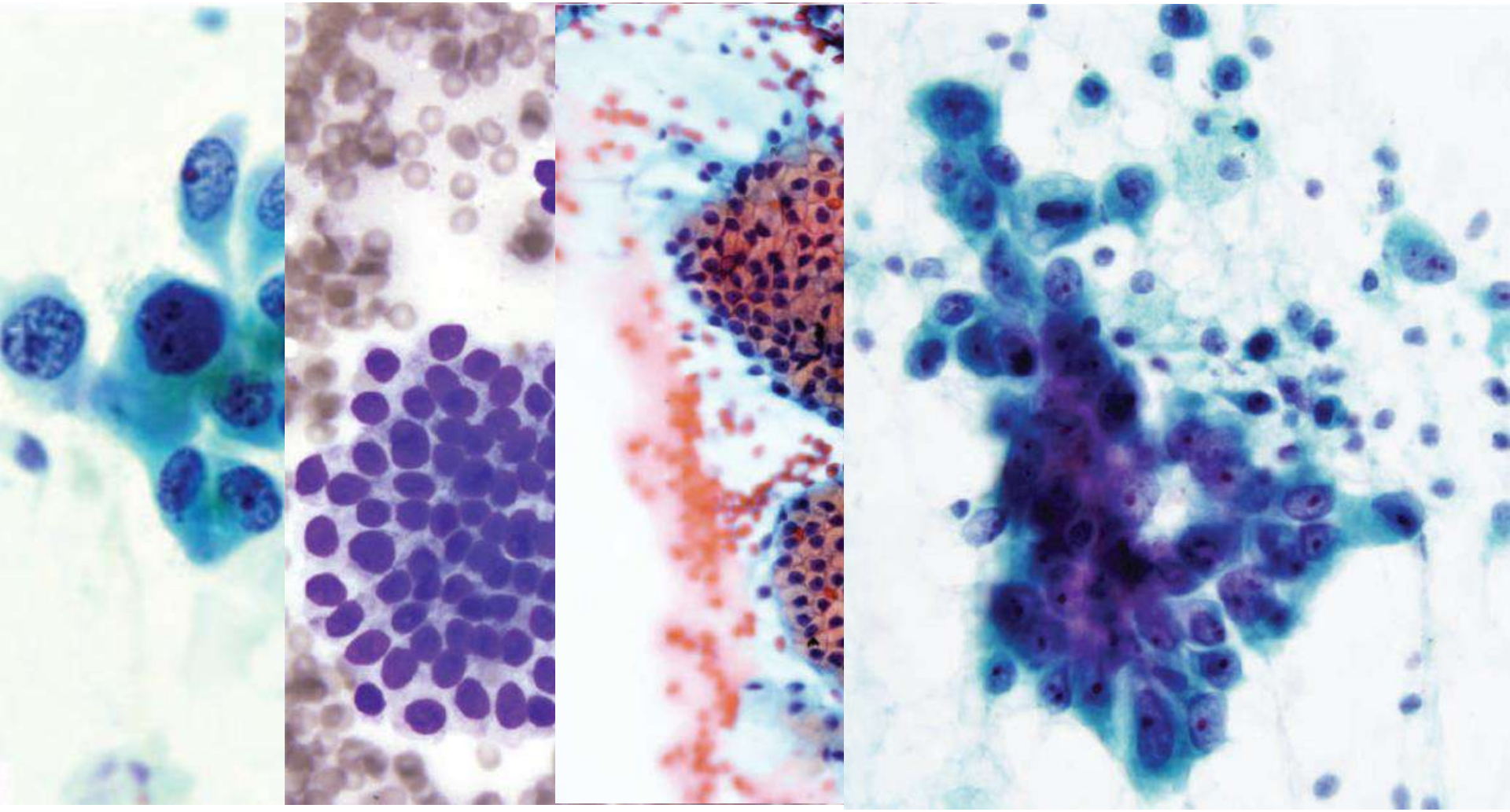
Adenocarcinoma

US Surgeon General's 2014 report:

1. The evidence is sufficient that the **risk of developing adenocarcinoma** of the lung from cigarette smoking has **increased since 1960s**
2. The evidence is sufficient that the **risk of adenocarcinoma in smokers** results from **changes in the design and composition of cigarettes since the 1950s**
3. The evidence is not sufficient to specify which design changes are responsible, **suggestive ventilated filters and increased levels of tobacco specific nitrosamines**
4. The evidence shows that the **decline of squamous cell carcinoma follows the trend of declining smoking prevalence**

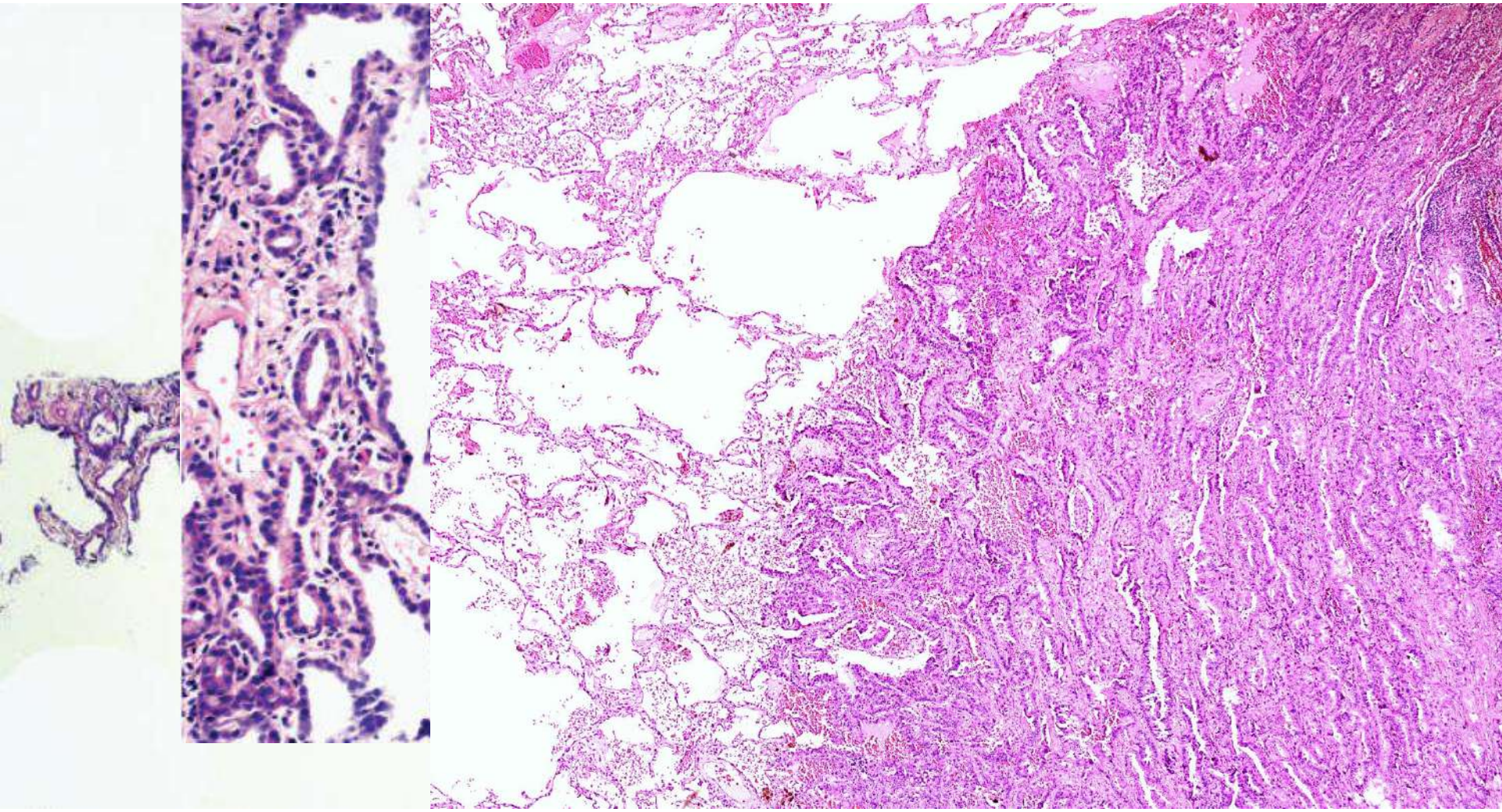
- US public health service office of the surgeon general and national center for chronic disease prevention and health promotion. The health consequences of smoking – 50 years of progress. 2014

Adenocarcinoma Cytology



- Travis WD. Diagnosis of lung cancer in small biopsies and cytology. Implications of the 2011 international association for study of lung cancer/ American thoracic society/ European respiratory society classification. Arch pathol lab med. 2013

Adenocarcinoma Histopatology

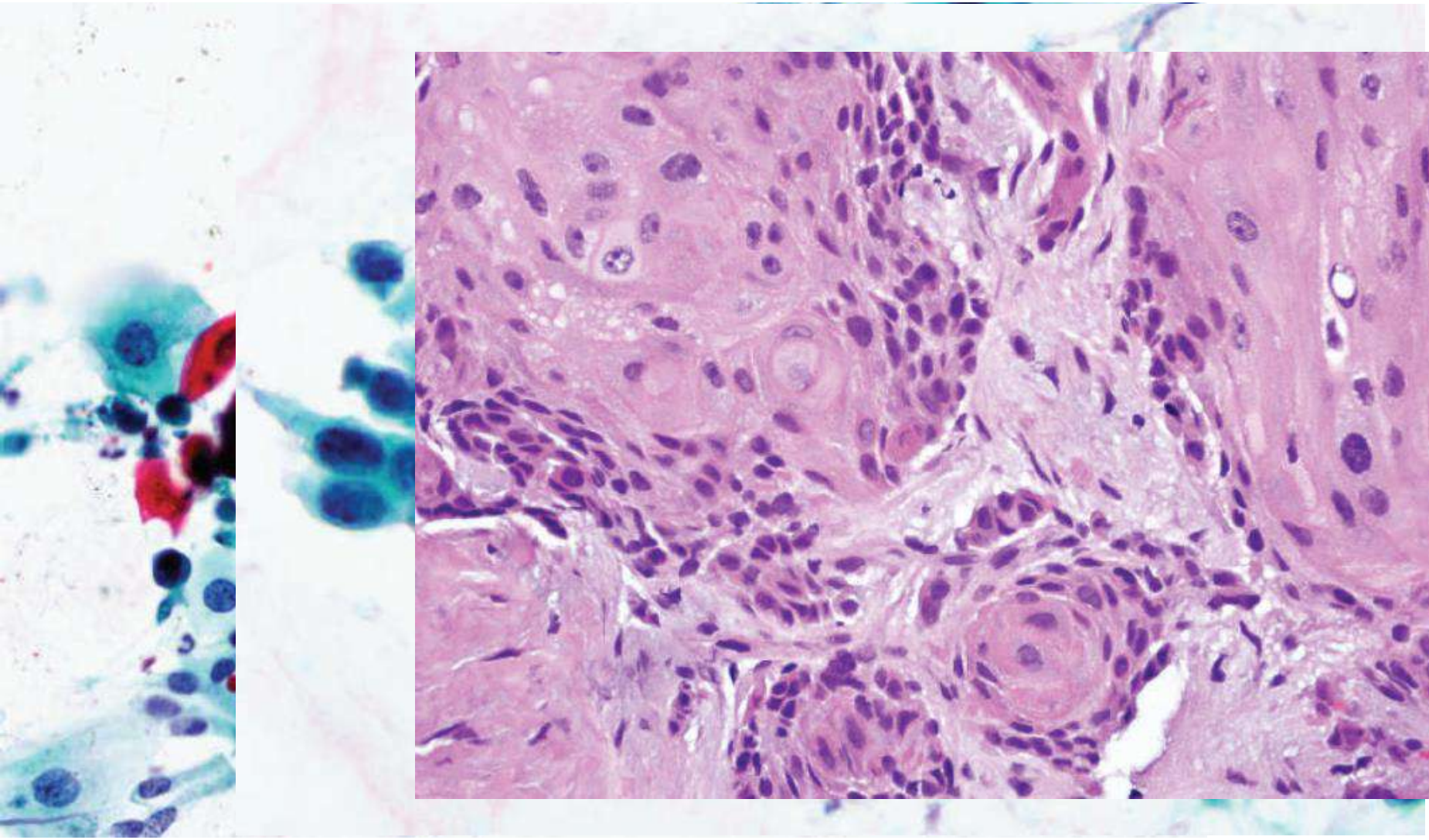


- Travis WD. Diagnosis of lung cancer in small biopsies and cytology. Implications of the 2011 international association for study of lung cancer/ American thoracic society/ European respiratory society classification. Arch pathol lab med. 2013

Squamous cell carcinoma

- Def: malignant epithelial tumor shows keratinization and/or intercellular bridges
- Clinical features: similar to adenocarcinoma
- Localization: arise in main or lobar bronchus
- Squamous marker: p40, p63, CK5/6

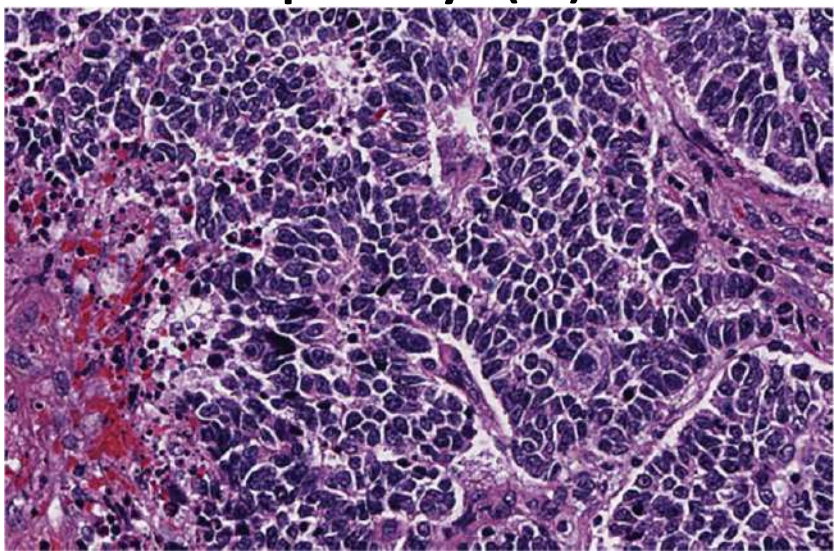
Squamous cell carcinoma



- Travis WD. Diagnosis of lung cancer in small biopsies and cytology. Implications of the 2011 international association for study of lung cancer/ American thoracic society/ European respiratory society classification. Arch pathol lab med. 2013

Small Cell Lung Cancer (SCLC)

- Small cells, scant cytoplasm, ill defined cell border, finely granular chromatin, inscopicuous nucleoli
- Expression neuroendocrine marker
- Hilar/ perihilar mass, lymphadenopathy (+)



Terima kasih…