



Targeted Genetic Testing for Selection of Therapy for Non-Small Cell Lung Cancer (NSCLC)

Effective: March 1, 2026

Next Review: November 2026

Last Review: January 2026

IMPORTANT REMINDER

Medical Policies are developed to provide guidance for members and providers regarding coverage in accordance with contract terms. Benefit determinations are based in all cases on the applicable contract language. To the extent there may be any conflict between the Medical Policy and contract language, the contract language takes precedence.

PLEASE NOTE: Contracts exclude from coverage, among other things, services or procedures that are considered investigational or cosmetic. Providers may bill members for services or procedures that are considered investigational or cosmetic. Providers are encouraged to inform members before rendering such services that the members are likely to be financially responsible for the cost of these services.

DESCRIPTION

Targeted testing for specific gene variants, including *EGFR* and *BRAF* analysis, can be used to predict treatment response to targeted therapy in patients with advanced NSCLC.

MEDICAL POLICY CRITERIA

- I. Testing for *NTRK*, *NRG1*, and *RET* gene fusions and *BRAF*, *EGFR*, *ALK*, *ERBB2* (HER2), *KRAS*, *MET*, *PD-L1*, and *ROS1* variants may be considered **medically necessary** for individuals with non-small cell lung cancer (NSCLC) for selection of therapy.
- II. The Oncomine™ Dx Target test may be considered **medically necessary** for individuals with NSCLC for selection of therapy.
- III. Testing for purposes other than treatment selection in NSCLC is considered **investigational**.

NOTE: A summary of the supporting rationale for the policy criteria is at the end of the policy.

POLICY GUIDELINES

The Oncomine™ Dx Target test was approved by the FDA as a companion diagnostic to aid in selecting NSCLC patients for treatment with gefitinib (Iressa®), crizotinib (Xalcori®), or a combination of dabrafenib (Tafinlar®) and trametinib (Mekinist®). The test identifies tumors that have *EGFR* variants, *ROS1* fusions, and/or the *BRAF* V600E variant.

The FDA approved cobas® *EGFR* Mutation Test v2 is only intended to be used to aid in identifying patients with NSCLC whose tumors have defined *EGFR* mutations and for whom safety and efficacy of a drug have been established. This test may be run on either tumor or plasma samples.

LIST OF INFORMATION NEEDED FOR REVIEW

It is critical that the list of information below is submitted for review to determine if the policy criteria are met. If any of these items are not submitted, it could impact our review and decision outcome.

1. Name of the genetic test(s) or panel test
2. Name of the performing laboratory and/or genetic testing organization (more than one may be listed)
3. The exact gene(s) and/or variants being tested
4. Relevant billing codes
5. Brief description of how the genetic test results will guide clinical decisions that would not otherwise be made in the absence testing
6. Medical records related to this genetic test
 - o History and physical exam
 - o Conventional testing and outcomes
 - o Conservative treatment provided, if any

CROSS REFERENCES

1. [KRAS, NRAS, and BRAF Variant Analysis and MicroRNA Expression Testing for Colorectal Cancer](#), Genetic Testing, Policy No. 13
2. [Genetic and Molecular Diagnostic Testing](#), Genetic Testing, Policy No. 20
3. [BRAF Gene Mutation Testing To Select Melanoma or Glioma Patients for Targeted Therapy](#), Genetic Testing, Policy No. 41
4. [Evaluating the Utility of Genetic Panels](#), Genetic Testing, Policy No. 64
5. [Expanded Molecular Testing of Cancers to Select Targeted Therapies](#), Genetic Testing, Policy No. 83
6. [Circulating Tumor DNA and Circulating Tumor Cells for Management \(Liquid Biopsy\) of Solid Tumor Cancers](#), Laboratory, Policy No. 46
7. [Molecular Testing in the Management of Pulmonary Nodules](#), Laboratory, Policy No. 73
8. [Medication Policy Manual](#), Note: Click the link for the appropriate Medication Policy. Once the medication policy site is open, do a find (Ctrl+F) and enter drug name in the find bar to locate the appropriate policy.

BACKGROUND

TARGETED THERAPY FOR NON-SMALL CELL LUNG CANCER (NSCLC)

Treatment options for NSCLC depend on disease stage and include various combinations of surgery, radiation therapy, chemotherapy, and best supportive care. In up to 85% of cases, the cancer has spread locally beyond the lungs at diagnosis, precluding surgical eradication. In addition, up to 40% of patients with NSCLC present with metastatic disease.^[1] Treatment of

advanced NSCLC has generally been with platinum-based chemotherapy, with a median survival of 8 to 11 months and a one-year survival of 30% to 45%.^[2, 3] More recently, the identification of specific, targetable oncogenic “driver” variants in a subset of NSCLCs has resulted in a reclassification of lung tumors to include molecular subtypes, which are predominantly of adenocarcinoma histology.

EPIDERMAL GROWTH FACTOR RECEPTOR (*EGFR*)

EGFR is a receptor tyrosine kinase (TK) frequently overexpressed and activated in NSCLC. Laboratory and animal experiments have shown that therapeutic interdiction of the EGFR pathway could be used to halt tumor growth in solid tumors that express EGFR.^[4] These observations led to the development of two main classes of anti-EGFR agents for use in various types of cancer: small molecule TKIs and monoclonal antibodies (MAbs) that block EGFR-ligand interaction.^[5] The prevalence of *EGFR* variants in NSCLC varies by population, with the highest prevalence in non-smoking, Asian women, with adenocarcinoma, in whom *EGFR* variants have been reported to be up to 30-50%. The reported prevalence in the Caucasian population is approximately 10%.^[6]

Variants in two regions of the *EGFR* gene (exons 18-24)—small deletions in exon 19 and a point mutation in exon 21 (L858R)—appear to predict tumor response to first and second generation tyrosine kinase inhibitors (TKIs) such as erlotinib, gefitinib and afatinib.^[7, 8] In addition, a single point mutation in exon 20 (T790M) appears to predict tumor response to third generation TKIs such as osimertinib. These can be detected by direct sequencing or polymerase chain reaction (PCR) technologies.

Testing is intended for use in patients with advanced NSCLC. Patients with either small deletions in exon 19 or a point mutation in exon 21 (L858R) of the tyrosine kinase domain of the EGFR gene are considered good candidates for treatment with first and second generation TKIs. Patients with the point mutation in exon 20 (T790M), which is indicative of acquired resistance to first and second generation TKIs, are considered good candidates for third generation TKIs. Patients found to be wild-type are unlikely to respond to TKIs, so other treatment options should be considered.

ALK

ALK is a TK that is aberrantly activated in NSCLC due to a chromosomal rearrangement that leads to a fusion gene and expression of a protein with constitutive activity that has been demonstrated to play a role in controlling cell proliferation. The *EML4-ALK* fusion gene results from an inversion within the short arm of chromosome 2. The *EML4-ALK* rearrangement (“ALK-positive”) is detected in 3% to 6% of NSCLC patients, with the highest prevalence in never-smokers or light ex-smokers who have adenocarcinoma.

BRAF

RAF proteins are serine/threonine kinases that are downstream of RAS in the RAS-RAF-ERK-MAPK pathway. In this pathway, the *BRAF* gene is the most frequently altered in NSCLC, in approximately 1-3% of adenocarcinomas. Unlike melanoma, about 50% of the variants in NSCLC are non-V600E variants.^[9] Most *BRAF* variants occur more frequently in smokers.

ERBB2

ERBB2 is the gene that codes for the human epidermal growth factor receptor 2 (HER2) protein. HER2 is a member of the HER (EGFR) family of TK receptors and has no specific ligand. When activated, it forms dimers with other EGFR family members. HER2 is expressed in approximately 25% of NSCLC. *ERBB2* variants are detected mainly in exon 20 in 1% to 2% of NSCLC, predominantly in adenocarcinomas in nonsmoking women.^[9]

KRAS

KRAS is a G-protein involved in the EGFR-related signal transmission. The *KRAS* gene, which encodes RAS proteins, can harbor oncogenic variants that result in a constitutively activated protein, independent of signaling from the EGF receptor, possibly rendering a tumor resistant to therapies that target the EGF receptor. Variants in the *KRAS* gene, mainly codons 12 and 13, have been reported in 20-30% of NSCLC, and occur most often in adenocarcinomas in heavy smokers.

MET

MET amplification is one of the critical events for acquired resistance in *EGFR*-mutated adenocarcinomas refractory to EGFR TKIs.

NRG1

NRG1 gene fusions are relatively rare but can act as oncogenic drivers for NSCLC and other cancer types. They are mainly seen in lung invasive mucinous adenocarcinomas.

NTRK

NTRK gene fusions encode tropomyosin receptor kinase fusion proteins that act as oncogenic drivers for solid tumors including lung, salivary gland, thyroid, and sarcoma. It is estimated that NTRK gene fusions occur in 0.2% of patients with NSCLC and do not typically overlap with other oncogenic drivers.

PD-L1

Programmed cell ligand-1 (PD-L1) is a transmembrane protein expressed on the surface of multiple tissue types, including many tumor cells. Blocking the PD-L1 protein may prevent cancer cells from inactivating T cells.

RET

RET (rearranged during transfection) is a proto-oncogene that encodes a receptor TK growth factor. Translocations that result in fusion genes with several partners have been reported. RET fusions occur in 0.6% to 2% of NSCLCs and 1.2% to 2% of adenocarcinomas.

ROS1

ROS1 codes for a receptor TK of the insulin receptor family, and chromosomal rearrangements result in fusion genes. The prevalence of *ROS1* fusions in NSCLC varies from 0.9% to 3.7%. Patients with *ROS1* fusions are typically never-smokers with adenocarcinoma.

REGULATORY STATUS

The FDA Centers for Devices and Radiological Health (CDRH), for Biologics Evaluation and

Research (CBER), and for Drug Evaluation and Research (CDER) developed a draft guidance on in vitro companion diagnostic devices, which was released on July 14, 2011,^[8] to address the “emergence of new technologies that can distinguish subsets of populations that respond differently to treatment.” As stated, the FDA encourages the development of treatments that depend on the use of companion diagnostic devices “when an appropriate scientific rationale supports such an approach.” In such cases, the FDA intends to review the safety and effectiveness of the companion diagnostic test as used with the therapeutic treatment that depends on its use. The rationale for co-review and approval is the desire to avoid exposing patients to preventable treatment risk.

The Oncomine™ Dx Target test is an FDA approved companion diagnostic test for *EGFR* variants, *ROS1* gene fusions, *RET* variants, *ERBB2* variants, and the *BRAF* V600E variant, to aid in selection of the following targeted therapies for NSCLC:

- gefitinib (Iressa®)
- crizotinib (Xalcori®)
- dabrafenib (Tafinlar®) plus trametinib (Mekinist®)
- pralsetinib (Gavreto®)
- amivantamab (Rybrevant®)
- fam-trastuzumab deruxtecan-nxki (Enhertu®)
- selpercatinib (Retevmo®)

There are two other U.S. Food and Drug Administration (FDA)-approved companion diagnostic tests that specifically test for *EGFR* variants in NSCLC, intended to be used with select FDA approved *EGFR* tyrosine kinase inhibitors (TKIs):

- The cobas® *EGFR* Mutation Test v2 is a companion diagnostic test for the detection of exon 19 deletions and exon 20 and 21 (T790M and L858R, respectively) substitution variants in the *EGFR* gene in NSCLC tumor tissue. The FDA states:

“The test is intended to be used as an aid in selecting patients with NSCLC for whose tumors have defined *EGFR* variants and for whom safety and efficacy of a drug have been established as follows:

- Tarceva® (erlotinib) - Exon 19 deletions and L858R
- Tagrisso® (osimertinib) - T790M”

This test (v2) was approved 11/13/2015 as a result of an expansion of the original cobas® *EGFR* Mutation Test to cover testing for the T790M point mutation for use of osimertinib.

- The theascreen® *EGFR* Rotor Gene Q polymerase chain reaction (PCR) Kit is an automated molecular assay designed to detect the presence of *EGFR* exon 19 deletions and the exon 21 (L858R) substitution variant in NSCLC tumor tissue. The test is intended to be used to select patients with NSCLC for whom GILOTRIF® (afatinib) or IRESSA® (gefitinib) is indicated.

EVIDENCE SUMMARY

Human Genome Variation Society (HGVS) nomenclature^[10] is used to describe variants found in DNA and serves as an international standard. It is being implemented for genetic testing

medical evidence review updates starting in 2017. According to this nomenclature, the term “variant” is used to describe a change in a DNA or protein sequence, replacing previously-used terms, such as “mutation.” Pathogenic variants are variants associated with disease, while benign variants are not. The majority of genetic changes have unknown effects on human health, and these are referred to as variants of uncertain significance.

The focus of the following review is on evidence related to the ability of test results to:

- Guide decisions in the clinical setting related to either treatment, management, or prevention, and
- Improve health outcomes as a result of those decisions.

The clinical utility of testing for variants in the *EGFR* gene and others to guide TKI treatment in patients with advanced NSCLC has been unequivocally demonstrated. Testing for variants in the other genes is also well-supported by published evidence. Therefore, this review will focus on literature that has been published on the investigational indications described in this policy.

No studies were identified that evaluated targeted genetic testing for patients with NSCLC for purposes other than treatment selection.

PRACTICE GUIDELINE SUMMARY

NATIONAL COMPREHENSIVE CANCER NETWORK (NCCN)^[11]

NCCN guidelines for the treatment of metastatic NSCLC (v.3.2026) recommend testing for genetic variants in *EGFR*, *ALK*, *KRAS*, *ROS1*, *BRAF*, *NTRK1/2/3*, *MET*, *RET*, *ERBB2*, and *NRG1*, and testing for HER2 and PD-L1 expression for patients with non-squamous NSCLC (i.e., adenocarcinoma, large cell carcinoma, or NSCLC not otherwise specified). For patients with squamous cell carcinoma, the guidelines recommend PD-L1 testing, and considering *EGFR*, *ALK*, *KRAS*, *NTRK*, *MET*, *RET*, *ROS1*, *NRG1*, and *BRAF* molecular testing and HER2 expression testing.

According to these recommendations, molecular testing for all advanced or metastatic NSCLC should be conducted as a part of multigene panel testing.

COLLEGE OF AMERICAN PATHOLOGISTS, INTERNATIONAL ASSOCIATION FOR THE STUDY OF LUNG CANCER, AND ASSOCIATION FOR MOLECULAR PATHOLOGY (CAP/IASLC/AMP)

The 2018 updated guidelines issued jointly by the CAP/IASLC/AMP recommend:^[12]

- *ROS1* testing must be performed on all lung adenocarcinoma patients, irrespective of clinical characteristics. (Strong Recommendation)
- *ROS1* IHC may be used as a screening test in lung adenocarcinoma patients; however, positive *ROS1* IHC results should be confirmed by a molecular or cytogenetic method. (Expert Consensus Opinion)
- *BRAF* molecular testing is currently not indicated as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *BRAF* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative. (Expert Consensus Opinion)
- *RET* molecular testing is not recommended as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *RET* as part of larger testing panels

performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative. (Expert Consensus Opinion)

- *ERBB2* (HER2) molecular testing is not indicated as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *ERBB2* (HER2) mutation analysis as part of a larger testing panel performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative. (Expert Consensus Opinion)
- *KRAS* molecular testing is not indicated as a routine stand-alone assay as a sole determinant of targeted therapy. It is appropriate to include *KRAS* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative. (Expert Consensus Opinion)
- *MET* molecular testing is not indicated as a routine stand-alone assay outside the context of a clinical trial. It is appropriate to include *MET* as part of larger testing panels performed either initially or when routine *EGFR*, *ALK*, and *ROS1* testing are negative. (Expert Consensus Opinion)

Regarding cell-free DNA (cfDNA) testing, the guidelines state:

- There is currently insufficient evidence to support the use of circulating cfDNA molecular methods for the diagnosis of primary lung adenocarcinoma. (No Recommendation)
- In some clinical settings in which tissue is limited and/or insufficient for molecular testing, physicians may use a cfDNA assay to identify *EGFR* mutations. (Recommendation)
- Physicians may use cfDNA methods to identify *EGFR* T790M mutations in lung adenocarcinoma patients with progression or secondary clinical resistance to *EGFR*-targeted TKI; testing of the tumor sample is recommended if the plasma result is negative. (Expert Consensus Opinion)
- There is currently insufficient evidence to support the use of circulating tumor cell molecular analysis for the diagnosis of primary lung adenocarcinoma, the identification of *EGFR* or other mutations, or the identification of *EGFR* T790M mutations at the time of *EGFR* TKI resistance. (No Recommendation)

AMERICAN SOCIETY OF CLINICAL ONCOLOGY

In 2021, the American Society of Clinical Oncology (ASCO) and Ontario Health published updated guidelines on therapy for stage IV NSCLC with driver alterations.^[13] The updated recommendations were based on a systematic review of RCTs from December 2015 to January 2020 and meeting abstracts from ASCO 2020. The recommendations include the following:

- All patients with nonsquamous NSCLC should have the results of testing for potentially targetable mutations (alterations) before implementing therapy for advanced lung cancer, regardless of smoking status, when possible.
- Targeted therapies against *ROS-1* fusions, *BRAF* V600e mutations, *RET* fusions, *MET* exon 14 skipping mutations, and *NTRK* fusions should be offered to patients, either as initial or second-line therapy when not given in the first-line setting.
- Chemotherapy is still an option at most stages.

The above guidelines were updated in 2023 to add amivantamab monotherapy and mobocertinib monotherapy for second-line treatment in advanced NSCLC with an *EGFR* exon

20 insertion, and sotorasib monotherapy for second-line treatment in advanced NSCLC with a *KRAS*-G12C mutation.^[14]

In 2022, ASCO published a guideline on the management of stage III NSCLC.^[15] The recommendations were based on a literature search of systematic reviews, meta-analyses, and randomized controlled trials published from 1990 through 2021. Relevant recommendations include the following:

- Presence of oncogenic driver alterations, available therapies, and patient characteristics should be taken into account.
- Patients with resected stage III NSCLC with *EGFR* exon 19 deletion or exon 21 L858R mutation may be offered adjuvant osimertinib after platinum-based chemotherapy.

SUMMARY

NTRK, NRG1, AND RET* GENE FUSIONS AND *BRAF, EGFR, ALK, KRAS, MET, PD-L1, ERBB2, AND ROS1

There is enough research to show that testing for *NTRK*, *NRG1*, and *RET* gene fusions and *BRAF*, *EGFR*, *ALK*, *KRAS*, *MET*, *PD-L1*, *ERBB2* (HER2), and *ROS1* variants can help to guide treatment for individuals with non-small cell lung cancer (NSCLC). In addition, many clinical guidelines based on research recommend testing for those with this disease. Therefore, this testing may be considered medically necessary for selection of therapy.

There is not enough research to show that for *NTRK*, *NRG1*, and *RET* gene fusions and *BRAF*, *EGFR*, *ALK*, *KRAS*, *MET*, *PD-L1*, *ERBB2* (HER2), and *ROS1* variants can improve health outcomes for individuals with NSCLC when not used for treatment selection. Therefore, this testing is considered investigational when policy criteria are not met.

ONCOMINE™ DX TARGET TEST

The Oncomine™ Dx Target Test is an FDA-approved companion diagnostic test to help identify individuals with non-small cell lung cancer (NSCLC) that may benefit from certain medications. The test identifies tumors that have variants in the *EGFR*, *ROS1*, and *BRAF* genes, which may respond to targeted treatments. Therefore, use of the Oncomine™ Dx Target test may be considered medically necessary to select individuals with advanced or metastatic NSCLC for targeted treatment.

There is not enough research to show that the Oncomine™ Dx Target Test can improve health outcomes related to NSCLC when not used for treatment selection. Therefore, the use of this test is considered investigational for individuals that do not meet policy criteria.

REFERENCES

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- vinorelbine or gemcitabine in advanced non-small cell lung cancer. *Eur J Cancer*. 2005;41(1):81-92. PMID: 15617993
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CODES

Codes	Number	Description
CPT	0022U	Targeted genomic sequence analysis panel, nonsmall cell lung neoplasia, DNA and RNA analysis, 23 genes, interrogation for sequence variants and rearrangements, reported as presence/or absence of variants and associated therapy(ies) to consider

Codes	Number	Description
	0478U	Oncology (non-small cell lung cancer), DNA and RNA, digital PCR analysis of 9 genes (EGFR, KRAS, BRAF, ALK, ROS1, RET, NTRK 1/2/3, ERBB2, and MET) in formalin-fixed paraffin-embedded (FFPE) tissue, interrogation for single-nucleotide variants, insertions/deletions, gene rearrangements, and reported as actionable detected variants for therapy selection
	81191	NTRK1 (neurotrophic receptor tyrosine kinase 1) (eg, solid tumors) translocation analysis
	81192	NTRK2 (neurotrophic receptor tyrosine kinase 2) (eg, solid tumors) translocation analysis
	81193	NTRK3 (neurotrophic receptor tyrosine kinase 3) (eg, solid tumors) translocation analysis
	81194	NTRK (neurotrophic-tropomyosin receptor tyrosine kinase 1, 2, and 3) (eg, solid tumors) translocation analysis
	81210	<i>BRAF</i> (<i>B-Raf proto-oncogene, serine/threonine kinase</i>) (e.g., colon cancer, melanoma), gene analysis, V600 variant(s)
	81235	EGFR (epidermal growth factor receptor) (eg, non-small cell lung cancer) gene analysis, common variants (eg, exon 19 LREA deletion, L858R, T790M, G719A, G719S, L861Q)
	81275	KRAS (v-Ki-ras2 Kirsten rat sarcoma viral oncogene) (eg, carcinoma) gene analysis, variants in exon 2 (eg, codons 12 and 13)
	81276	KRAS (Kirsten rat sarcoma viral oncogene homolog) (eg, carcinoma) gene analysis; additional variant(s) (eg, codon 61, codon 146)
	81404	Molecular pathology procedure, Level 5 (eg, analysis of 2-5 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 6-10 exons, or characterization of a dynamic mutation disorder/triplet repeat by Southern blot analysis) – which includes <i>RET</i> (ret proto-oncogene) (eg, multiple endocrine neoplasia, type 2B and familial medullary thyroid carcinoma), common variants (eg, M918T, 2647_2648delinsTT, A883F)
	81405	Molecular pathology procedure, Level 6 (eg, analysis of 6-10 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 11-25 exons, regionally targeted cytogenomic array analysis) – which includes <i>KRAS</i> (Kirsten rat sarcoma viral oncogene homolog) (eg, Noonan syndrome), full gene sequence; and <i>RET</i> (ret proto-oncogene) (eg, multiple endocrine neoplasia, type 2A and familial medullary thyroid carcinoma), targeted sequence analysis (eg, exons 10, 11, 13-16)
	81406	Molecular pathology procedure, Level 7 (eg, analysis of 11-25 exons by DNA sequence analysis, mutation scanning or duplication/deletion variants of 26-50 exons) – which includes <i>BRAF</i> (<i>B-Raf proto-oncogene, serine/threonine kinase</i>) (eg, Noonan syndrome), full gene sequence
	81479	Unlisted molecular pathology procedure
	84999	Unlisted chemistry procedure
HCPCS	None	

Date of Origin: August 2010