ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
ΙΔΡΥΘΕΝ ΤΟ 1837

Ιατρική Σχολή
Α΄ ΠΑΘΟΛΟΓΙΚΗ ΚΛΙΝΙΚΗ
Διευθυντής: Καθηγήτρια Ελένη Ι. Γκόγκα

NSCLC+SCLC: The era of IO

Δημήτρης Ζιώγας MD, PhD

Παθολόγος Ογκολόγος

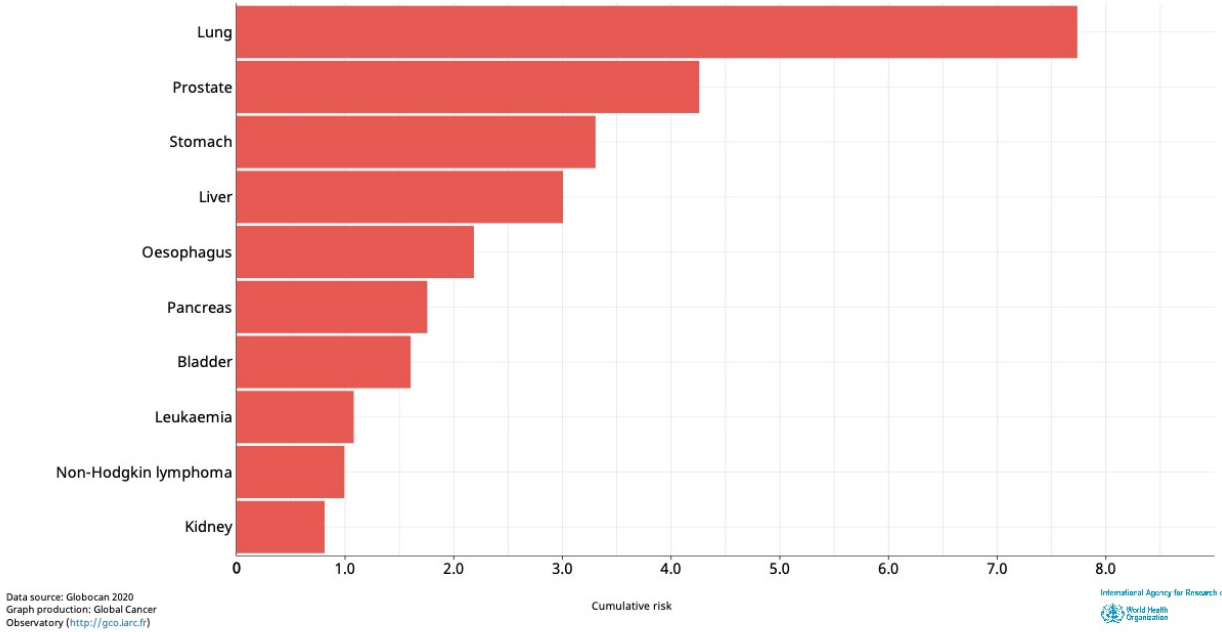
Επιστημονικός Συνεργάτης/Πανεπιστημιακός Υπότροφος

‘Α Παθολογική Κλινική

Γενικό Νοσοκομείο Λαϊκό

Lung Cancer Incidence/Mortality: US

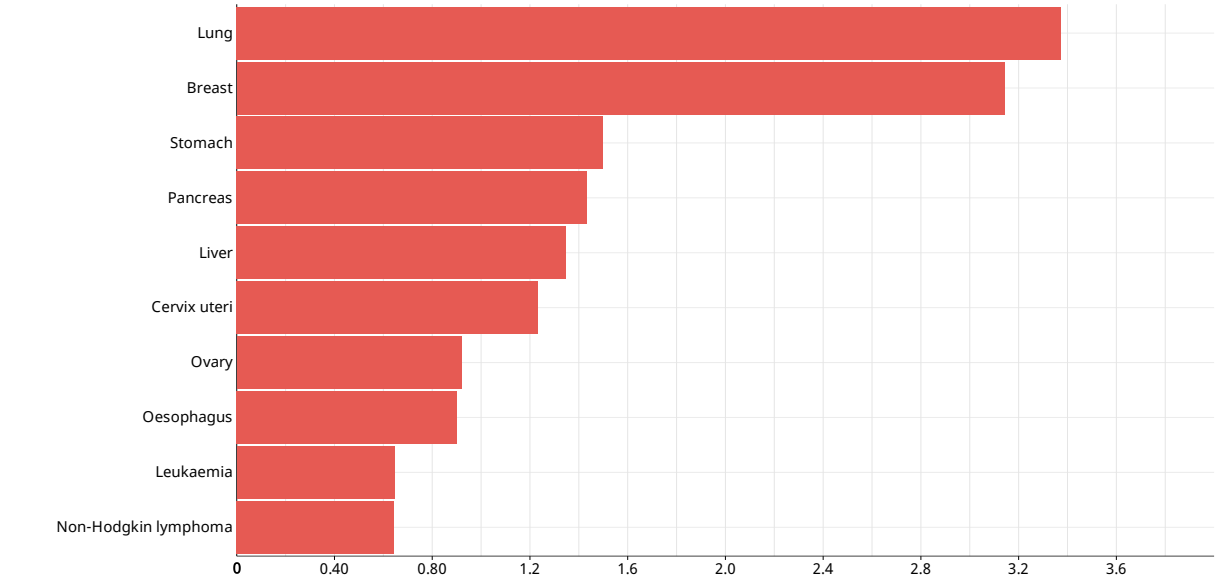
Estimated cumulative risk mortality in 2020, worldwide, males, all ages



Data source: Globocan 2020
Graph production: Global Cancer Observatory (http://gco.iarc.fr)

International Agency for Research on Cancer
World Health Organization

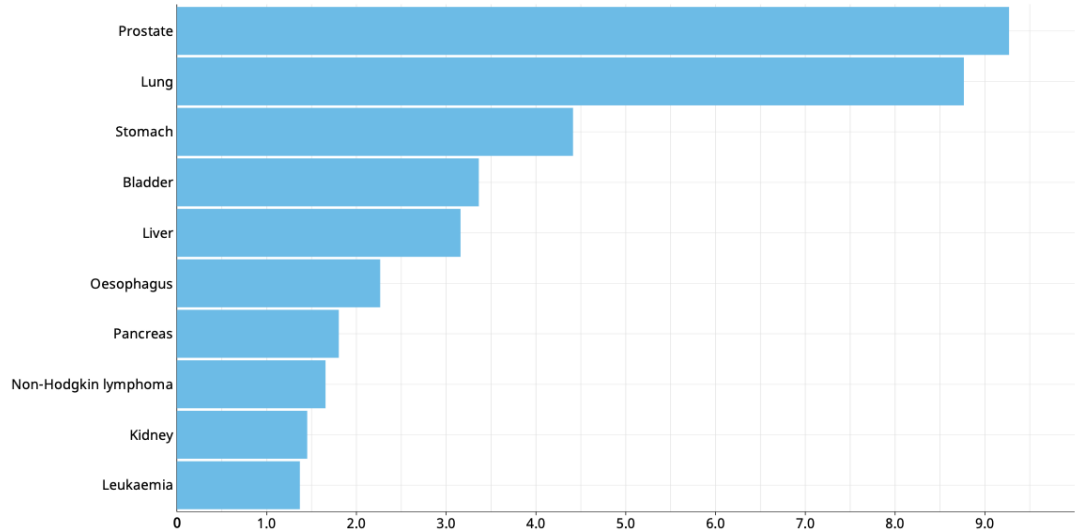
Estimated cumulative risk mortality in 2020, worldwide, females, all ages



Data source: Globocan 2020
Graph production: Global Cancer Observatory (http://gco.iarc.fr)

International Agency for Research on Cancer
World Health Organization

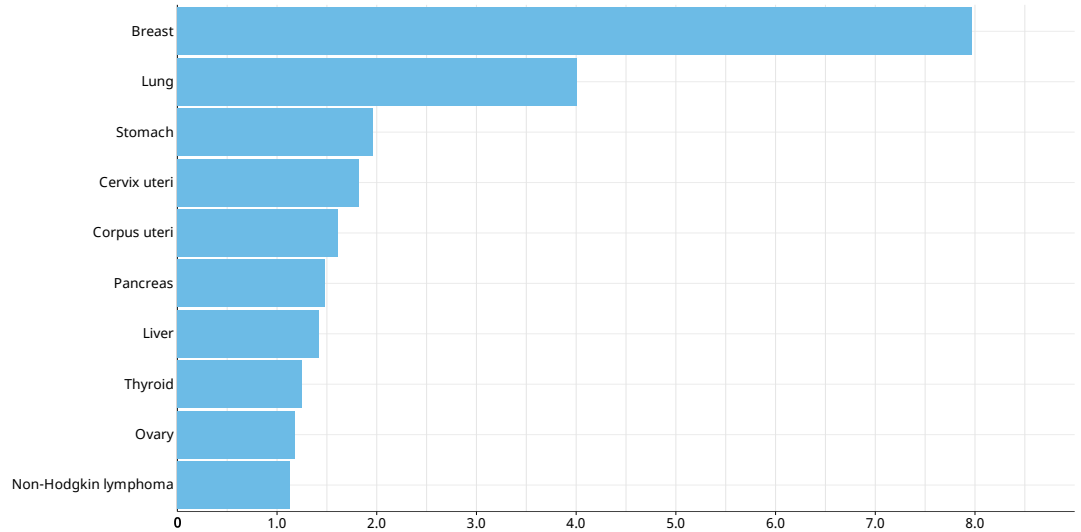
Estimated cumulative risk of incidence in 2020, worldwide, males, all ages



Data source: Globocan 2020
Graph production: Global Cancer Observatory (http://gco.iarc.fr)

International Agency for Research on Cancer
World Health Organization

Estimated cumulative risk of incidence in 2020, worldwide, females, all ages



Data source: Globocan 2020
Graph production: Global Cancer Observatory (http://gco.iarc.fr)

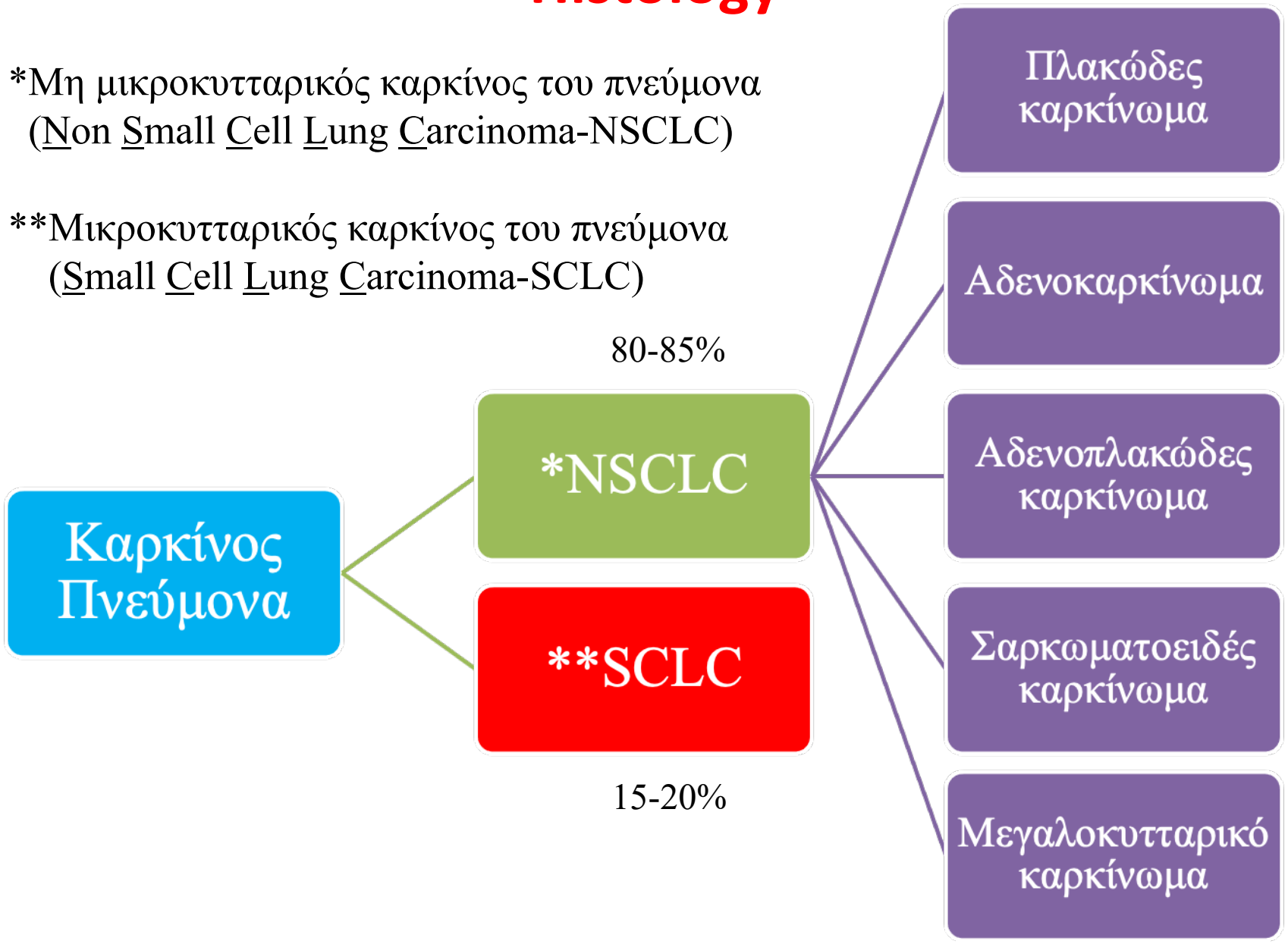
International Agency for Research on Cancer
World Health Organization

Histology-Molecular subtypes

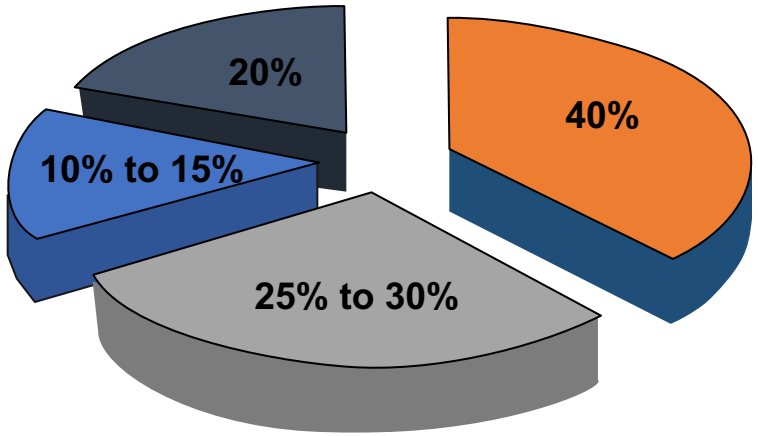
Histology

*Μη μικροκυτταρικός καρκίνος του πνεύμονα
(Non Small Cell Lung Carcinoma-NSCLC)

**Μικροκυτταρικός καρκίνος του πνεύμονα
(Small Cell Lung Carcinoma-SCLC)

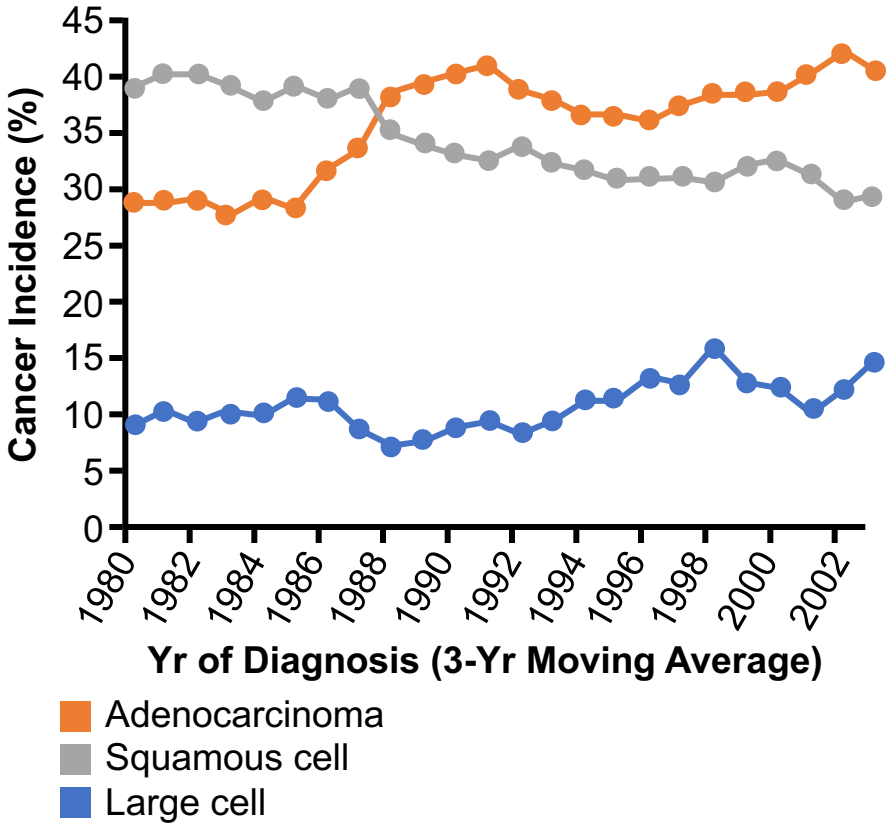


Traditional View of Lung Cancer



- Adenocarcinoma
- Squamous cell carcinoma
- Large cell carcinoma
- Other or not otherwise specified

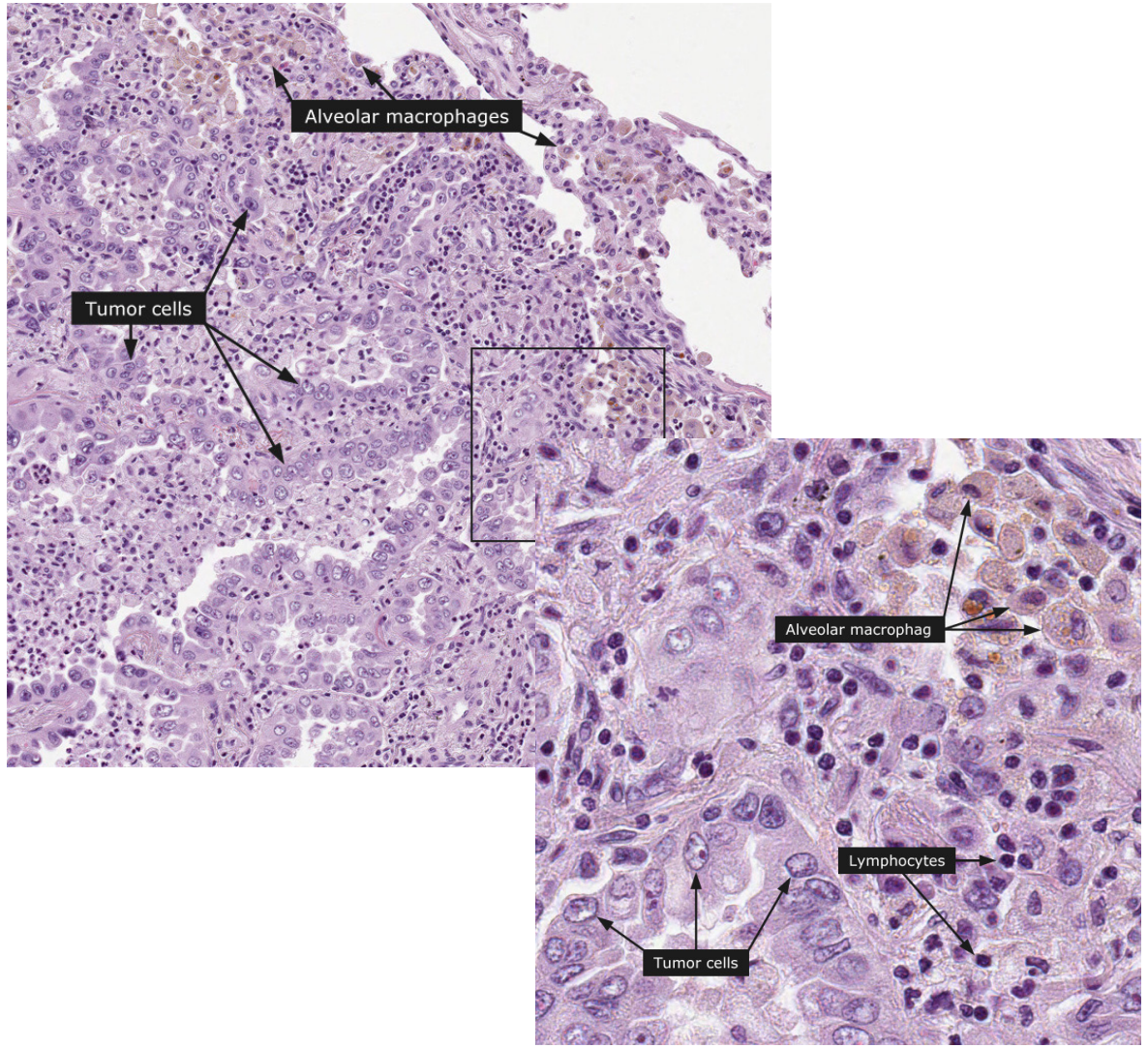
85% of lung cancers are NSCLC



American Cancer Society database.
Wahbah M, et al. Ann Diagn Pathol. 2007;11:89-96.

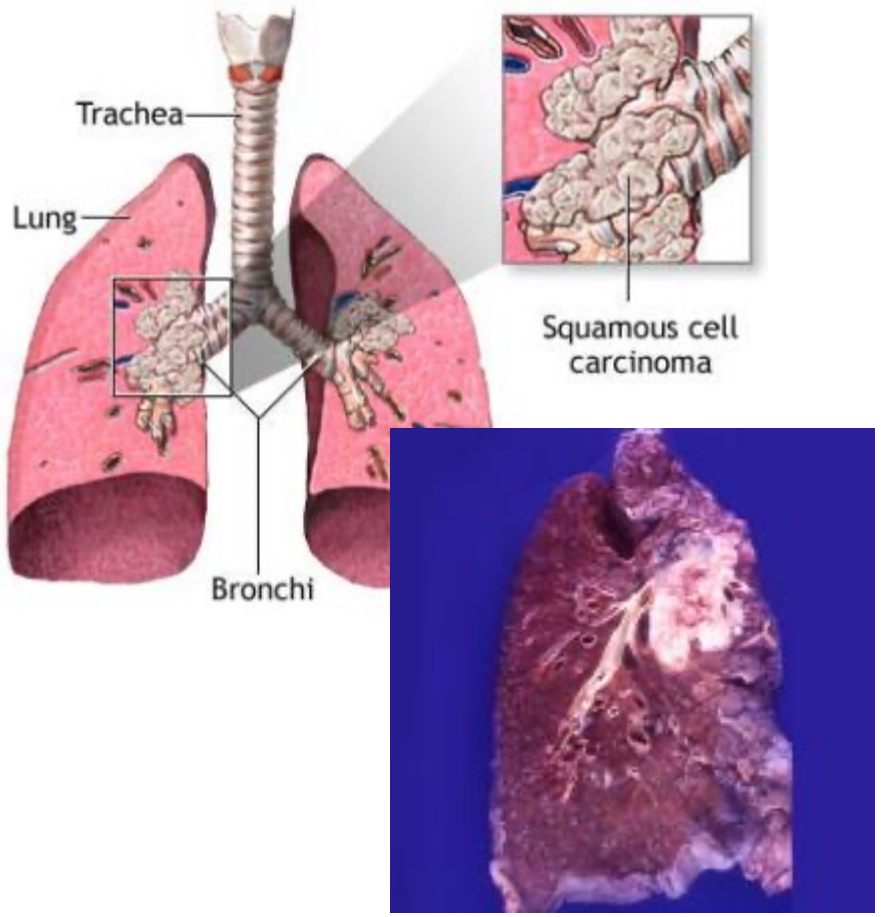
Lung adenocarcinoma (αδενοκαρκίνωμα πνεύμονα)

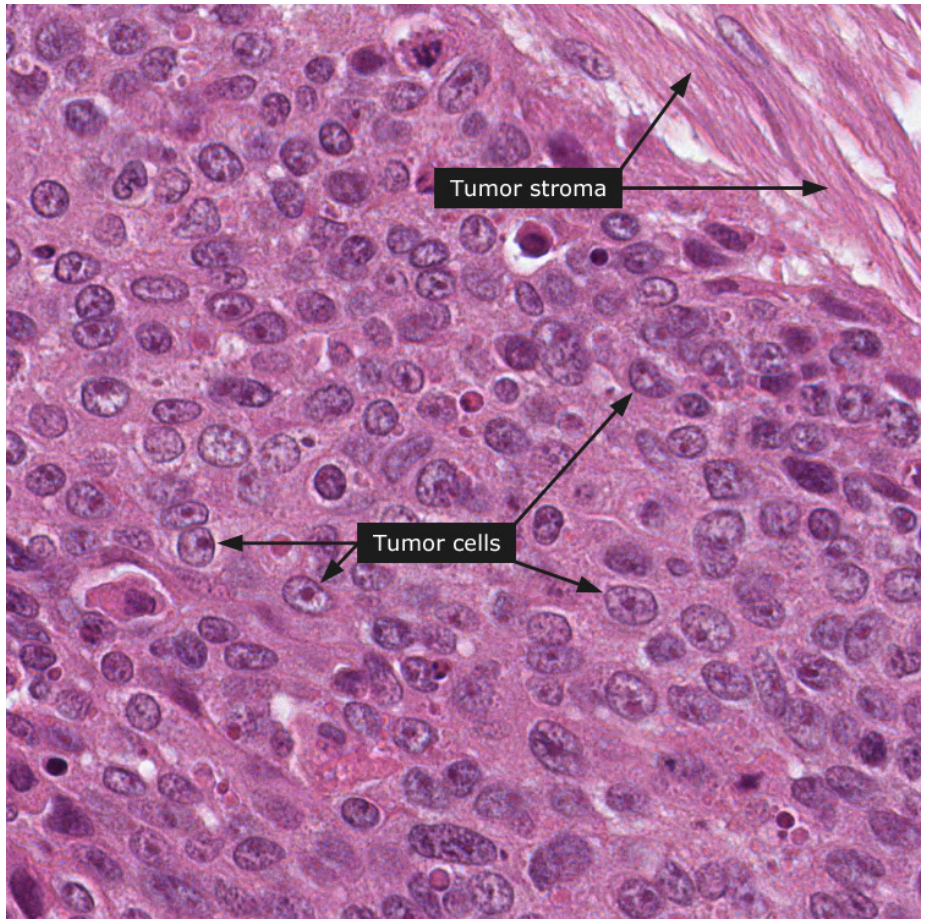
- **TTF-1 (+)**
- **P63/p40(-)**
- **CK5/6(-)**



Modern Pathology (2011) 24, 1348–1359
http://www.proteinatlas.org

Squamous cell carcinoma of lung (Πλακώδες καρκίνωμα πνεύμονα)

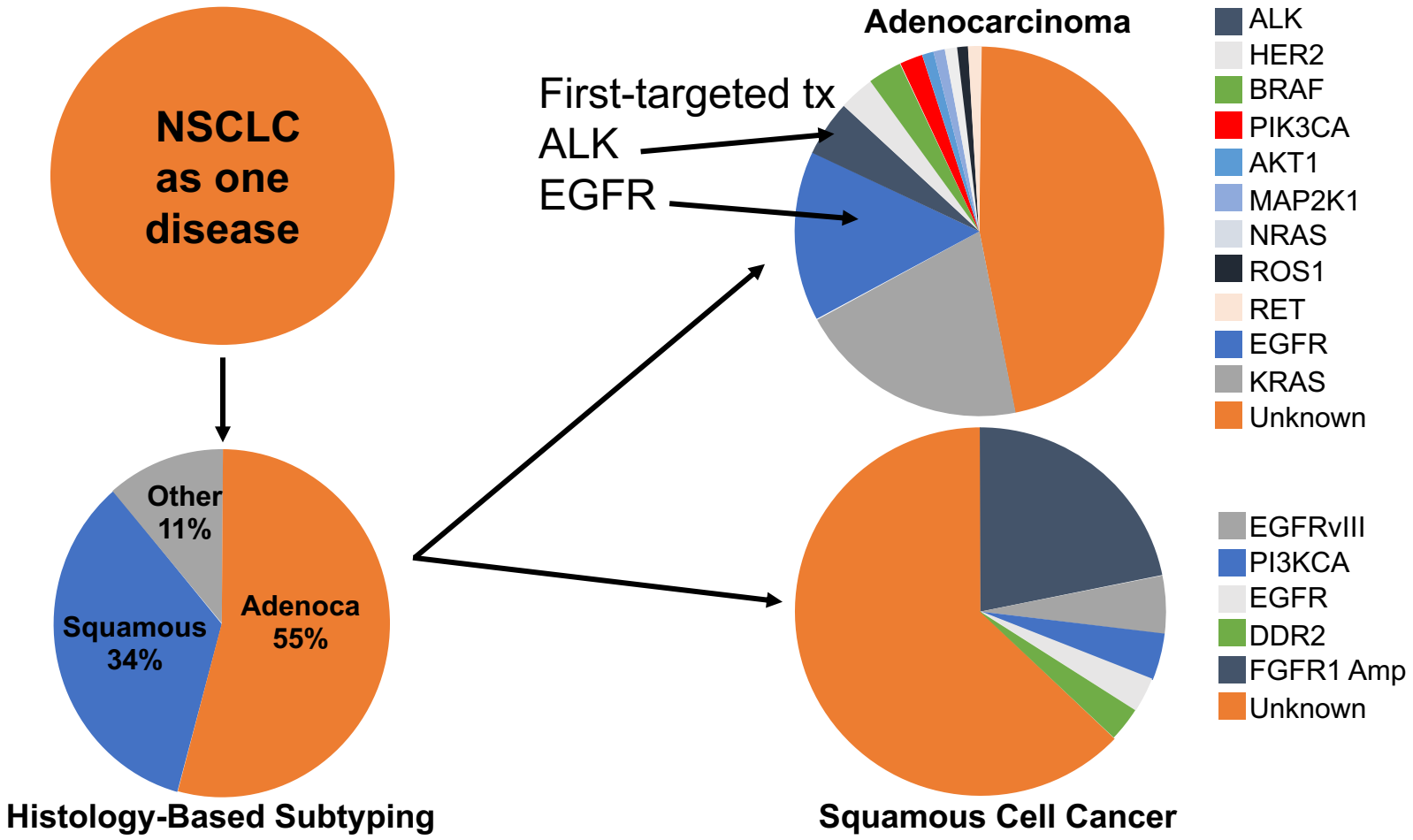




- **TTF-1 (-)**
- **p63/p40(+)**
- **CK5/6(+)**

Modern Pathology (2011) 24, 1348–1359
http://www.proteinatlas.org

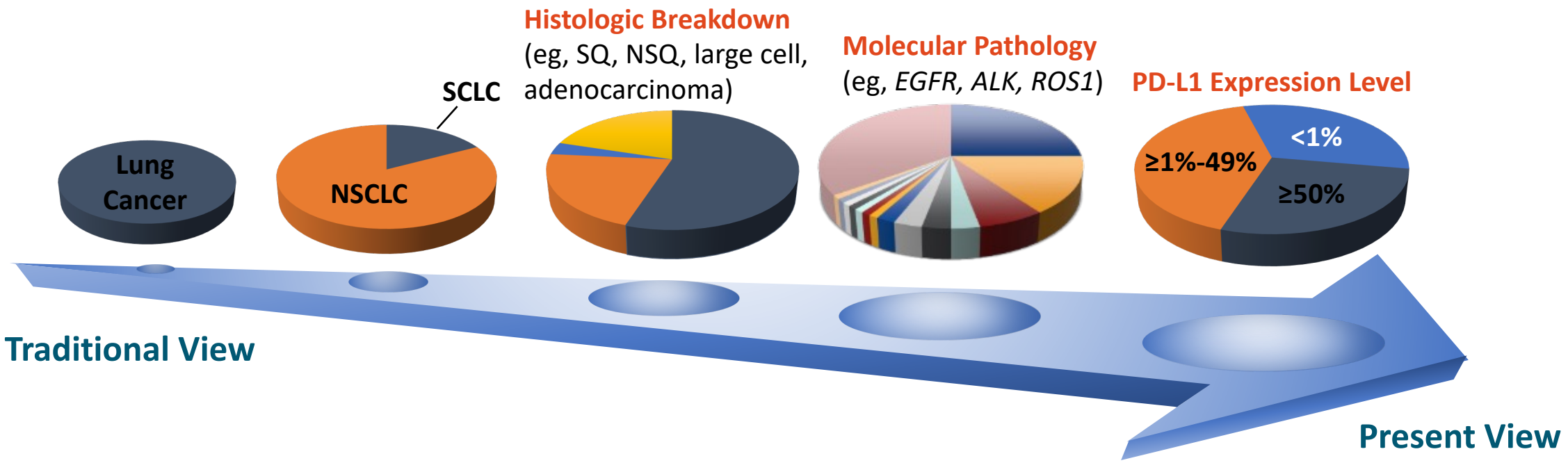
Evolution of NSCLC Subtyping to a Multitude of Molecular-Defined Subsets



Li T, et al. J Clin Oncol. 2013;31:1039-1049.

Evolution of Therapy in Lung Cancer

- Not 1 disease, but many



Cooper. Pathology. 2011;43:103. Langer. JCO. 2010;28:5311. Galon. Immunity. 2013;39:11.
Pao. Lancet Oncol. 2011;12:175. Krigsfeld. AACR 2017. Abstr CT143.

Epidemiology of NSCLC

- 83% of lung cancer,
  - the leading cause of cancer mortality in the US
    - New cases, 2015: US, 221,200; global, 2M
    - Deaths, 2015: US, 158,040; global, 1.5M

- ~ 80% of cases are associated with smoking

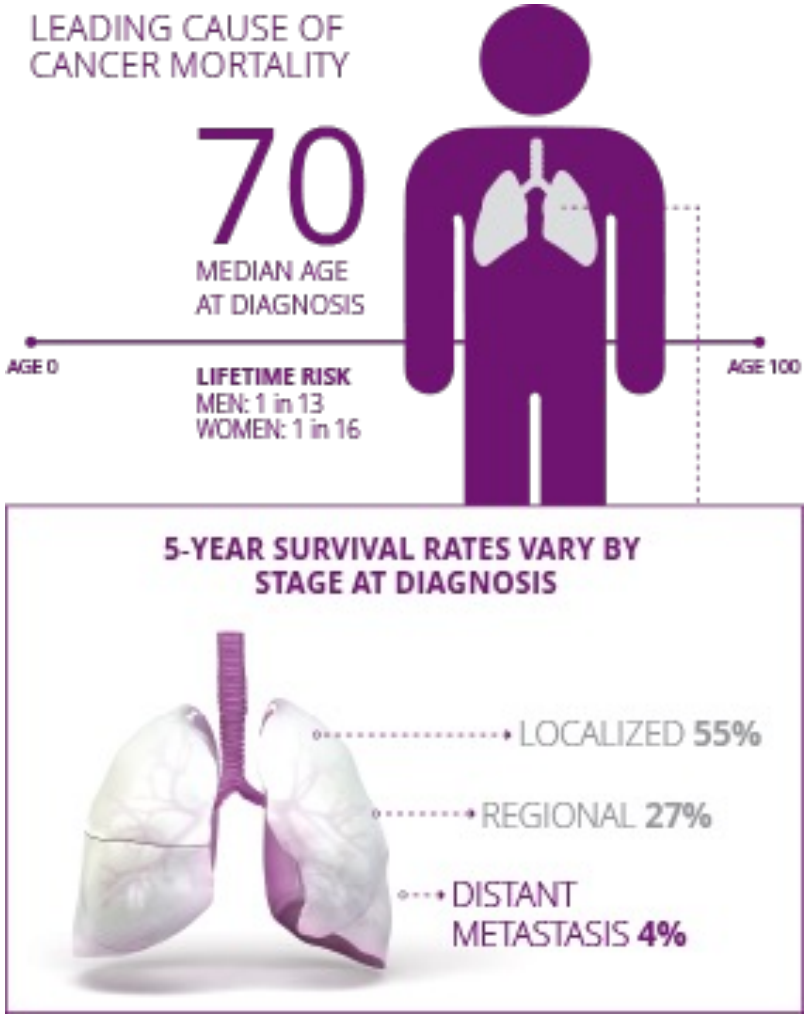
- In non-smokers is most often adenocarcinoma and disproportionately affects women vs. men

- Incidence of NSCLC varies among racial, ethnic, and socioeconomic groups

- 55% of NSCLC patients are diagnosed with advanced disease

- 5-year OS rate for metastatic NSCLC is 4%

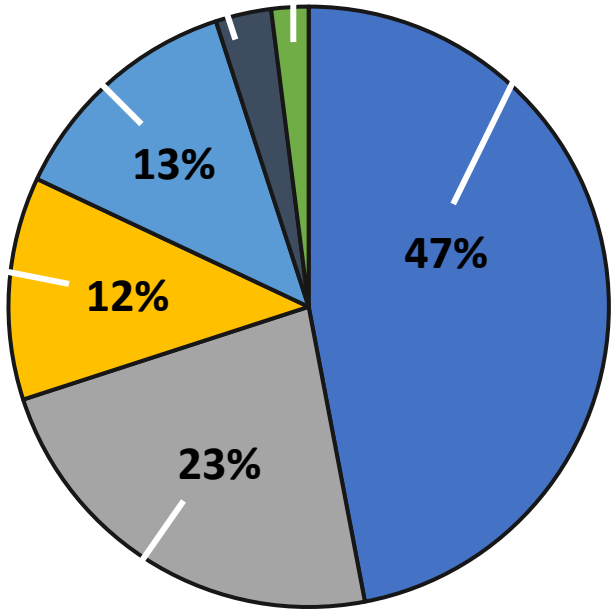
- Contributes to more deaths than breast, colon, and prostate cancer combined



1. Cancer statistics, 2016. CA Cancer J Clin. 2016;66:7-30.
2. American Cancer Society. Cancer facts and figures 2016. http://www.cancer.org/acs/groups/content/@research/documents/document/acspc-047079.pdf

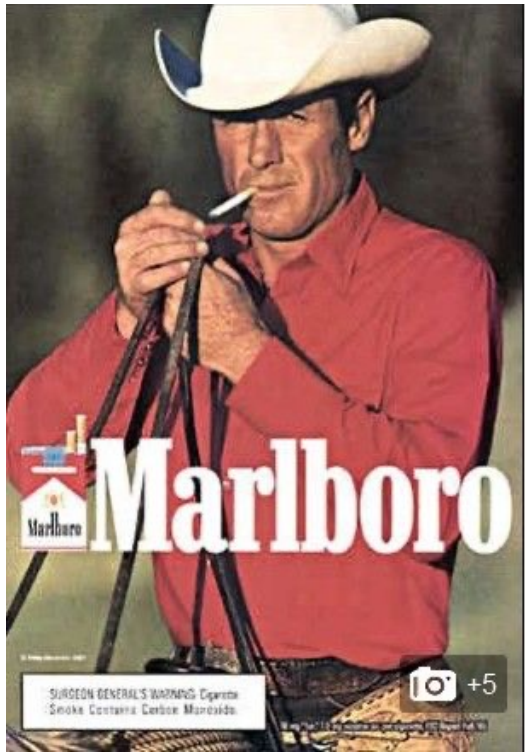
Small-Cell Lung Cancer

- SCLC accounts for ~ 13% of all lung cancers in the US
- Previously called oat-cell carcinoma
- Associated with a history of significant tobacco use
- Unique biology: rapid proliferation, abrupt presentation, bulky central tumor, hematogenous metastases at onset
- Poor outcomes



Oronsky. Neoplasia. 2017;19:842. Alvarado-Luna. Transl Lung Cancer Res. 2016;5:26. Howlander. SEER Cancer Statistics Review, 1975-2014.

Etiology of SCLC



Clinical presentation of NSCLC

- Cough,
- Fatigue,
- Dyspnea,
- Pain,
- Weight loss

- Metastasis common in lung, adrenal glands, liver, brain, bones; symptoms are site specific

- Diagnosis requires lesion assessment via x-ray, CT, PET imaging, and finally pathologic assessment in combination with molecular testing

- Biomarker assessment may assist in predicting treatment response

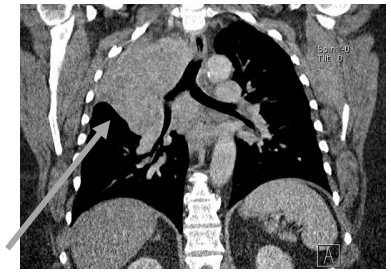
- Molecular analysis, histology, cancer stage, patient age, and PS will be taken into account for the therapeutic decision

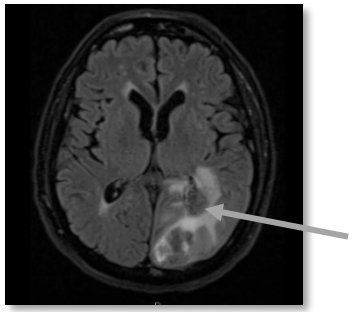
Symptoms of lung cancer in over 3500 patients at presentation

Symptom	Patients (percent)
Cough	45-74
Weight loss	46-68
Dyspnea	37-58
Chest pain	27-49
Hemoptysis	27-29
Bone pain	20-21
Hoarseness	8-18

Clinical Presentation of SCLC

- Local symptoms: cough, 50%; dyspnea, 40%; chest pain, 35%; hemoptysis, 20%; hoarseness, 10%
- Distant symptoms: weight loss, 50%; weakness, 40%; anorexia, 30%; paraneoplatic syndrome, 15%; fever, 10%
- Paraneoplastic syndromes: ectopic hormone-associated syndromes, immune-mediated neurologic syndromes



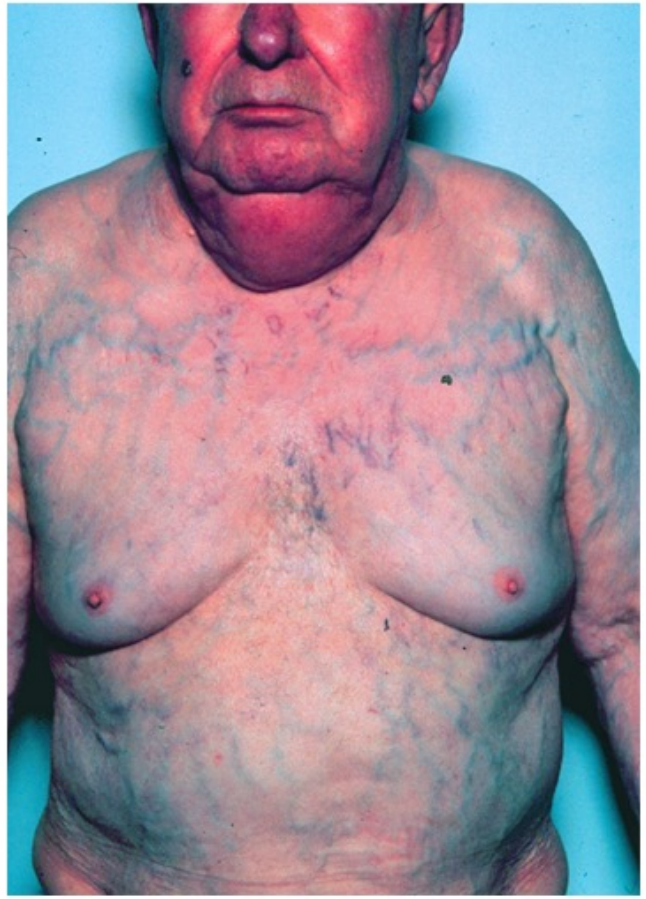


Metastatic Site, %	At Presentation	At Autopsy
Mediastinal LNs	66-80	73-87
Liver	21-27	69
Bone	27-41	54
Adrenal glands	5-31	35-65
Bone marrow	15-30	NA
Brain	10-14	28-50
Retroperitoneal LNs	3-12	29-52
Supraclavicular LNs	17	42
Pleural effusion	16-20	30
Contralateral lung	1-12	8-27
Soft tissues	5	19

Jackman. Lancet. 2005;366:1385. Images courtesy of Anna F. Farago, MD, PhD.

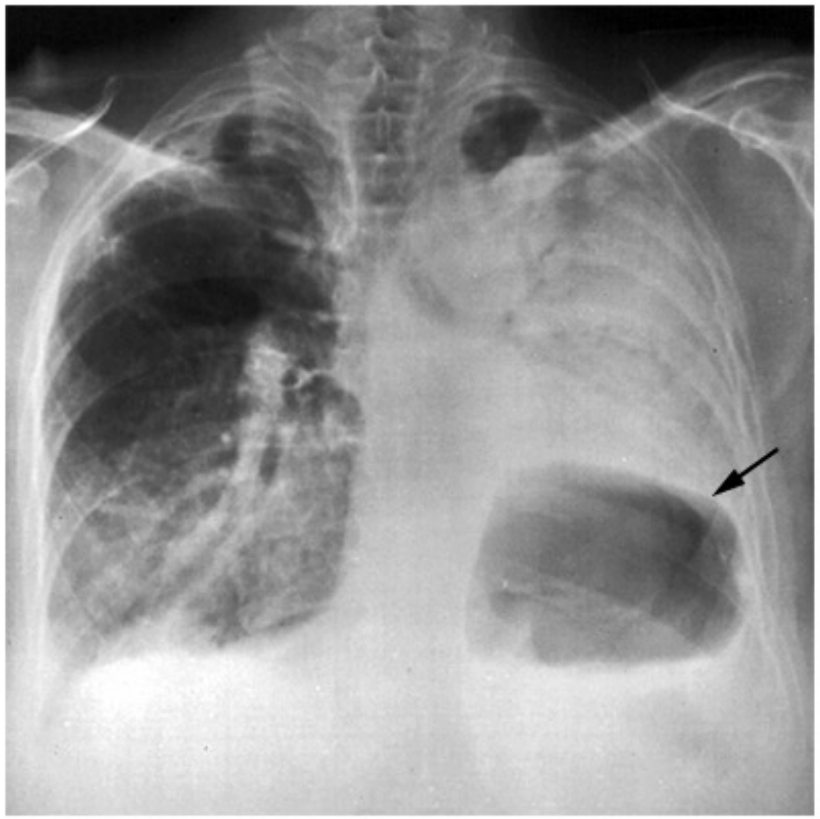
Clinical presentation of lung cancer (1)

Superior vena cava syndrome



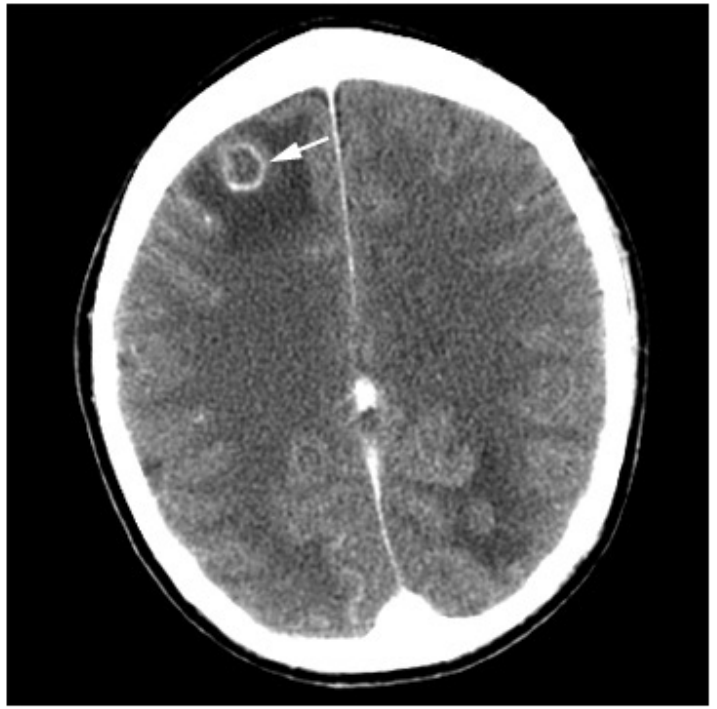
A prominent venous pattern on the chest, facial edema, and a plethoric appearance is present in this man with SVC obstruction from lung cancer.

Peripheral nerve involvement



Left hemidiaphragm (arrow) paralysis (confirmed by fluoroscopy) in a patient with extensive adenocarcinoma of the lung.

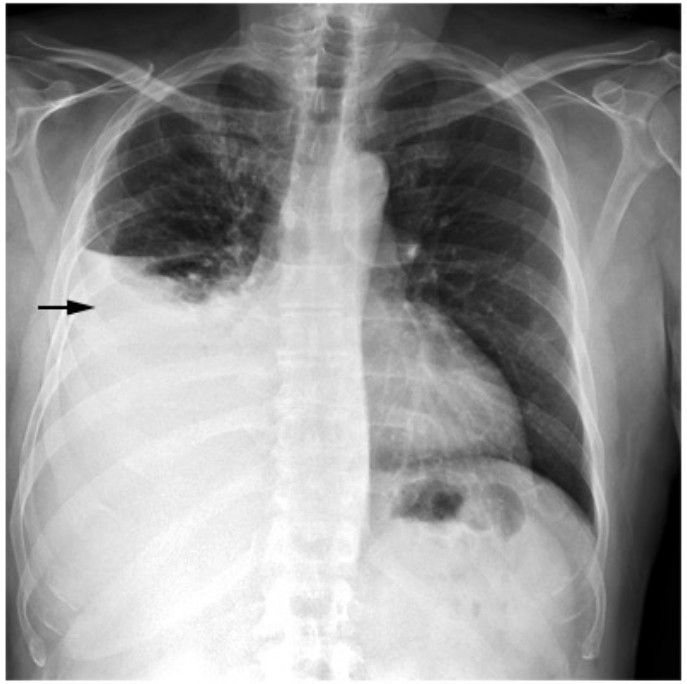
Brain metastasis

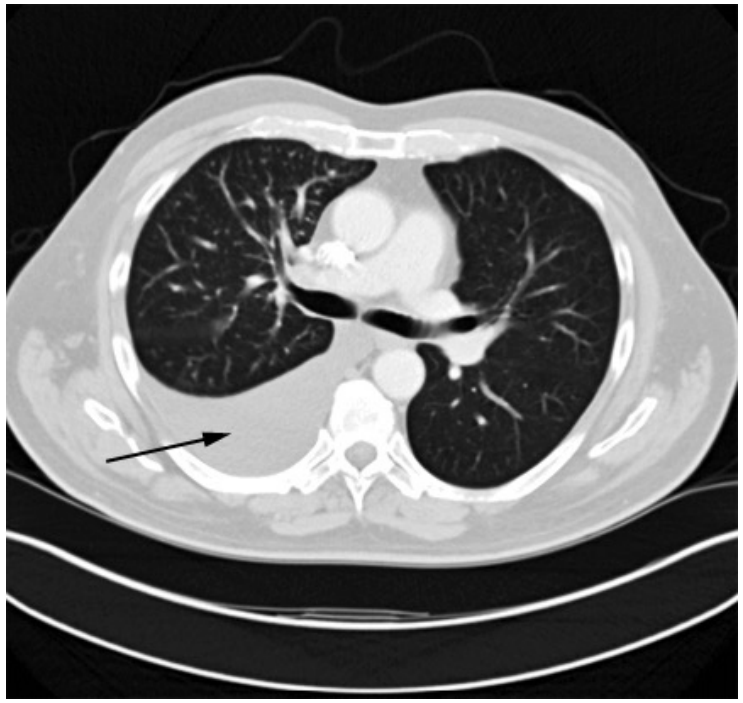


Head CT showing one of multiple ring enhancing lesions (arrow) in the brain of a 45-year-old male presenting with headaches. Biopsies of a right upper lobe mass and subcarinal adenopathy showed adenocarcinoma.

Clinical presentation of lung cancer (2)

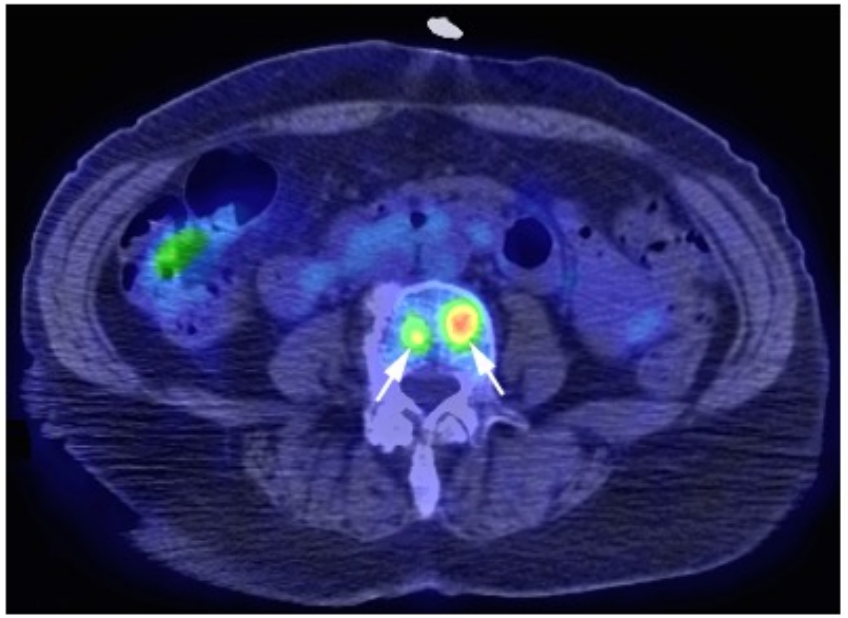
Malignant pleural effusion





Large pleural effusion (arrow) in a 60-year-old man cytologically positive for adenocarcinoma consistent with lung primary.

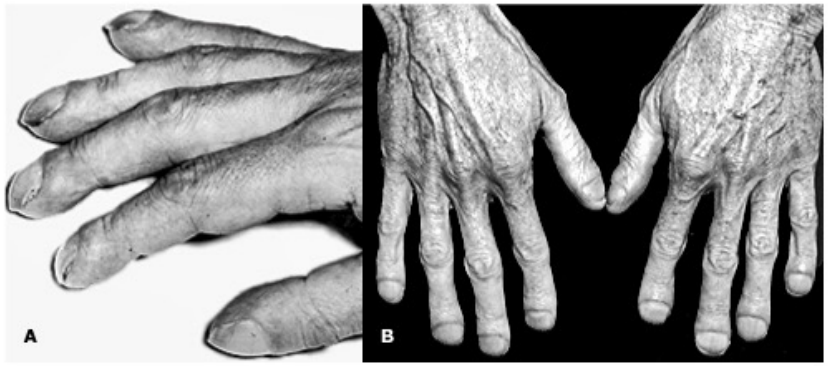
Bone metastasis



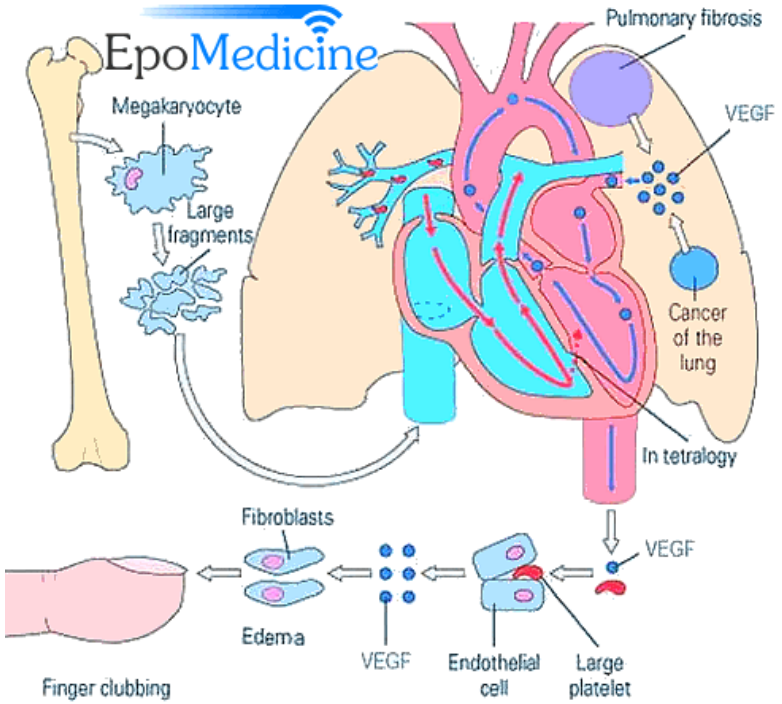
PET-CT showing two foci of hypermetabolic activity in a lumbar vertebral body (arrows) in a 76-year-old woman with metastatic adenocarcinoma of the lung.

Paraneoplastic syndromes in lung cancer

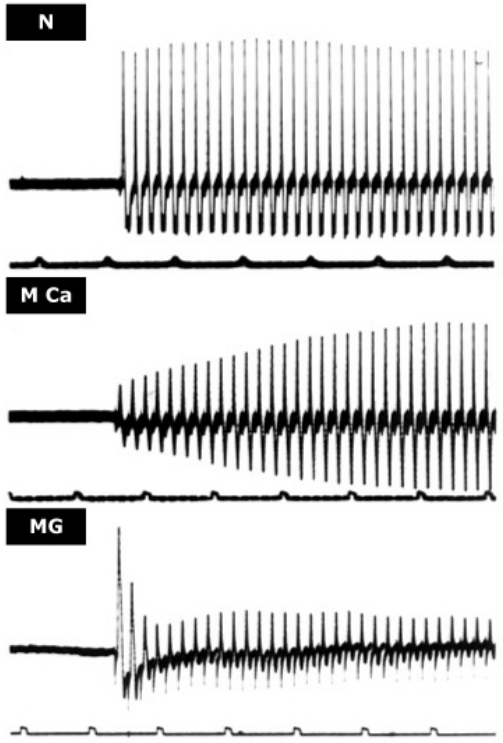
Clubbing



A) Side and B) top view of nail bed hypertrophy causing a distal enlargement of the fingers in a patient with lung cancer.



Lambert Eaton myasthenic syndrome (LEMS)



Electromyograms showing action potentials from a normal **(N)** patient, a patient with LEMS **(MCa)**, and a patient with myasthenia gravis **(MG)** for comparison.

Hypertrophic pulmonary osteoarthropathy

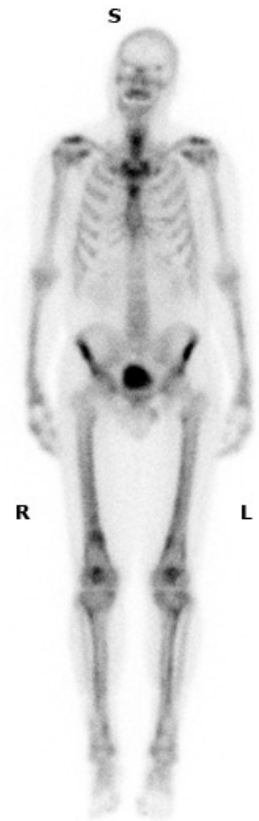


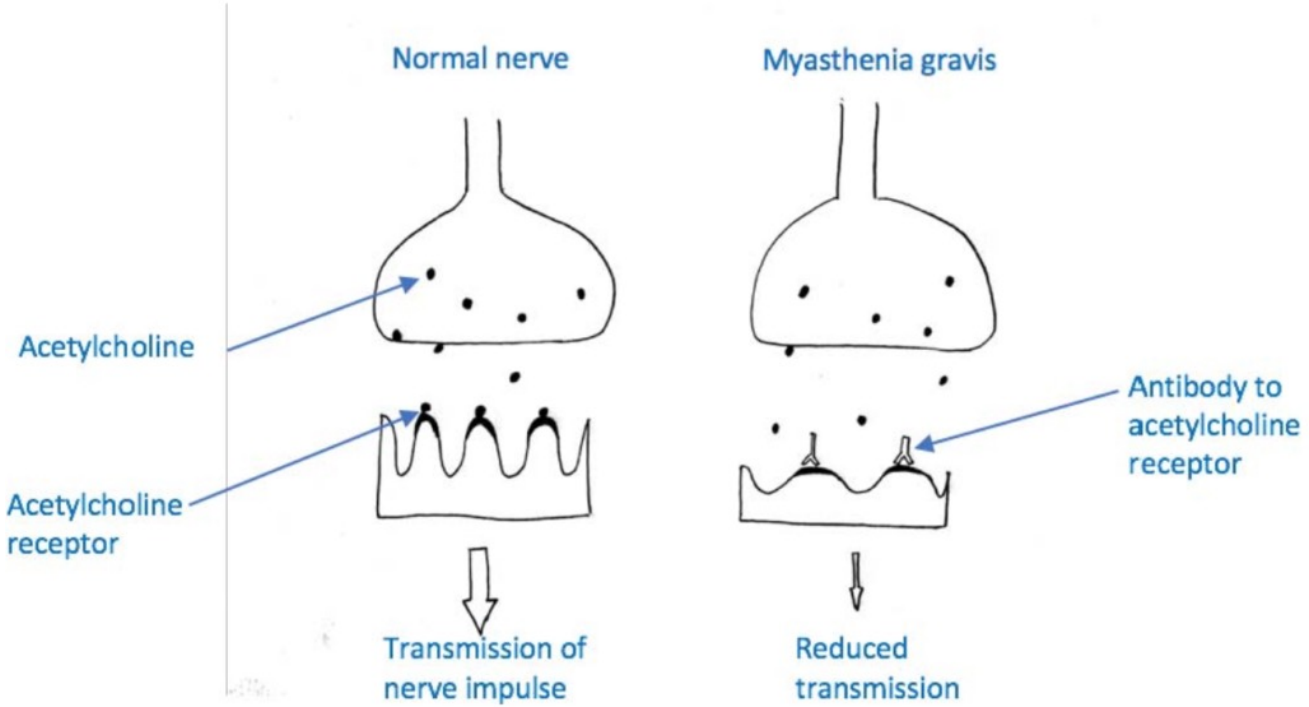
Image from a 69-year-old male with non-small cell lung cancer (NSCLC) and bilateral lower extremity pain. Whole body bone of shows patchy uptake in the periosteal aspects of the long bones typical of hypertrophic pulmonary osteoarthropathy. No clear evidence of skeletal metastases.

Overview of Myasthenia Gravis

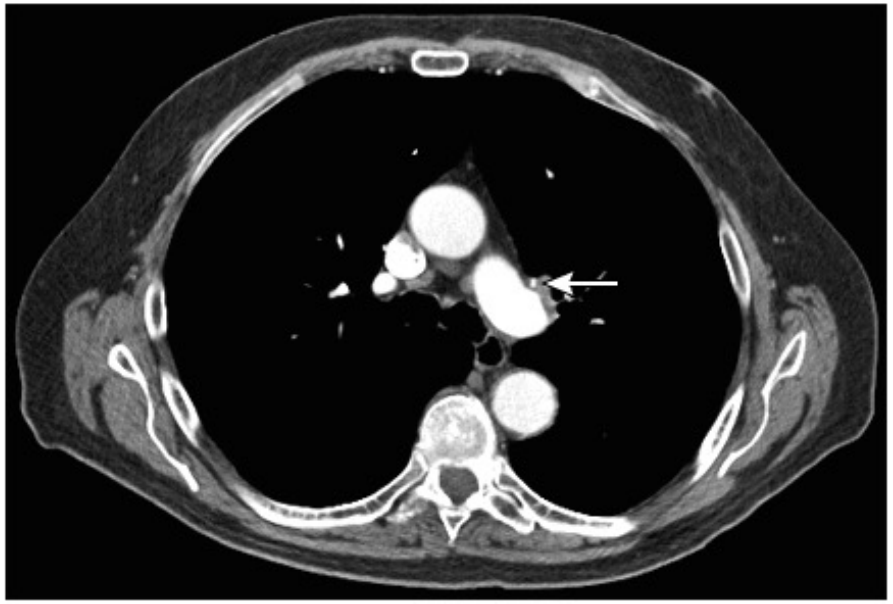
- Autoimmunity targeting functional post-synaptic components of the neuromuscular junction (e.g., Ach-R, MuSK, LRP4).

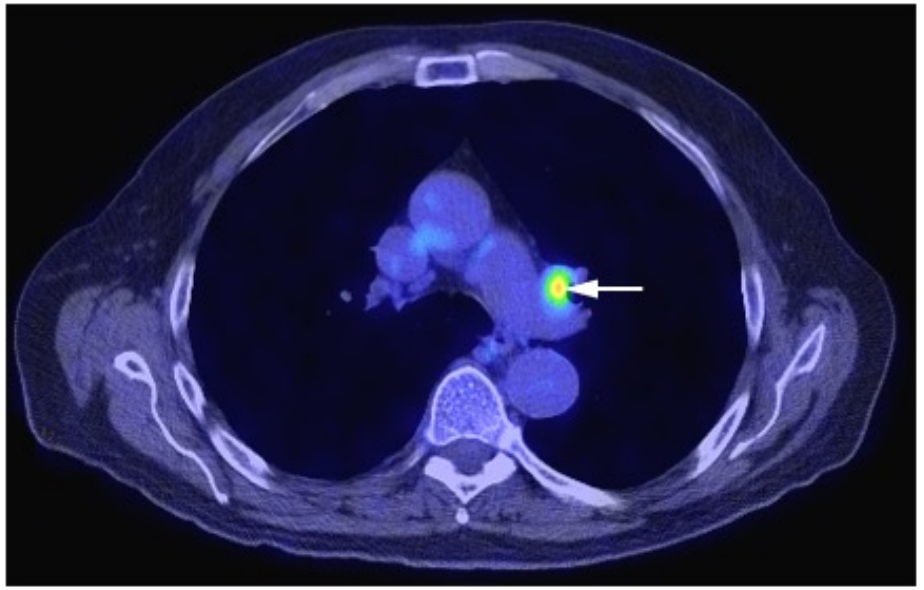
- Two major autoantibodies (found in 85% of patients)
  1. **Anti-Ach-R autoantibodies**
     - 80-95% of those with generalized MG
     - 50% of those with ocular MG

  2. **Anti-MuSK autoantibodies**
     - 50% of anti-AChR negative patients
     - 15% of MG patients have thymoma (99% with thymoma are anti-AChR+)

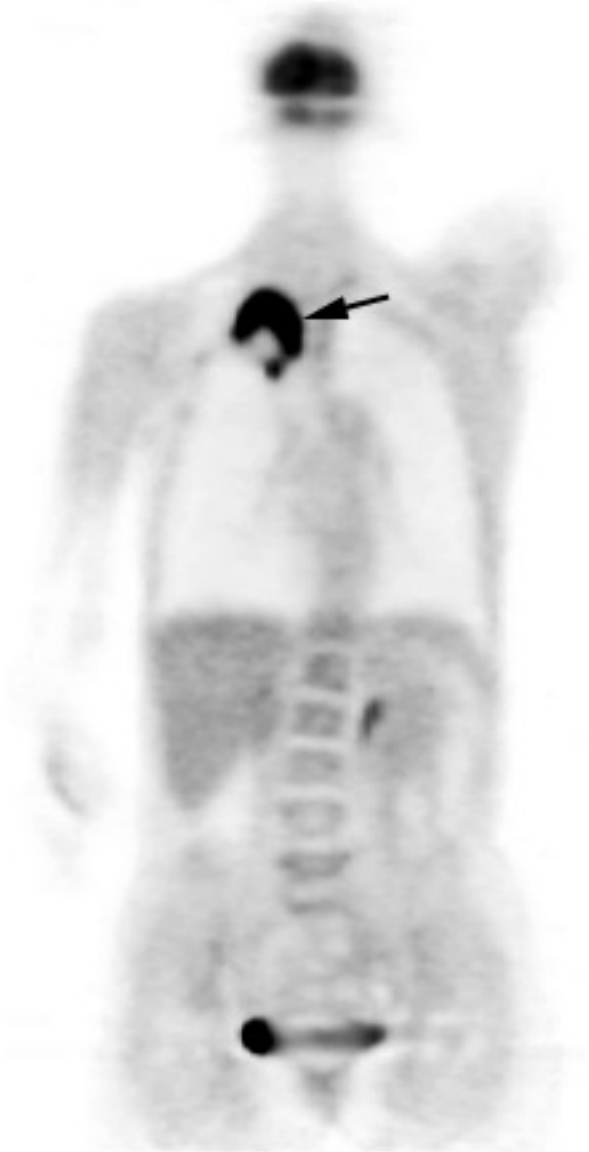


Imaging…

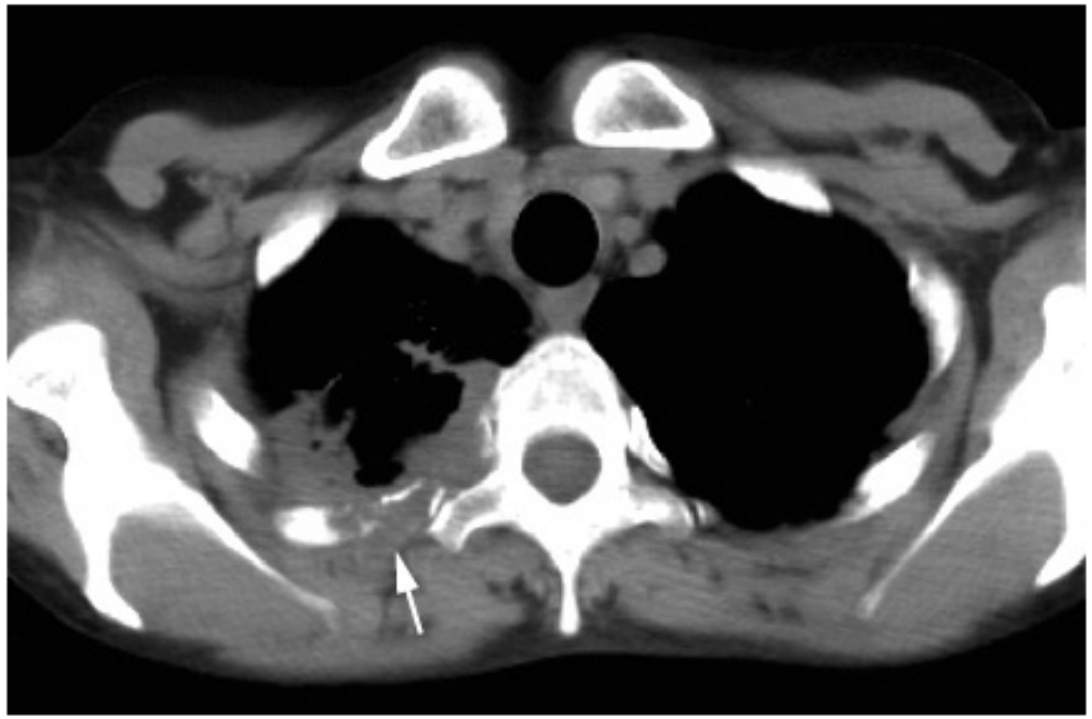




This 71-year-old man presented with a polyneuropathy thought secondary to a paraneoplastic syndrome as he had a high P/Q type calcium channel antibody present in serum. PET-CT showing intense metabolic activity (arrow) in a normal sized left hilar node after CT was negative. Ultrasound guided bronchoscopy sampling revealed small cell lung cancer in this hilar node.

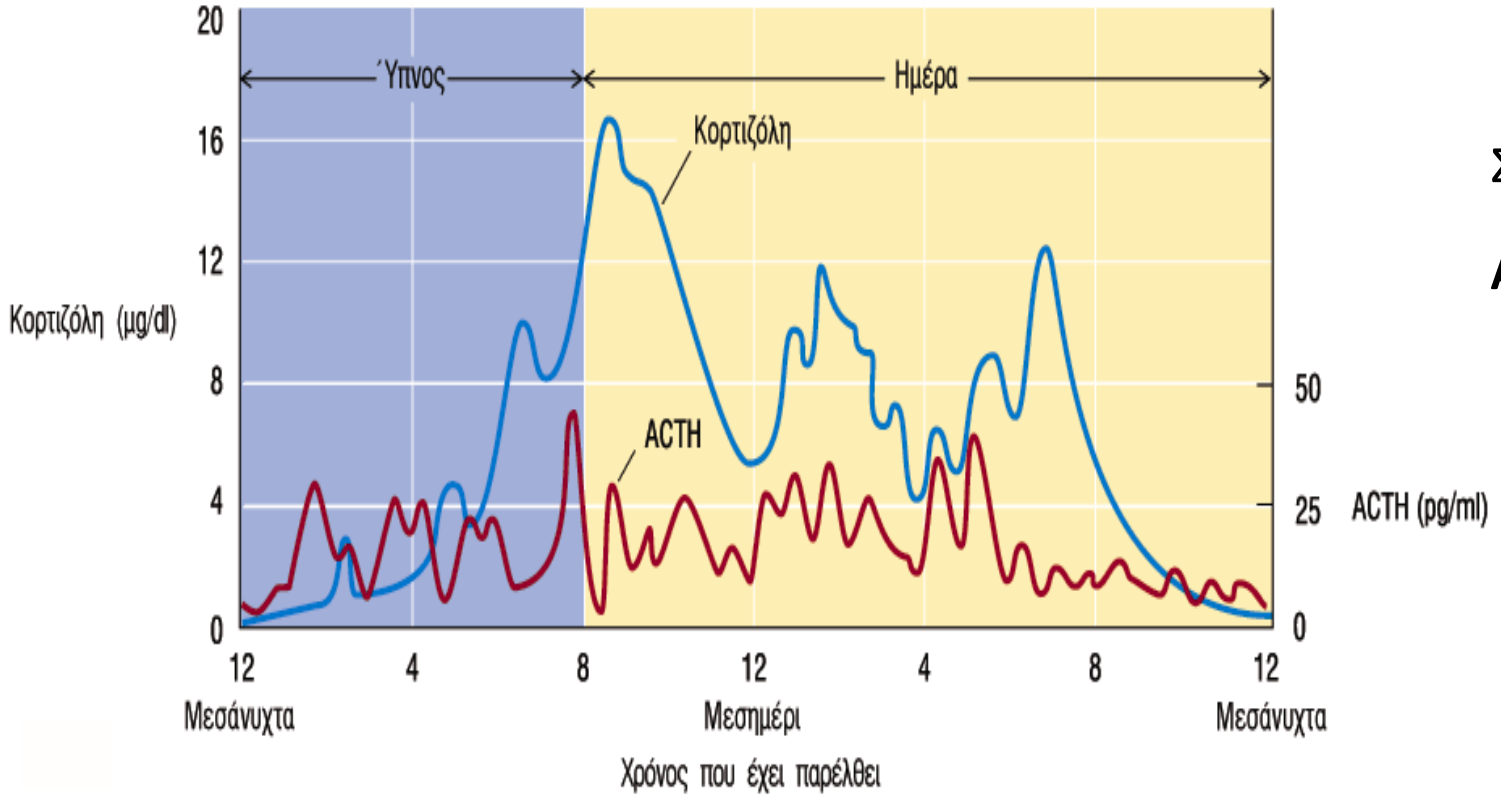


Pancoast tumor



CT image of a 60-year-old woman presenting with a four month history of right shoulder pain and numbness and tingling in the right arm. The cavitary mass involved the rib and was squamous histology on biopsy.

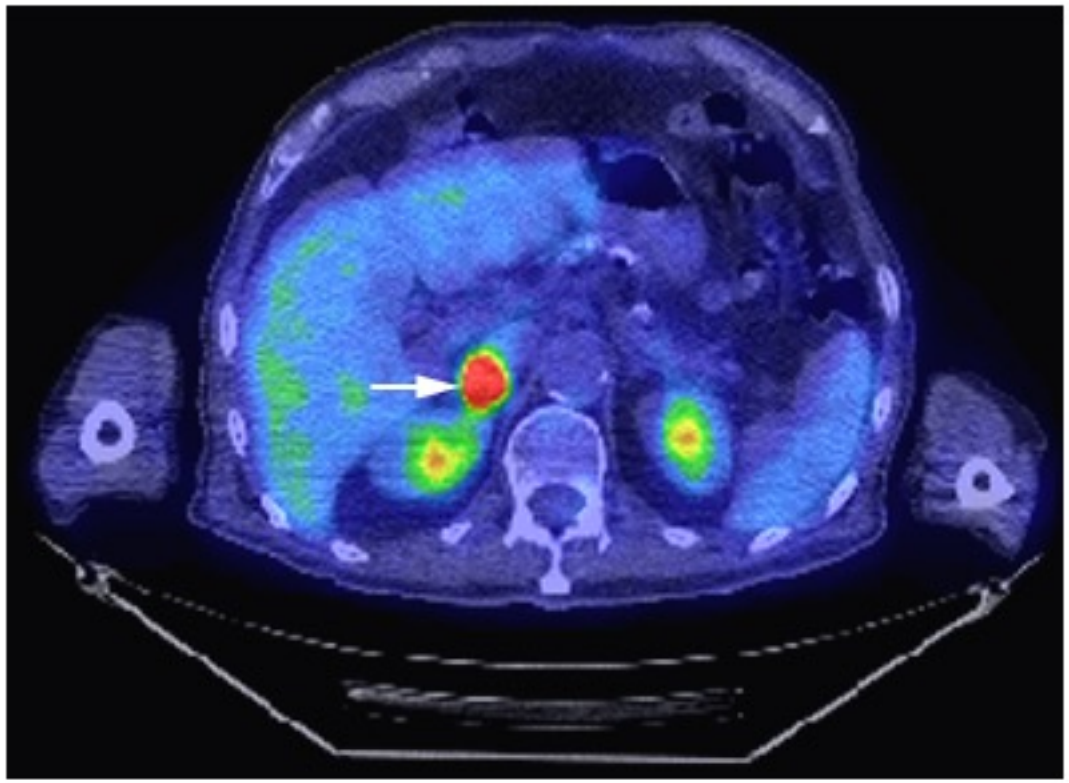
ΚΙΡΚΑΔΙΟΣ ΡΥΘΜΟΣ ΕΚΚΡΙΣΗΣ ΚΟΡΤΙΖΟΛΗΣ



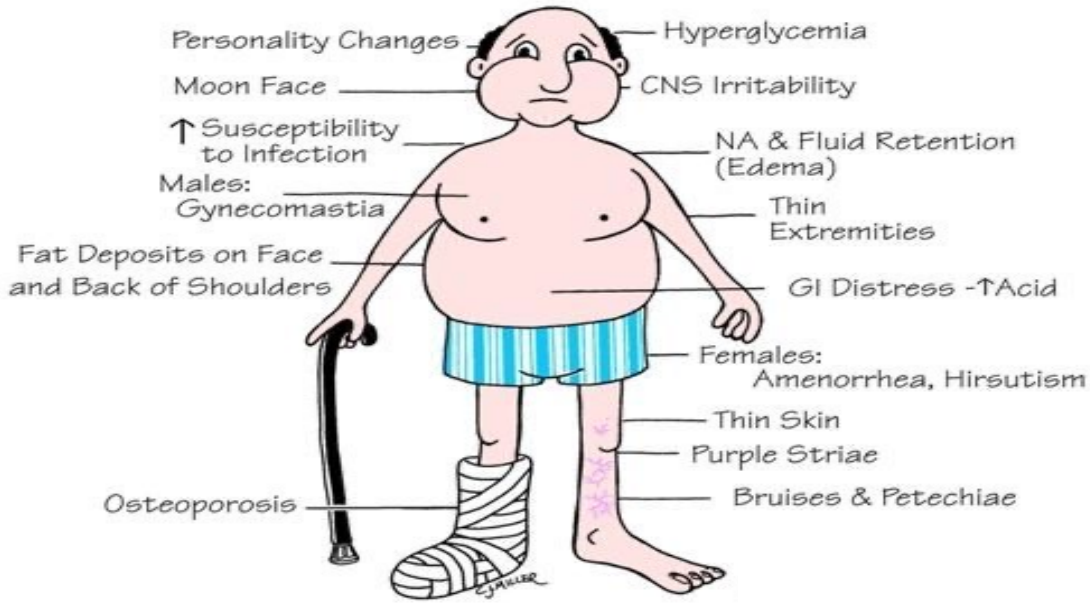
Σύνδρομο Cushing:

Απώλεια κιρκάδιου ρυθμού κορτιζόλης

Adrenal metastasis



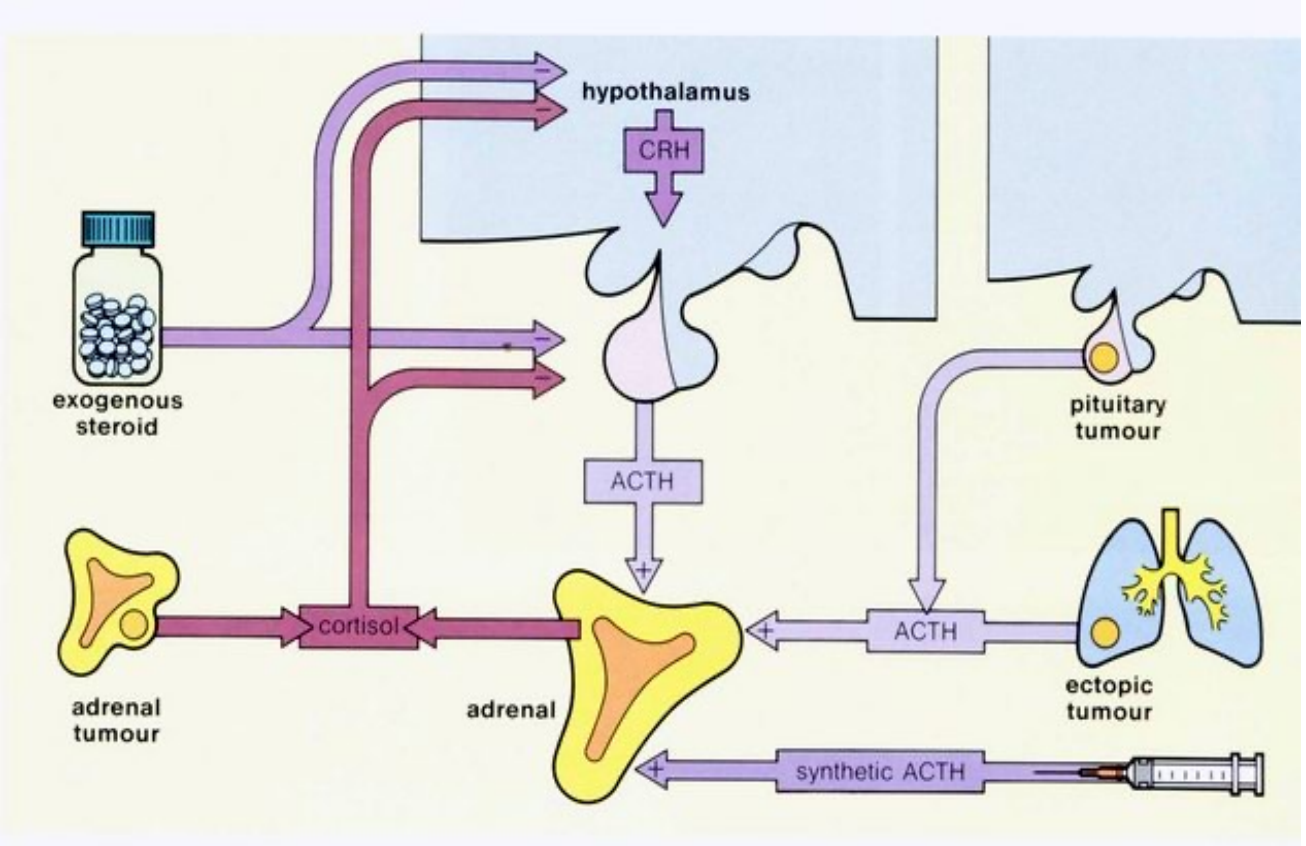
PET-CT showing intense metabolic activity in the right adrenal gland in a 76-year-old with a right paratracheal mass. CT guided adrenal biopsy confirmed stage IV non-small cell carcinoma of the lung.



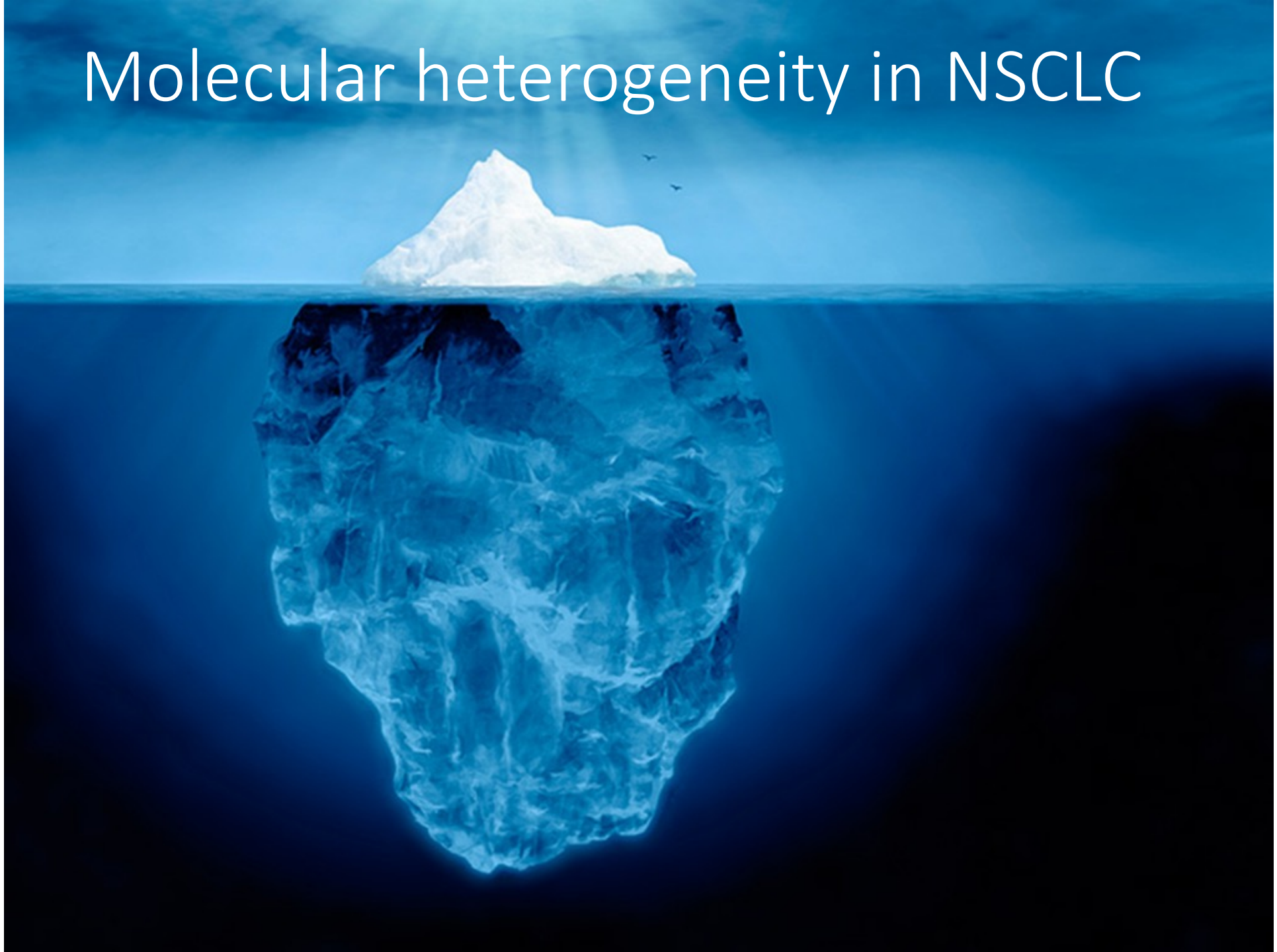
Cushing's syndrome

Causes of Cushing's syndrome

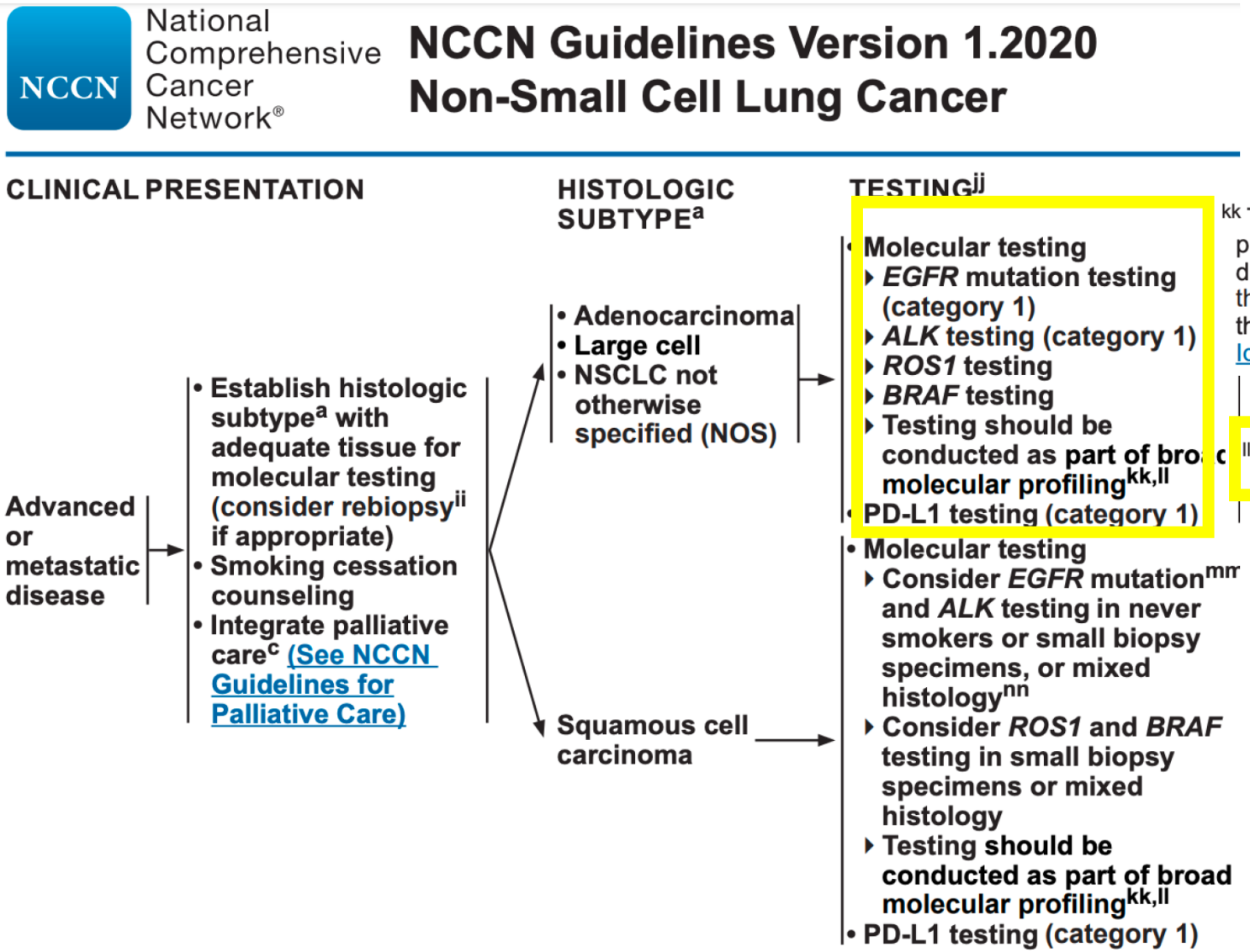
Diagnosis	Percent of patients
ACTH-dependent Cushing's syndrome	**80**
Cushing's disease	68
Ectopic ACTH syndrome	12
Ectopic CRH syndrome	<<1
ACTH-independent Cushing's syndrome	**20**
Adrenal adenoma	10
Adrenal carcinoma	8
Micronodular hyperplasia	<1
Macronodular hyperplasia	<1
Pseudo-Cushing's syndrome	
Major depressive disorder	1
Alcoholism	<<1



Molecular heterogeneity in NSCLC



Testing is key part of initial evaluation



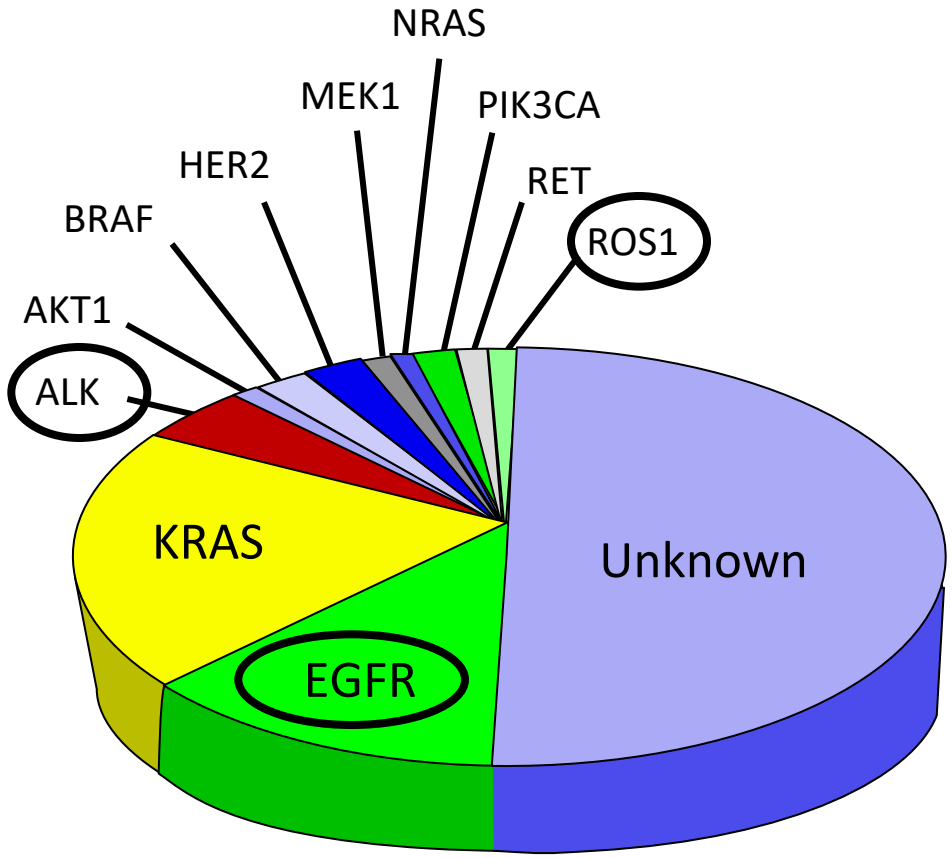
[kk] The NCCN NSCLC Guidelines Panel strongly advises broader molecular profiling with the goal of identifying rare driver mutations for which effective drugs may already be available, or to appropriately counsel patients regarding the availability of clinical trials. Broad molecular profiling is a key component of the improvement of care of patients with NSCLC. See Emerging Biomarkers to Identify Patients for Therapies (NSCL-H).

[ll] Testing should include the neurotrophin receptor kinase (NTRK) gene fusion; if positive, see NSCL-27.

Molecular testing in NSCLC

Molecular/ Genetic Abnormality	Prevalence and Clinical Association	Clinical Consequence
EGFR-activating mutations	• 10% to 15% patients with advanced NSCLC (in North America); ~50% of patients in Asia with advanced NSCLC • Adenocarcinoma histology • Never or light smokers[20-22]	**Prognosis** similar to NSCLC without actionable mutations in absence of targeted therapy[22,23] **Predictive** for improved sensitivity and outcomes with EGFR-targeted TKIs[18,25,26]
ALK rearrangements	• 2% to 7% of all NSCLC tumors[19] • Never or light smokers; more likely to be male and younger[27] • *EGFR* mutations and *ALK* rearrangements are almost always mutually exclusive[28]	**Prognosis** similar to NSCLC without actionable mutations in absence of targeted therapy[27,29] **Predictive** for improved sensitivity and outcomes with ALK-targeted TKIs[19,30,31]
KRAS mutations	• ~ 30% of lung adenocarcinomas[32,33]	Role as a **prognostic** or **predictive** factor in NSCLC remains controversial[33-36] May be negative **predictor** of response to EGFR-targeted TKIs[33]
MET/HGFR pathway alterations	• *MET* amplification in ~ 5% of lung adenocarcinoma[37] • MET amplification in ~ 20% of EGFR TKI–resistant NSCLC[38]	Potentially **prognostic** for poor survival[39] **Predictive** for and potential driver of EGFR TKI resistance[38-41]; MET amplification associated with response to crizotinib[42,43]
ROS gene rearrangements	• ~ 1% to 6% of adenocarcinomas[44,45] • Never or former light smokers	Potential oncogenic driver; may be **prognostic** for improved survival even in absence of targeted therapy[46] **Predictive** for improved sensitivity to and outcomes with crizotinib[47]
RET gene rearrangements	• RET gene rearrangements, including *KIF5B* gene and *CDD6* gene fusions[48] are found in 1% to 2% of NSCLC[49,50] • Never or former light smokers	Potential oncogenic driver[48,49] **Predictive** response to cabozantinib[51]
PD-L1 expression	• ~ 60% of NSCLC[52]	May be **prognostic** for poor survival[53] **Predictive** for response to pembrolizumab[54] and for response to nivolumab in non-squamous NSCLC[55] In both non-squamous and squamous NSCLC, nivolumab is indicated regardless of PD-L1 expression

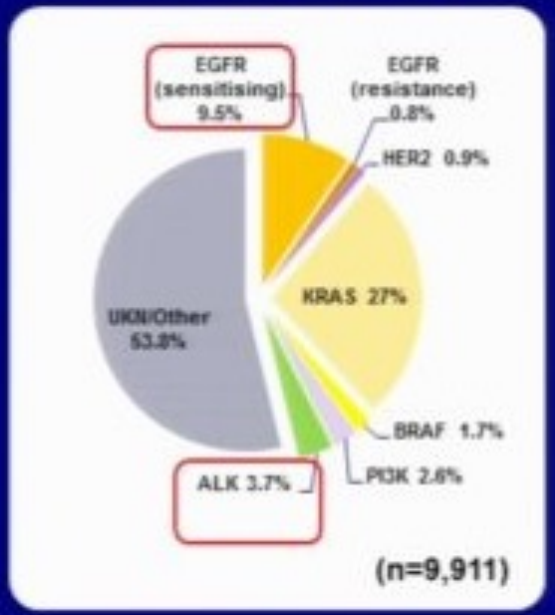
Μοριακοί υπότυποι ΜΜΚΠ βάσει οδηγών μεταλλάξεων



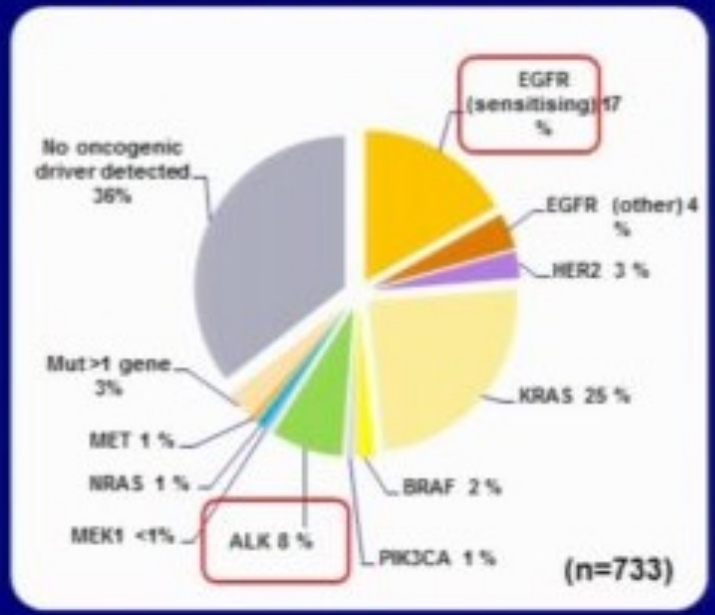
Συχνότητα οδηγών μεταλλάξεων στον ΜΜΚΠ (%)	
AKT1	1
ALK	3-7
BRAF	1-3
EGFR	10-35
HER2	2-4
KRAS	15-25
MEK1	1
NRAS	1
PIK3CA	1-3
RET	1-2
ROS1	1

EGFR mutations and ALK translocations in lung cancer

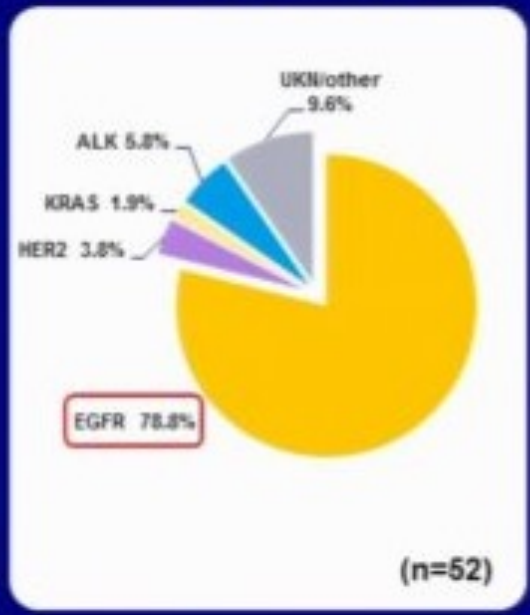
Europe[1]
All histology
(Biomarkers France)



US[2]
Adenocarcinoma
(Lung Cancer Mutation Consortium)



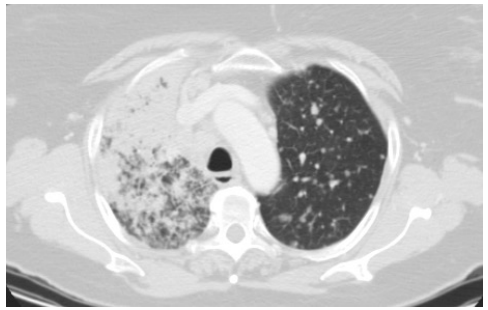
East Asia[3]
Adenocarcinoma,
never smokers



1. Barlesi, et al. ASCO 2013
2. Johnson, et al. ASCO 2013
3. Sun, et al. J Clin Oncol 2010

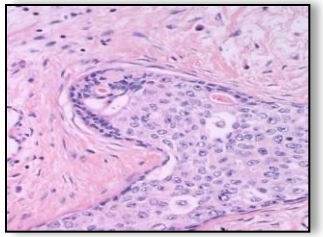
Targeted Therapy

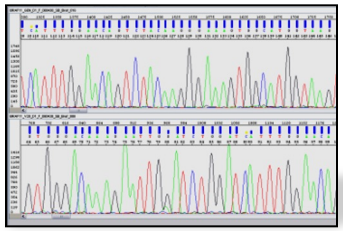
Clinical Information

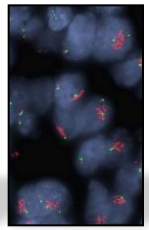


Targeted Therapy

Biopsy

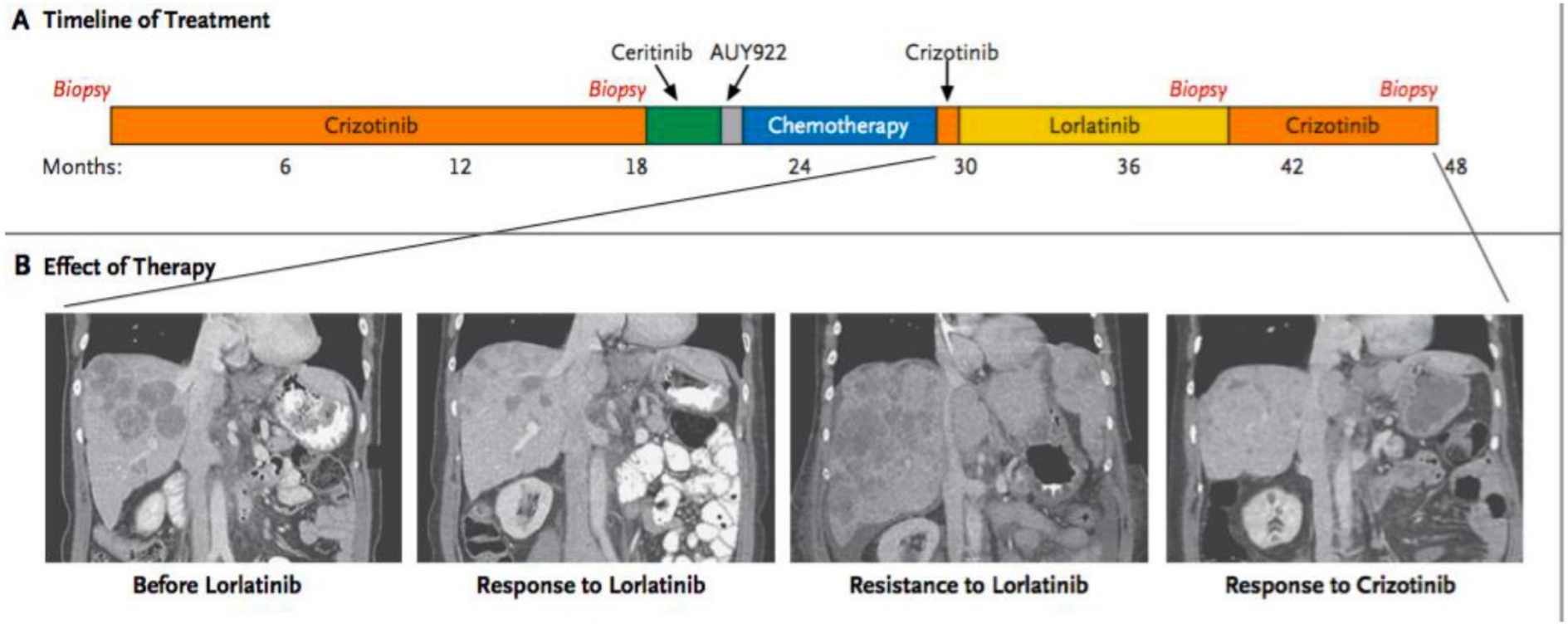






Routine and Molecular Pathology

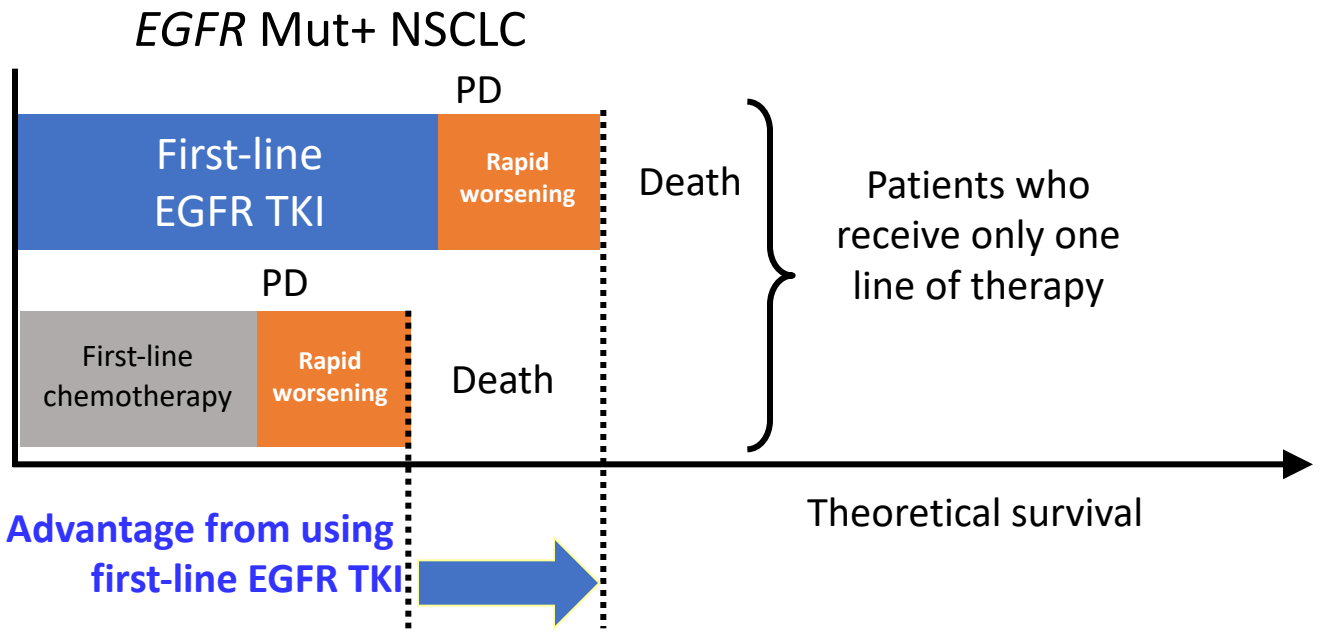
The Art of Precision Medicine



Resensitization to Crizotinib by the Lorlatinib *ALK* Resistance Mutation L1198F

Shaw et al. NEJM 2016

When is the most appropriate time to use EGFR TKIs in EGFR Mut+ NSCLC?



After 1st-line platinum-doublet chemotherapy, only 50–60% of patients receive 2nd-line therapy[1]

Delaying EGFR TKI therapy therefore risks the possibility that patients die sooner[2]

1. Stinchcombe, et al. J Thorac Oncol 2009;
2. Gridelli, et al. Lung Cancer 2011

Metastatic NSCLC: consensus on pathology and molecular tests, first-, second- and third-line therapy

- 1st ESMO Consensus Conference in Lung Cancer: Lugano 2011

12. What is the preferred first-line treatment in patients with a tumor harboring an activating EGFR mutation?

Recommendation 12: an EGFR TKI is the preferred first-line treatment in patients whose tumor harbors an activating EGFR mutation.

Strength of recommendation: A

Level of evidence: I

Felip, et al. Ann Oncol 2012

FDA approved EGFR TKIS

- Gefitinib
- Erlotinib
- Afatinib
- Osimertinib

Table 1. A personalised medicine synopsis table for metastatic NSCLC [20]

Biomarker	Method	Use	LOE, GOR
EGFR mutation	Any appropriate validated method, subject to external quality assurance	Used to select patients for EGFR TKI therapy, identifying those, with sensitising mutations, most likely to respond	V, A
ALK gene rearrangement	Any appropriate validated method, subject to external quality assurance. Standard approach has been FISH, or less often, multiplex PCR or RT-PCR. Certain IHC approaches may be used as a substitute primary test. IHC may also be used to screen patients, positive cases confirmed by an orthogonal method (FISH, PCR)	Used to select patients for ALK tyrosine kinase inhibitor therapy, identifying those, with a positive test, most likely to respond	V, A

Adapted from [20] by permission of Oxford University Press.

NSCLC, non-small-cell lung cancer; LOE, level of evidence; GOR, grade of recommendation; EGFR, epidermal growth factor receptor; TKI, tyrosine kinase inhibitor; ALK, anaplastic lymphoma kinase; FISH, fluorescence *in situ* hybridisation; PCR, polymerase chain reaction; RT-PCR, reverse transcription polymerase chain reaction; IHC, immunohistochemistry.

FDA approved ALK inhibitors

- Crizotinib
- Ceritinib,
- Alectinib,
- Brigatinib

Staging

TNM staging system

Primary Tumor (T)

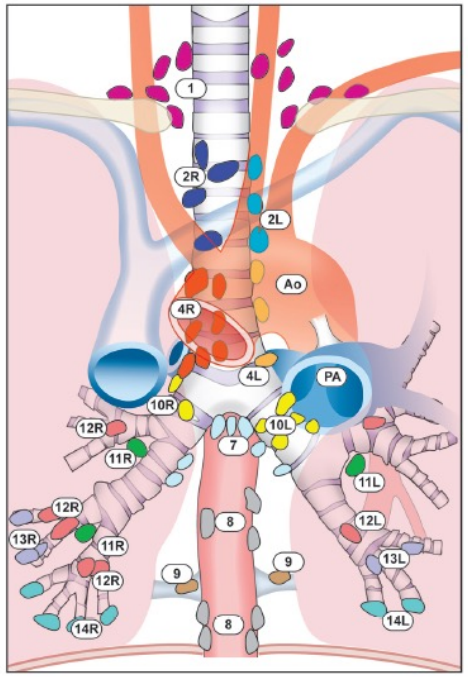
- **TX** Primary tumor cannot be assessed, or tumor proven by the presence of malignant cells in sputum or bronchial washings but not visualized by imaging or bronchoscopy
- **T0** No evidence of primary tumor
- **Tis** Carcinoma in situ
- **T1** Tumor 3 cm or less in greatest dimension, surrounded by lung or visceral pleura, without bronchoscopic evidence of invasion more proximal than the lobar bronchus (for example, not in the main bronchus)[1]
- **T1a** Tumor 2 cm or less in greatest dimension
- **T1b** Tumor more than 2 cm but 3 cm or less in greatest dimension
- **T2** Tumor more than 3 cm but 7 cm or less or tumor with any of the following features (T2 tumors with these features are classified T2a if 5 cm or less): involves main bronchus, 2 cm or more distal to the carina; invades visceral pleura (PL1 or PL2); associated with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung
- **T2a** Tumor more than 3 cm but 5 cm or less in greatest dimension
- **T2b** Tumor more than 5 cm but 7 cm or less in greatest dimension
- **T3** Tumor more than 7 cm or one that directly invades any of the following: parietal pleural (PL3), chest wall (including superior sulcus tumors), diaphragm, phrenic nerve, mediastinal pleura, parietal pericardium; or tumor in the main bronchus less than 2 cm distal to the carina[1] but without involvement of the carina; or associated atelectasis or obstructive pneumonitis of the entire lung or separate tumor nodule(s) in the same lobe
- **T4** Tumor of any size that invades any of the following: mediastinum, heart, great vessels, trachea, recurrent laryngeal nerve, esophagus, vertebral body, carina, separate tumor nodule(s) in a different ipsilateral lobe

Distant Metastasis (M)

- **M0** No distant metastasis
- **M1** Distant metastasis
- **M1a** Separate tumor nodule(s) in a contralateral lobe, tumor with pleural nodules or malignant pleural (or pericardial) effusion[2]
- **M1b** Distant metastasis (in extrathoracic organs)

ANATOMIC STAGE/PROGNOSTIC GROUPS			
Occult Carcinoma	TX	N0	M0
Stage 0	Tis	N0	M0
Stage IA	T1a	N0	M0
	T1b	N0	M0
Stage IB	T2a	N0	M0
Stage IIA	T2b	N0	M0
	T1a	N1	M0
	T1b	N1	M0
	T2a	N1	M0
Stage IIB	T2b	N1	M0
	T3	N0	M0
Stage IIIA	T1a	N2	M0
	T1b	N2	M0
	T2a	N2	M0
	T2b	N2	M0
	T3	N1	M0
	T3	N2	M0
	T4	N0	M0
	T4	N1	M0
Stage IIIB	T1a	N3	M0
	T1b	N3	M0
	T2a	N3	M0
	T2b	N3	M0
	T3	N3	M0
	T4	N2	M0
	T4	N3	M0
Stage IV	Any T	Any N	M1a
	Any T	Any N	M1b

TNM staging system



Supraclavicular zone

- **1** Low cervical, supraclavicular, and sternal notch nodes

Superior Mediastinal Nodes

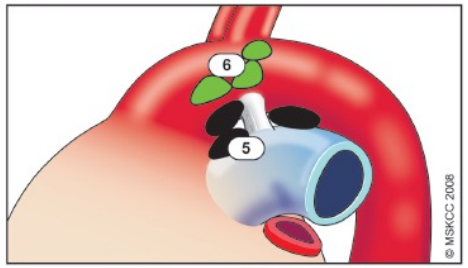
Upper zone

- **2R** Upper Paratracheal (right)
- **2L** Upper Paratracheal (left)
- **3a** Pre-vascular
- **3p** Retrotracheal
- **4R** Lower Paratracheal (right)
- **4L** Lower Paratracheal (left)

Aortic Nodes

AP zone

- **5** Subaortic
- **6** Para-aortic (ascending aorta or phrenic)



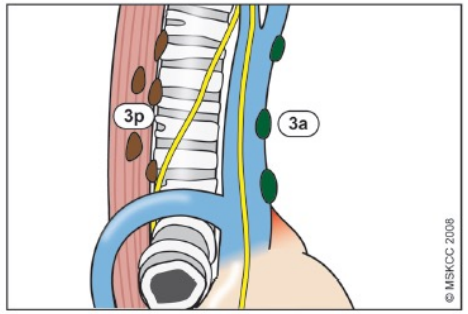
Inferior Mediastinal Nodes

Subcarinal zone

- **7** Subcarinal

Lower zone

- **8** Paraesophageal (below carina)
- **9** Pulmonary ligament



N_1 Nodes

Hilar/Interlobar zone

- **10** Hilar
- **11** Interlobar

Peripheral zone

- **12** Lobar
- **13** Segmental
- **14** Subsegmental

Regional Lymph Nodes (N)

- **NX** Regional lymph nodes cannot be assessed
- **N0** No regional lymph node metastases
- **N1** Metastasis in ipsilateral peribronchial and/or ipsilateral hilar lymph nodes and intrapulmonary nodes, including involvement by direct extension
- **N2** Metastasis in ipsilateral mediastinal and/or subcarinal lymph node(s)
- **N3** Metastasis in contralateral mediastinal, contralateral hilar, ipsilateral or contralateral scalene, or supraclavicular lymph node(s)

SCLC Diagnosis and Staging

- Diagnosis by FNA or biopsy
- Staging workup
  - CT chest/abdomen/pelvis
  - Brain MRI
  - PET scan to rule out distant metastases

- TNM staging system vs VA staging system

TNM Staging	VA Staging	Incidence, %
T1-T2, N0, M0 (stage I)	Limited stage	~ 5
T any, N any, M0 (stage I-III)	Limited stage; disease burden contained within radiation field	~ 30
T any, N any, M1 (stage IV)	Extensive stage; disease burden beyond radiation field	~ 65

Kalemkerian. Cancer Imaging. 2011;11:253. Alvarado-Luna. Transl Lung Cancer Res. 2016;5:26. Sabari. Nat Rev Clin Oncol. 2017;14:549.

Staging of SCLC

- The prognosis of SCLC strongly depends on the stage of the disease[1]
- The most common staging system is the VALG staging system. It classifies patients into two clinical categories[1,2]
- SCLC is also staged using the TNM system[1,3]

Stage	T	N	M
IA	T1mi,a,b,c	N0	M0
IB	T2a	N0	M0
IIA	T2b	N0	M0
IIB	T1a,b,c T2a,b T3	N1 N1 N0	M0 M0 M0
IIIA	T1a,b,c,T2a,b T3 T4	N2 N1 N0, N1	M0 M0 M0
IIIB	T1a,b,c,T2a,b T3,T4	N3 N2	M0 M0
IIIC	T3,T4	N3	M0
IVA	Any T	Any N	M1a,b
IVB	Any T	Any N	M1c

LS-SCLC
(30% to 40% patients; confined to ipsilateral hemithorax or tolerable radiation field)

ES-SCLC
(60% to 70% patients; extends beyond ipsilateral hemithorax)

1. Kahnert K et al. *Clin Lung Cancer*. 2016;17(5):325–333. **2.** Oronsky B et al. *Neoplasia.* 2017;19(10):842–847. **3.** NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Small Cell Lung Cancer. V3.2020. © National Comprehensive Cancer Network, Inc. 2019. All rights reserved. Accessed February 27, 2020. To view the most recent and complete version of the guideline, go online to NCCN.org. **4.** Cascone T et al. Small cell carcinoma of the lung. In: Kantarjian M, Wolff RA, eds. *The MD Anderson Manual of Medical Oncology*. 3rd ed. New York, NY: McGraw-Hill Education; 2016.

Screening in heavy smokers

American Cancer Society guidelines recommend yearly lung cancer screening for people who meet certain criteria that put them at higher risk for developing the disease. These higher risk patients are aged 55 to 74 years and are in fairly good health, currently smoke or have quit within the past 15 years, and have a smoking history equivalent to a pack a day for 30 years.

For these higher risk patients, the recommendations also say they need to:

- receive smoking cessation counseling if they are still smoking.
- be involved in shared decision-making about the benefits, limitations, and harms of screening.
- have access to a high-volume, high-quality lung cancer screening and treatment center.

Questions & Answers