NCCN 2025
Lung Cancer Congress

Biomarker Testing in Non-Small Cell Lung Cancer: What's New and What's Next

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NCCN National Comprehensive Cancer Network®

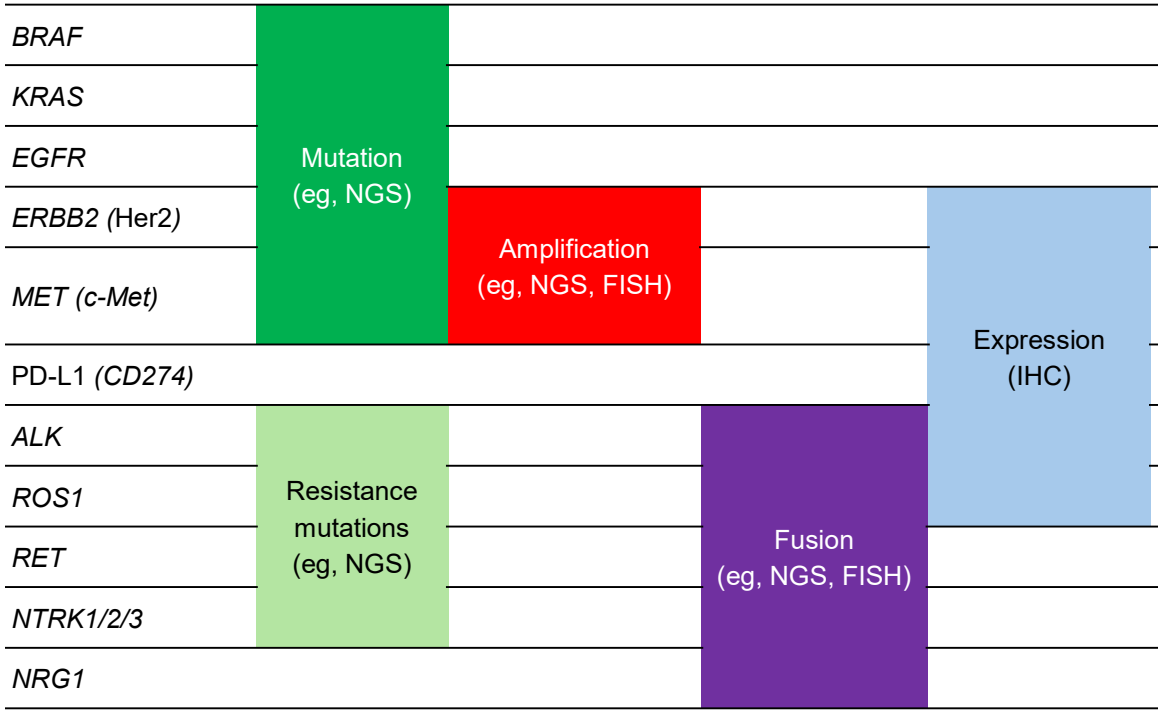
Overview

- Brief review of established predictive genetic biomarkers
- Gene targets and assays for recent therapy approvals
- NCCN Emerging Biomarkers
- Biomarkers with *possible* significance for prognosis, ADC benefit prediction, and resistance to established therapies
- Liquid biopsy strategies and use cases in NSCLC
- Applications of AI and machine vision
- Barriers to comprehensive biomarker testing

Scope

- Emphasis on individual rather than composite biomarkers
- Emphasis on molecular genetic rather than expression-based biomarkers

NCCN Recommended NSCLC Biomarkers



	NCCN Category of Evidence for Testing		
		Metastasis	
	Pre-adjuvant	SCC	Non-SCC
BRAF			2A
KRAS			2A
EGFR	2A	2A	1
ERBB2 (Her2)		2A	
MET (c-Met)		2A for ex14	2A for ex14 & SP44
PD-L1 *(CD274)*	2A		
ALK	2A	2A	1
ROS1		2A	2A
RET			1
NTRK1/2/3		2A	2A
NRG1		2A	2A

The NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Non-Small Cell Lung Cancer (V1.2026).

Common molecular genetic assays

Single or limited analyte

- Sanger sequencing
- Pyrosequencing
- Tailored NGS panels (e.g., amplicon-based)
- PCR-based genotyping e.g.,
  - Allele-specific
  - Droplet digital
- Fluorescence in situ hybridization (FISH)

Comprehensive Genomic Profiling

- Broad profiling of genetic abnormalities from DNA +/- RNA
- Large multi-gene captures (>100) to whole exome or transcriptomes
- Germline comparison sequencing available from some laboratories

PRINCIPLES OF BIOMARKER-DIRECTED THERAPY FOR ADVANCED OR METASTATIC DISEASE

***EGFR* Exon 19 Deletion or L858R Mutation**
- First-line therapy
  - Afatinib[1]
  - Erlotinib[2]
  - Dacomitinib[3]
  - Gefitinib[4,5]
  - Osimertinib[6]
  - (Carboplatin or Cisplatin)/ Osimertinib/Pemetrexed (nonsquamous)[7]
  - Erlotinib + Ramucirumab[8]
  - Erlotinib + Bevacizumab (nonsquamous)[9]
  - Lazertinib + Amivantamab-vmjw[10]
  - Lazertinib[10]
- Subsequent therapy
  - Osimertinib[11]
  - Carboplatin/Pemetrexed + Amivantamab-vmjw (nonsquamous)[12]
  - Datopotamab deruxtecan-dlnk (nonsquamous)[13]
  - Lazertinib + Amivantamab-vmjw[14,15]

***EGFR* S768I, L861Q, and/or G719X Mutations**
- First-line therapy
  - Afatinib[1,16]
  - Erlotinib[2]
  - Dacomitinib[3]
  - Gefitinib[4,5]
  - Osimertinib[6,17]

***EGFR* S768I, L861Q, and/or G719X Mutations** (continued)
- Subsequent therapy
  - Osimertinib[11]
  - Datopotamab deruxtecan-dlnk (nonsquamous)[13]

***EGFR* Exon 20 Insertion Mutation**
- First-line therapy
  - Carboplatin/Pemetrexed + Amivantamab-vmjw (nonsquamous)[18]
- Subsequent therapy
  - Amivantamab-vmjw[19]
  - Sunvozertinib[20]
  - Datopotamab deruxtecan-dlnk (nonsquamous)[13]

***KRAS* G12C Mutation**[a]
- Subsequent therapy
  - Sotorasib[21]
  - Adagrasib[22]

***ALK* Gene Fusion**
- First-line therapy
  - Alectinib[23,24]
  - Brigatinib[25]
  - Ceritinib[26]
  - Crizotinib[23,27]
  - Ensartinib[28]
  - Lorlatinib[29]

***ALK* Gene Fusion** (continued)
- Subsequent therapy
  - Alectinib[30,31]
  - Brigatinib[32]
  - Ceritinib[33]
  - Ensartinib[34]
  - Lorlatinib[35]

***ROS1* Gene Fusion**
- First-line therapy
  - Crizotinib[36]
  - Entrectinib[37]
  - Repotrectinib[38]
  - Taletrectinib[39]
- Subsequent therapy
  - Lorlatinib[40]
  - Entrectinib[37]
  - Repotrectinib[38]
  - Taletrectinib[39]

***BRAF* V600E Mutation**
- First-line therapy
  - Dabrafenib/Trametinib[41]
  - Binimetinib/Encorafenib[42]
  - Dabrafenib[43]
  - Vemurafenib
- Subsequent therapy
  - Dabrafenib/Trametinib[43,44]
  - Binimetinib/Encorafenib[42]

***NTRK1/2/3* Gene Fusion**
- First-line/Subsequent therapy
  - Larotrectinib[45]
  - Entrectinib[46]
  - Repotrectinib[47]

***MET* Exon 14 Skipping Mutation**[a]
- First-line therapy/Subsequent therapy
  - Capmatinib[48]
  - Crizotinib[49]
  - Tepotinib[50]

***RET* Gene Fusion**
- First-line therapy
  - Selpercatinib[51]
  - Pralsetinib[52]
- Subsequent therapy
  - Cabozantinib[53,54]

***ERBB2 (HER2)* Mutation**
- Subsequent therapy
  - Fam-trastuzumab deruxtecan-nxki[a,55]
  - Ado-trastuzumab emtansine[a,56]
  - Zongertinib[57]

***NRG1* Gene Fusion**
- Subsequent therapy
  - Zenocutuzumab-zbco[58]

HER2-positive IHC 3+
- Subsequent therapy
  - Fam-trastuzumab deruxtecan-nxki[59]

HGFR (MET) (≥50% IHC 3+ and *EGFR* wild-type)
- Subsequent therapy
  - Telisotuzumab vedotin-tllv (nonsquamous)[60]

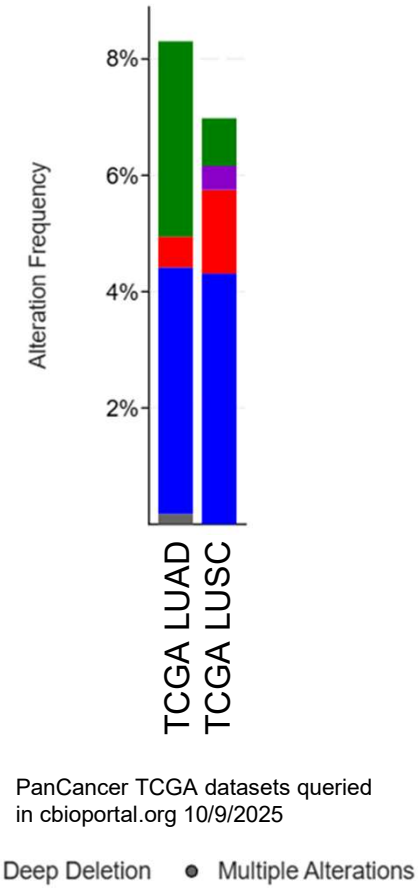
Biomarkers for Recent FDA Drug Approvals & NCCN Emerging Biomarkers

Her2 overexpression

- Associated with inferior prognosis[2]
- Correlates poorly with *ERBB2* amplification status[2]
- Fam-trastuzumab deruxtecan-nxki: FDA approved on 4/5/24 for metastatic tumors with Her2 IHC of 3+
  - Recommended by NCCN[1] for patients with 3+ IHC, citing DESTINY-Lung01[3] (although this trial included both 2+ and 3+ scores)

NRG1

- Neuregulin 1 proto-oncogene
  - Growth factor ligand for Her3 RTK
- Binding initiates heterodimerization with Her2 and activation of MAPK and PI3K/MTOR pathways



NRG1 fusion prevalence in NSCLC

TCGA

- 4 of 1053 samples (~0.4%) of NSCLC in TCGA data
  - *SDC::NRG1* 1 LUSC, 1 LUAD
  - *SMAD4::NRG1* 1 LUSC
  - *THAP7::NRG1* 1 LUSC

Queried from cBioportal.org[5-7] on 10/02/2025, see https://bit.ly/3Ky4q10

Bastard C, et al. 2025

- Observed by FISH in approximately 1% (N=4/446) of lung adenocarcinoma
- 2% (N=4/191) of cases with no alterations in *EGFR, KRAS, BRAF, HER2, MET, ALK, ROS1* and *RET*

Bastard C, et al. Cancers (Basel). 2025 Jul 15;17(14):2347. doi: 10.3390/cancers17142347. PMID: 40723230

NRG1 fusions detected by breakapart FISH

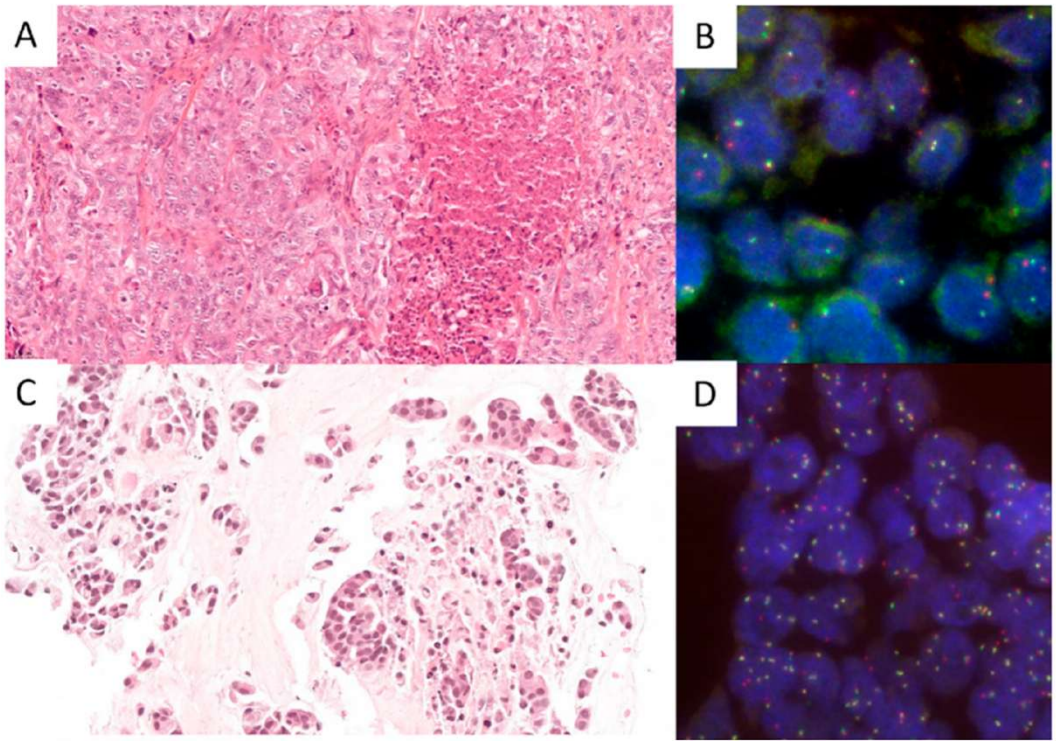


Figure adapted from Bastard C, et al. Cancers (Basel). 2025 Jul 15;17(14):2347. doi: 10.3390/cancers17142347. PMID: 40723230; PMCID: PMC12294116. Published under CC BY 4.0 license (https://creativecommons.org/licenses/by/4.0/)

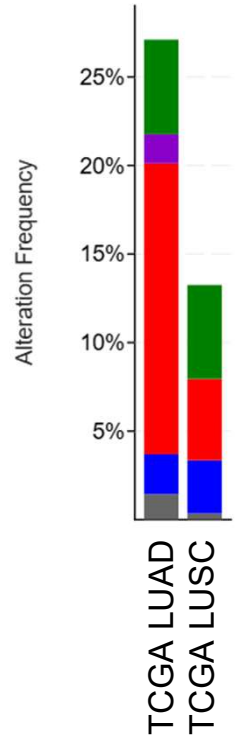
Zenocutuzumab-zbco: eNRGy phase 2

- *NRG1* fusions identified by NGS
- All tumors ORR 30% (95% CI 23-37), N=158
- NSCLC ORR 29% (95% CI 20-39), N=93
- FDA approval on 12/4/24 for *NRG1* fusion-positive NSCLC and pancreatic adenocarcinoma

Schram AM, et al. Efficacy of Zenocutuzumab in *NRG1* Fusion-Positive Cancer. N Engl J Med. 2025 Feb 6;392(6):566-576. doi: 10.1056/NEJMoa2405008. PMID: 39908431; PMCID: PMC11878197.

FGFR1, 2, 3, 4

- Fibroblast growth factor receptor family
  - Proto-oncogene RTKs
  - Activate PI3K/MTOR and MAPK pathways



PanCancer TCGA datasets queried in cbioportal.org 10/9/2025

Mutation Structural Variant Amplification Deep Deletion Multiple Alterations

Erdafitinib; RAGNAR phase 2 trial

All tumors, N=217

- ORR 30% (95% CI 24-36)
- *FGFR3* alterations/fusions most common
  - *FGFR3*::*TACC3*, N=49 (23%)
  - *FGFR3* p.S249C, N=25 (12%)
  - *FGFR2*::*BICC1*, N=12 (6%)
  - *FGFR2* p.Y375C, N=8 (4%)
  - *FGFR2*::*TACC2*, N=7 (3%)

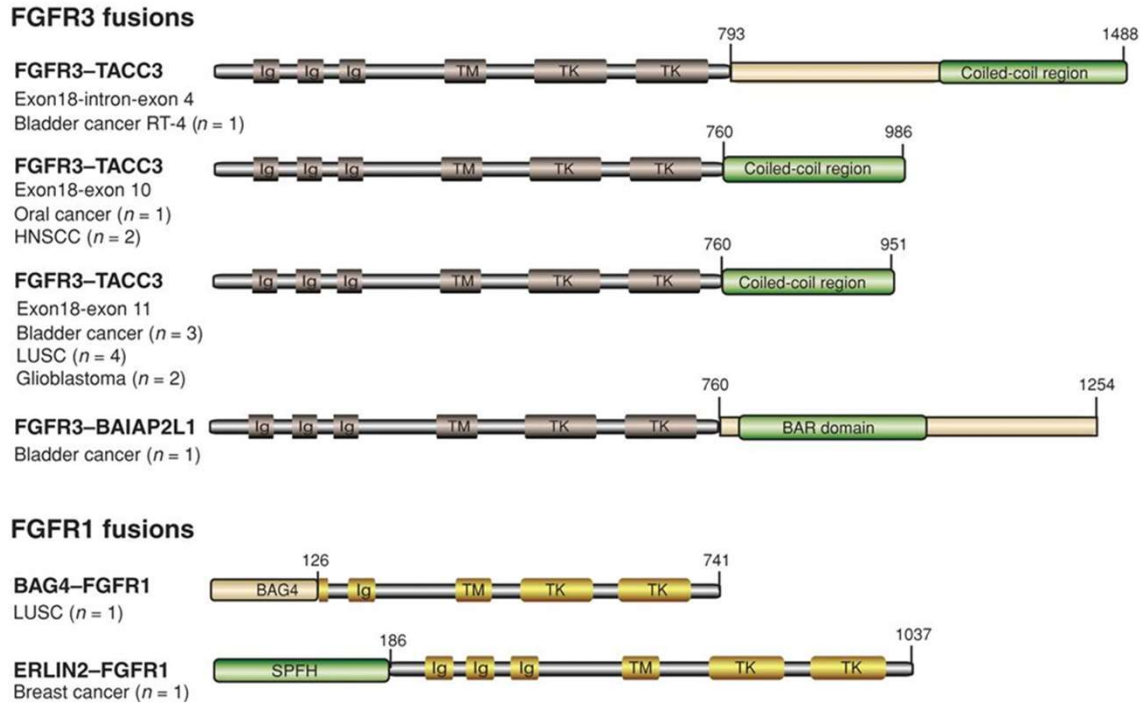
NSCLC, N=23

- Squamous ORR 21% (CI 5-51)
  - *FGFR3* predominates
- Nonsquamous ORR 33% (CI 8-70)
  - Balanced between *FGFR2* and *3*, fusions predominate

Pant S, et al. Lancet Oncol. 2023 Aug;24(8):925-935. PMID: 37541273

AACR American Association for Cancer Research

Adapted From: **Identification of Targetable FGFR Gene Fusions in Diverse Cancers**
Cancer Discov. 2013;3(6):636-647. doi:10.1158/2159-8290.CD-13-0050



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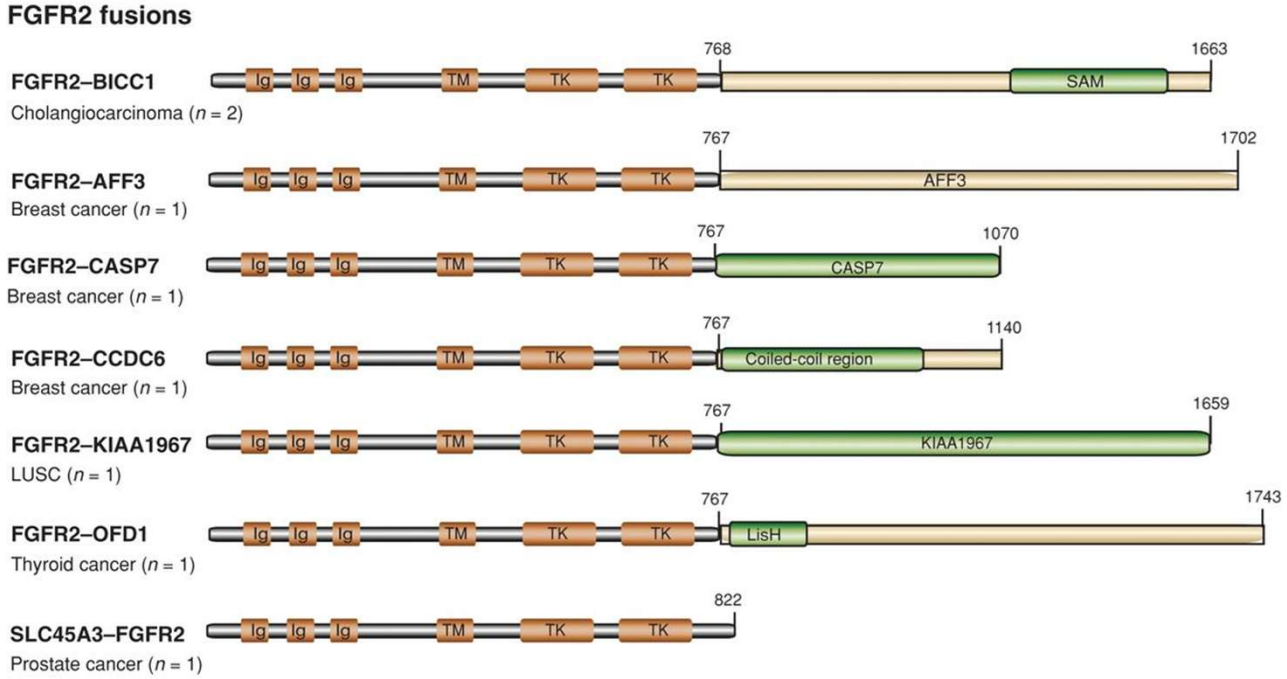
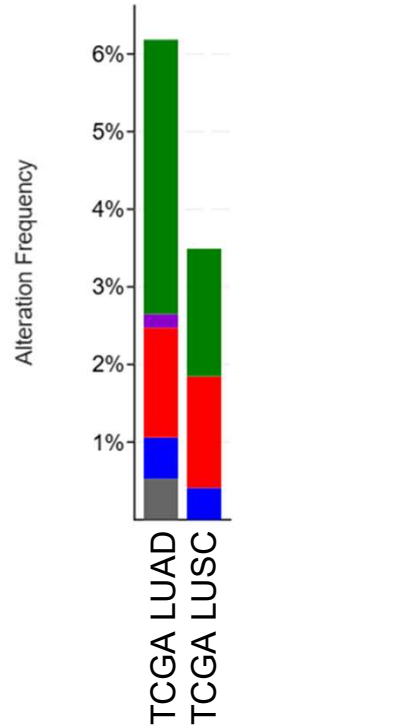
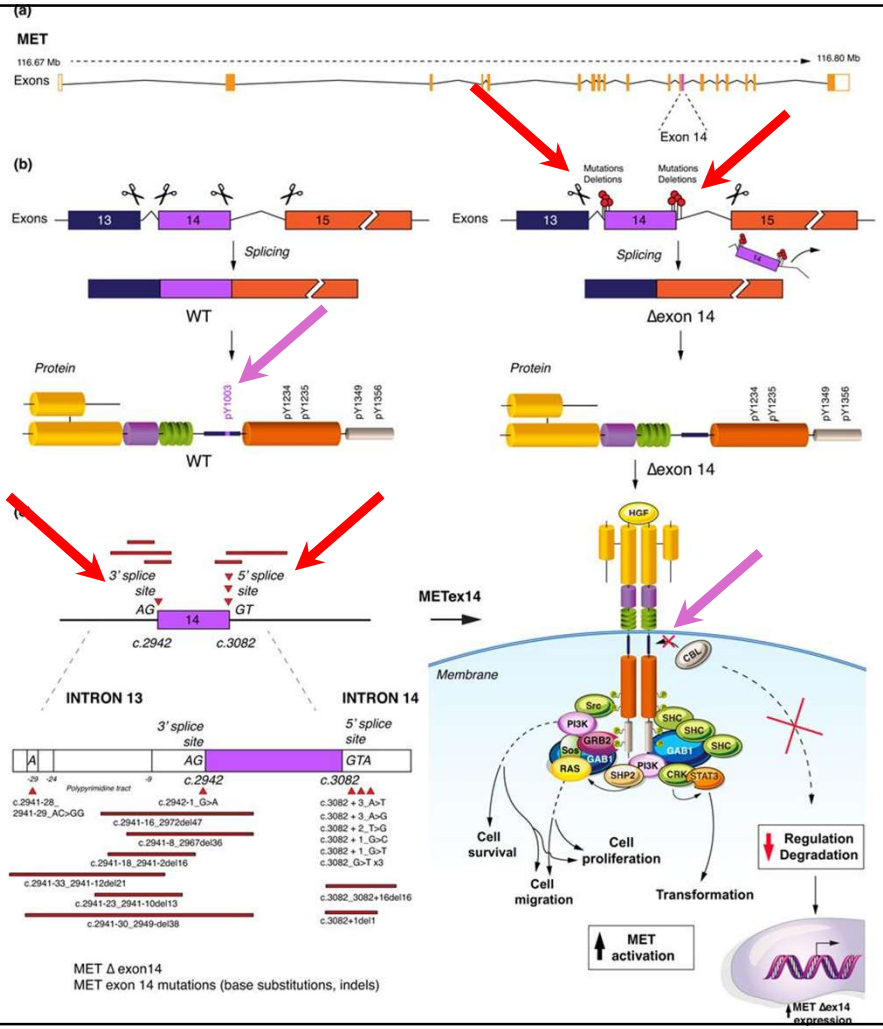


MET

- "Mesenchymal Epithelial Transition" proto-oncogene, aka. *c-MET*
  - RTK for hepatocyte growth factor (HGF)
  - Activates PI3K/MTOR and MAPK pathways
- Exon 14-skipping mutation status is an established biomarker for response to capmatinib, crizotinib, and tepotinib
- High-level amplification is linked to inferior prognosis[11, 12]





MET exon 14 skipping mutations

Figure credit: Friedlaender A, Drilon A, Banna GL, Peters S, Addeo A. Cancer. 2020 Nov 15;126(22):4826-4837. doi: 10.1002/cncr.33159. Epub 2020 Sep 5. PMID: 32888330

Telisotuzumab vedotin: LUMINOSITY ph 2

- 172 patients with nonsquamous *EGFR*-wt NSCLC with c-Met overexpression by IHC (SP-44)
  - High: >50% of tumor with IHC 3+
  - Intermediate: 25%-50% of tumor with IHC 3+
- Primary endpoint: ORR
  - c-Met high: 34.6% (95% CI 24.2-46.2, N=78)
  - c-Met intermediate: 28.6% (95% CI 21.7-36.2, N=83)
  - PFS 45.8 and 50.1 m, respectively; OS 14.6 and 14.2 m
- FDA approval for c-Met high via SP44 (5/24/2025)

Camidge DR, et al. J Clin Oncol. 2024 Sep 1;42(25):3000-3011. PMID: 38843488

SP44 MET immunohistochemistry

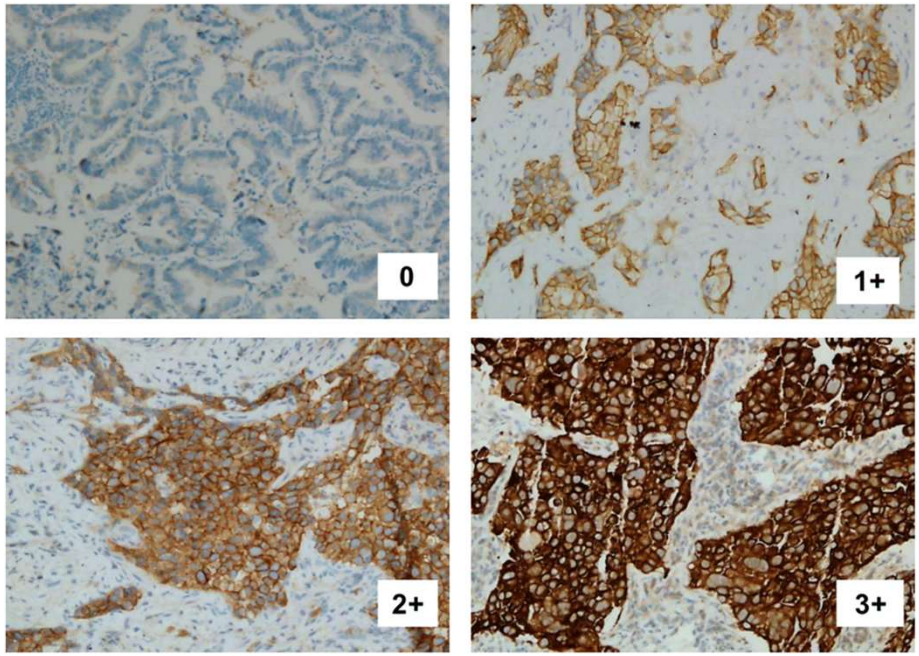


Figure source: Chen RL, et al. BMC Cancer. 2018 Nov 26;18(1):1171. PMID: 30477470; PMCID: PMC6258481. CC BY 4.0 (http://creativecommons.org/licenses/by/4.0/)

MET expression: more than mutations

- Guo, et al., analyzed Met expression in 181 patients with NSCLC
- MET IHC was positive in 39% (N=71)
- Of the 71 MET positive patients, only 3 demonstrated a genetic basis
  - 1 *MET* amplified
  - 2 *MET*ex14 mutation

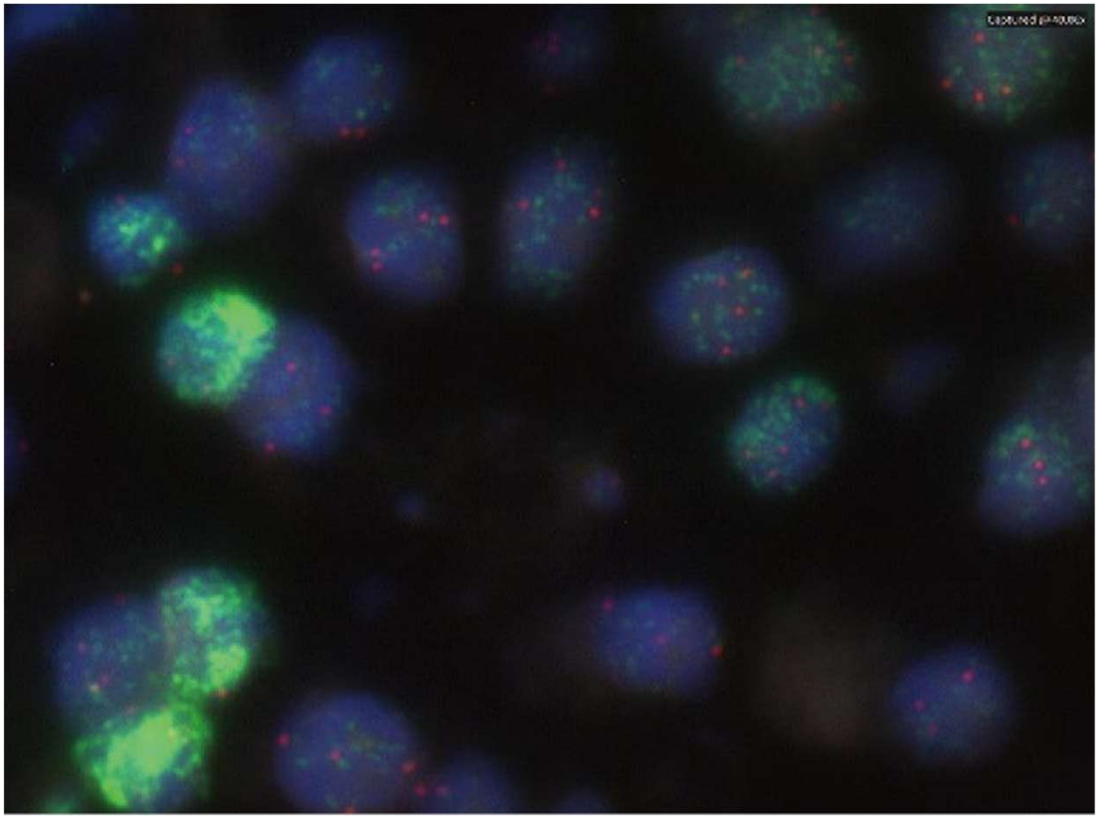
Guo R, et al. J Thorac Oncol. 2019 Sep;14(9):1666-1671. PMID: 31228623

High-level *MET* Amplification

	Assay	Threshold	Agent	ORR
Ou 2011[17]	FISH	**MET:CEP7 > 5**	Crizotinib	100% (N=1)
Camidge 2021[18]	FISH	**MET:CEP7 > 4**	Crizotinib	38.1% (N=21)
Wolf 2020[19]	FISH	**Copies >= 10**	Capmatinib	(29% in prev. treated, N=69) 40% tx naïve (N=15)
Le 2023[20]	ctDNA	**Copies >= 2.5**	Tepotinib	41.7% (N=24)

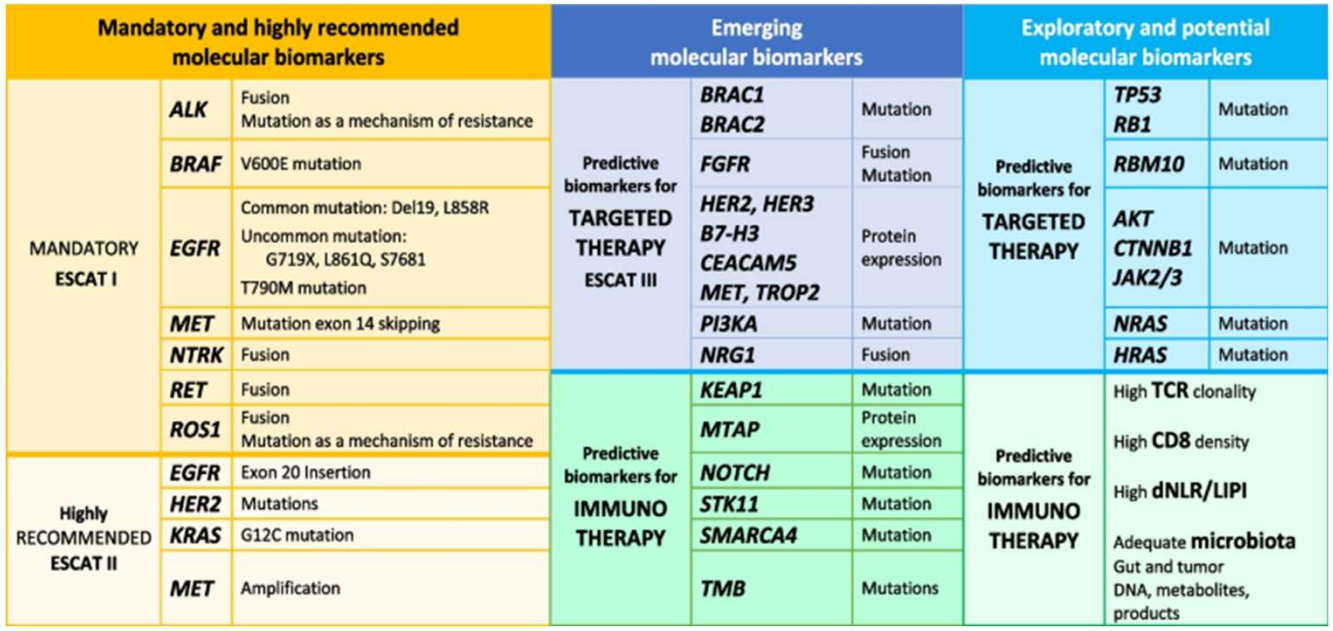
MET amplification by FISH

MET: Green probe
CEP7: Red probe



Champagnac A, et al. J Thorac Dis. 2020 May;12(5):2172-2178. PMID: 32642122. Published under CC BY-NC-ND 4.0 License (https://creativecommons.org/licenses/by-nc-nd/4.0/)

Additional Emerging and Exploratory biomarkers



Mandatory and highly recommended molecular biomarkers			Emerging molecular biomarkers			Exploratory and potential molecular biomarkers		
MANDATORY ESCAT I	*ALK*	Fusion Mutation as a mechanism of resistance	Predictive biomarkers for TARGETED THERAPY ESCAT III	*BRAC1* *BRAC2*	Mutation	Predictive biomarkers for TARGETED THERAPY	*TP53* *RB1*	Mutation
	BRAF	V600E mutation		*FGFR*	Fusion Mutation		*RBM10*	Mutation
	EGFR	Common mutation: Del19, L858R Uncommon mutation: G719X, L861Q, S7681 T790M mutation		*HER2, HER3* *B7-H3* *CEACAM5* *MET, TROP2*	Protein expression		*AKT* *CTNNB1* *JAK2/3*	Mutation
	MET	Mutation exon 14 skipping		*PI3KA*	Mutation		*NRAS*	Mutation
	NTRK	Fusion		*NRG1*	Fusion		*HRAS*	Mutation
	RET	Fusion	Predictive biomarkers for IMMUNO THERAPY	*KEAP1*	Mutation	Predictive biomarkers for IMMUNO THERAPY	High **TCR** clonality	
	ROS1	Fusion Mutation as a mechanism of resistance		*MTAP*	Protein expression		High **CD8** density	
Highly RECOMMENDED ESCAT II	*EGFR*	Exon 20 Insertion		*NOTCH*	Mutation		High **dNLR/LIPI**	
	HER2	Mutations		*STK11*	Mutation			
	KRAS	G12C mutation		*SMARCA4*	Mutation		Adequate **microbiota** Gut and tumor DNA, metabolites, products	
	MET	Amplification		*TMB*	Mutations			

Hofman P, et al. Virchows Arch. 2024 Feb;484(2):233-246. PMID: 37801103; PMCID: PMC10948551.
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Emerging Biomarkers: A Limited Survey

Expression status (e.g., IHC)	**Genetic status (e.g., NGS)**
HER3	*KEAP1*
B7-H3	*MTAP*
CEACAM5	*RBM10*
TROP2	*SMARCA4*

HER3

- RTK proto-oncogene of the ERBB family altered by mutation or amplification
- Forms heterodimers with other ERBB family members, especially ERBB2 (HER2)
- Activates MAPK, PI3K/MTOR pathways among others
- Frequently overexpressed in NSCLC[23, 24]
- Proposed mechanism of resistance to EGFR, ALK TKIs[25-28]
- May be targetable by investigational monoclonal antibodies (lumretuzumab, seribantumab, patritumab) and ADCs (patritumab-DXd)

B7-H3 (CD276)

- Immune checkpoint molecule and proto-oncogene in the B7 immunoglobulin superfamily
- Overexpression linked to inferior prognosis[29-31]
- Contributes to immune evasion, inhibits apoptosis[32-34]
- May predict of ICI resistance[30, 33]
- Therapeutic strategies include CAR-T, BiKE, and ADC[35]

CEACAM5 (CD66e)

- Carcinoembryonic antigen-related cell adhesion molecule 5
- Cell-surface glycoprotein overexpressed in 20-25% of NSCLC[36]
- May enhance cell proliferation and migration through p38 pathway[37]
- Therapeutic strategies include investigational bispecific antibodies, CAR-T, ADCs (tusamitamab ravtansine)[36, 38]

TROP2 (*TACSTD2*)

- Trophoblast cell-surface antigen 2
  - Transmembrane calcium signal transducer proto-oncogene
  - Overexpressed in NSCLC[39, 40]
  - Promotes cell proliferation via MAPK, PI3K/MTOR pathways
- Overexpression linked to ICI resistance[41-42]
- Target for ADCs, including Sacituzumab govitecan[43-45] and datopotamab deruxtecan (TROPION-Lung01, 05, 07/08)[46-48]

KEAP1

- Tumor suppressor that depresses intracellular NRF2 levels
  - NRF2 otherwise activates antioxidant and cellular protective pathways
- Mutation associated with inferior prognosis[49-52]
- Also linked to resistance to chemoradiation, TKI, and ICI[53-60]
- Possible therapeutic strategies include glutaminase and mTOR pathway inhibition[60]

MTAP

- Methylthioadenosine phosphorylase, tumor suppressor in the methionine salvage pathway
- Loss of function (e.g., deletion) leads to accumulation of 2-methylthioadenosine (MTA)
- Inferior prognosis, especially when codeleted with CDKN2A (p16)[61]
- Resistance to immunotherapy[62]
- Exploratory therapeutic strategies include MAT2A and PRMT5 inhibition[63-65]

RBM10

- RNA-binding motif protein 10, tumor suppressor
- Mutation/loss of expression may portend inferior prognosis, particularly with *TP53* and *KRAS* comutation[66-69]
- Associated with late resistance to EGFR TKIs[70, 71]
- Spliceosome inhibitors[71] and WEE1 kinase inhibitors[72] may offer new therapeutic strategies

SMARCA4

- Encodes the BRG1 subunit of the SWI/SNF chromatin remodeling complex, tumor suppressor
- Mutation associated with inferior prognosis[73-76] and response to chemotherapy[75, 77-79]
- Therapeutic strategies include:
  - SMARCA2 inhibition[76]
  - AURKA inhibition[80]
  - CDK4/6 inhibition[81]

Liquid Biopsy

Liquid biopsy: sample types, analytes, assays

Sample types

- Blood/plasma
- CSF
- *Sputum*
- *Effusions*
- *Bone marrow*

Plasma analytes

- ctDNA
- *cfRNA*
- *Methyl-ctDNA*
- *Proteins*
- *Exosomes*
- *Tumor-educated platelets*

Buffy coat

- Circulating tumor cells
- *Circulating endothelial cells, fibroblasts*
- *Immune cells*

Assays in Current Use

- PCR (e.g., ddPCR)
- NGS (e.g., DNA)

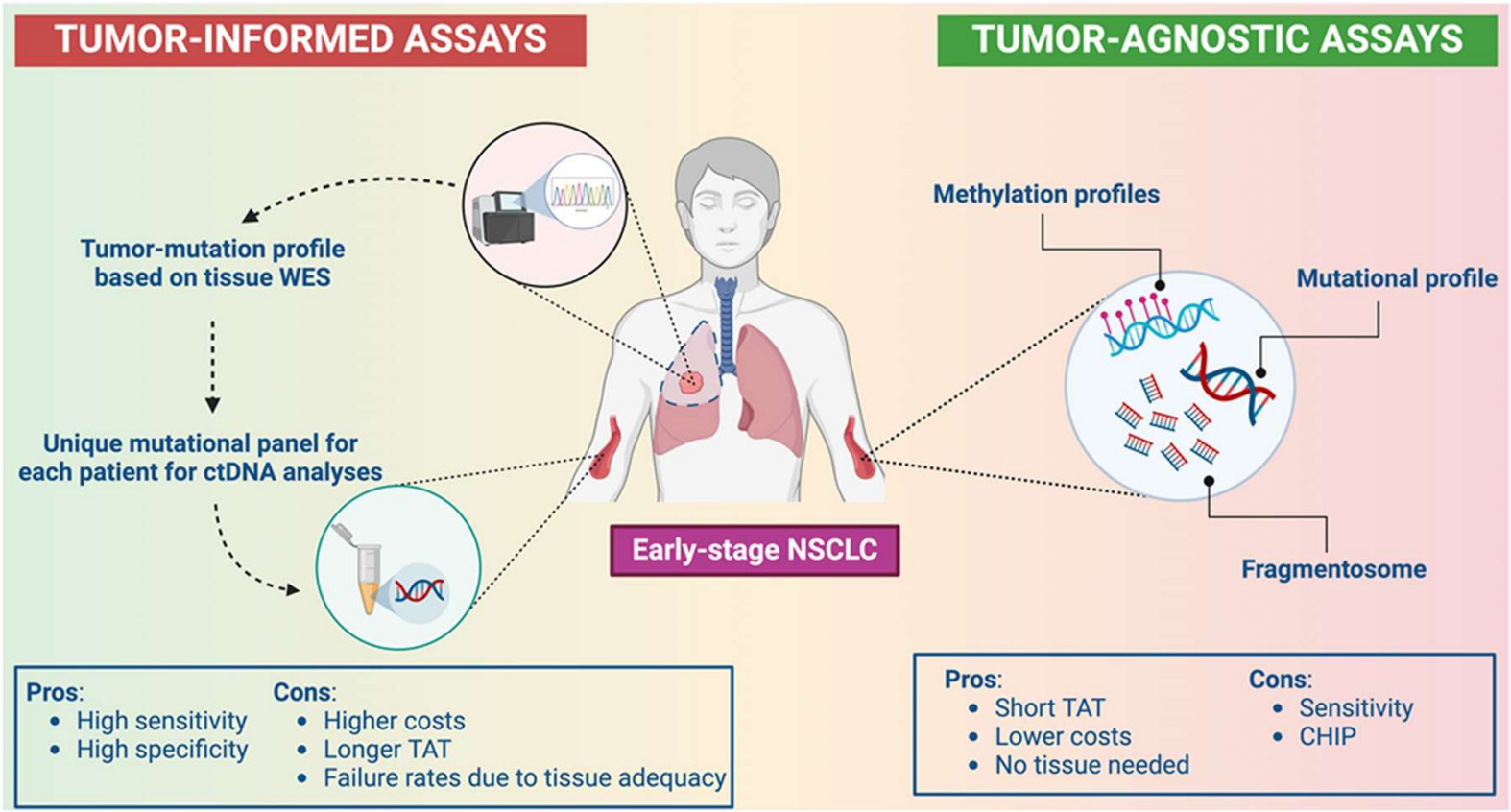


Figure source: Passarella G, et al. Discov Oncol. 2025 Jan 29;16(1):100. doi: 10.1007/s12672-025-01826-7. PMID: 39881042; PMCID: PMC11780067. CC BY-NC-ND 4.0 (http://creativecommons.org/licenses/by-nc-nd/4.0/)

ctDNA versus Traditional Tissue Biopsy

ctDNA

- May better reflect genetic heterogeneity across multiple tumor sites
- Potential for false positive mutations from hematopoietic cells
- Sensitivity depends on tendency of a particular tumor to shed DNA

Tissue

- Combines molecular data with histologic typing, grade/stage, etc
- Represents only a snapshot of the molecular state of the tumor
- Sensitivity depends on adequate tumor purity for detection of mutation

ctDNA Recommendations

NCCN (2A) [v1.2026, NSCL-H 7]

- Should not be used in lieu of tissue diagnosis
- Not recommended in stages I-III
- High specificity but “appreciable” false-negative rate
  - Complementary to tissue-based sequencing

IASLC (2021)

- “For patients with oncogene-addicted NSCLC progressing after a targeted therapy,... [a] “plasma first” approach should be considered as a standard of care in this setting."

Rolfo C, et al. J Thorac Oncol. 2021 Oct;16(10):1647-1662. PMID: 34246791.

Additional considerations

- Variable concordance between tissue and ctDNA mutation detection across studies[83-85]
  - Better with advanced disease and higher ctDNA fraction in blood
- Some support the value of co-testing ctDNA and tissue[86, 87] for highest possible hit-rate of targetable mutations and increased Quality-Adjusted Life-Years[88]

Liquid biopsy: positivity and purpose

Definitions of positivity

- Predictive oncogenic mutations (drivers) characteristic of the tumor (e.g. EGFR mutation)
- Drivers that are not specific to a single tumor and not therapeutically predictive (e.g. TP53)
- Non-oncogenic/driver alterations (passengers)

Purpose

- Targeted therapy selection
- Prognosis
- Minimal residual disease
- Adjuvant therapy decision-making
- Response monitor

Role of ctDNA Prognostic or Predictive?

Prognosis

- Numerous studies support the prognostic value of ctDNA for disease/recurrence-free survival after curative-intent therapy[89-102]

Predictive

- Predictive value of ctDNA testing as yet undetermined

ctDNA Predictive Studies in Progress

Pending publication

- MERMAID-1 (NCT04385368)
- MERMAID-2 (NCT04642469)

Recruiting

- ADAPT-E (NCT04585477)
- NCT04585490

Artificial Intelligence

Digital pathology advances

The confluence of artificial intelligence and whole-slide imaging may generate clinically relevant data beyond histologic diagnosis:

- Prediction of molecular biomarkers (e.g., *EGFR* mutation) based on WSI morphology[103-105]
- Evaluation of PD-L1 status[106]
- Evaluation of tumor-infiltrating lymphocytes and other features of the microenvironment[107-109]
- Prognosis/recurrence after surgery or RT[110, 111]

Multianalyte profiling and AI classifiers

Disease subtyping and prognostication algorithms can be trained on transcriptional or methylation profiling data, NGS or microarray

- A few examples in clinical translation:
  - Noninvasive detection of NSCLC using circulating oncRNA[112]
  - 27 gene RT-PCR predictor of ICI benefit in NSCLC[113]
  - Full transcriptome classifier for cancer of unknown primary[114]
- Limitations:
  - Availability limited to specific companies, laboratories
  - High tissue input requirements and assay cost for array, NGS
  - May require large (potentially expensive) datasets for training and validation (including prospective clinical trials)

Five key challenge areas and recommendations for achieving comprehensive biomarker testing

Challenge 1. Knowledge gaps regarding the value and indications for testing	Recommendation 1. Disseminate clear and consistent educational materials for biomarker testing to physicians and patients.
Challenge 2. Procuring adequate tissue for evaluation	Recommendation 2. Provide education to proceduralists performing tissue sampling for potential lung cancer and guide the development of institutional policies promoting adequate tissue collection.

Table adapted from: Fox AH, etc. Cancer. 2024 Dec 15;130(24):4188-4199. PMID: 39347617.

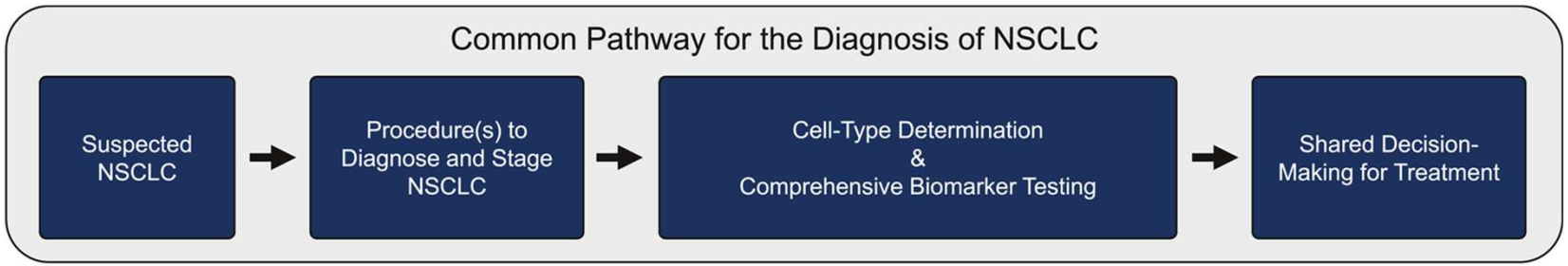
Five key challenge areas, continued

Challenge 3. Choice of assay and turnaround time	Recommendation 3. Promote the use of guideline-driven biomarker panel testing through testing algorithms and implementation of reflex testing.
Challenge 4. Accurate interpretation and communication of testing results	Recommendation 4. Encourage standardized biomarker test reports and develop algorithms that aid in directing treatment.
Challenge 5. Reimbursement, cost, and coverage	Recommendation 5. Remove the disconnect between payer policies and evidence-based guidelines for comprehensive biomarker testing to increase coverage and avoid delays.

Table adapted from: Fox AH, etc. Cancer. 2024 Dec 15;130(24):4188-4199. PMID: 39347617.

Barriers to comprehensive biomarker testing

a.



b.

Primary Care:	**Proceduralists**:	**Pathologists**:	**Payers**:	**Oncologists**:
• Nihilism over the lung cancer diagnosis • Lack of ability to engage after diagnosis	• Lack of awareness for testing • Adequate tissue collection for testing • Lack of feedback on performance	• Lack of awareness of testing and updates • Not saving enough tissue for biomarker testing	• Time and energy for prior authorization process • Lack of efficient and global process for reviewing evidence on utility	• Staying abreast of rapid changes in evidence • Difficulty interpreting biomarker test results • Lack of timely access to tumor boards

Table adapted from: Fox AH, etc. Cancer. 2024 Dec 15;130(24):4188-4199. PMID: 39347617.

1. Referenced with permission from the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Guideline Name V.8.2025. © National Comprehensive Cancer Network, Inc. 202X. All rights reserved. To view the most recent and complete version of the guideline, go online to NCCN.org.
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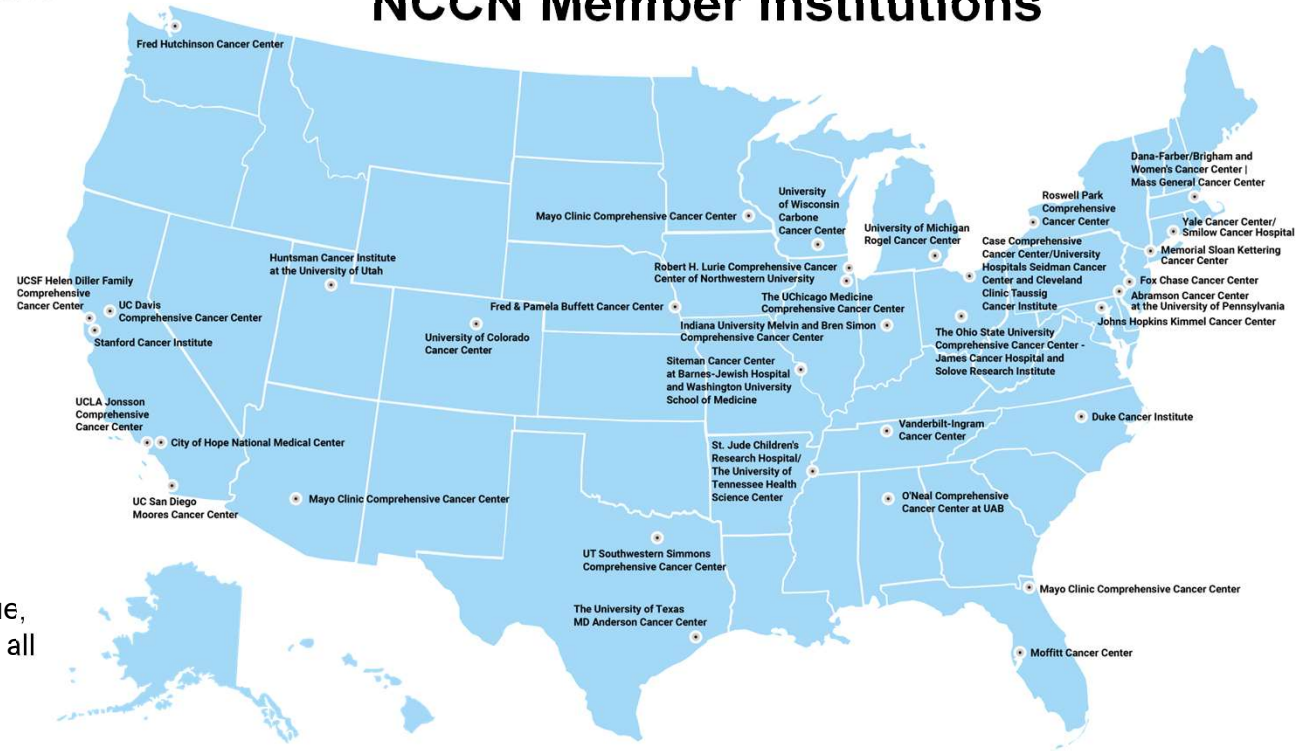

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An alliance of leading cancer centers devoted to patient care, research, and education

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To define and advance quality, effective, equitable, and accessible cancer care and prevention so all people can live better lives

Our Vision

Access to high-quality, high-value, patient-centered cancer care for all people globally

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