

Physician Insert

FOR IN VITRO DIAGNOSTIC USE.

Intended Use

TruSight™ Oncology Comprehensive is a qualitative *in vitro* diagnostic test that uses targeted next-generation sequencing to detect variants in 517 genes using nucleic acids extracted from formalin-fixed, paraffin embedded (FFPE) tumor tissue samples from cancer patients with solid malignant neoplasms using the Illumina® NextSeq™ 550Dx instrument. The test can be used to detect single nucleotide variants, multi-nucleotide variants, insertions, and deletions from DNA, and fusions in 24 genes and splice variants in one gene from RNA. The test also reports a Tumor Mutational Burden (TMB) score and Microsatellite Instability (MSI) status.

The test is intended to be used as a companion diagnostic to identify cancer patients who may benefit from treatment with the targeted therapies listed in Table 1, in accordance with the approved therapeutic product labeling.

In addition, the test is intended to provide tumor profiling information for use by qualified health care professionals in accordance with professional guidelines in oncology for patients with solid malignant neoplasms. Genomic findings other than those listed in Table 1 of the intended use statement are not conclusive or prescriptive for labeled use of any specific therapeutic product.

Table 1 Companion Diagnostic Indications

Tumor Type	Biomarker(s) Detected	Therapy
Solid Tumors	<i>NTRK1/2/3 fusions</i>	VITRAKVI® (larotrectinib)
Non-Small Cell Lung Cancer (NSCLC) or Thyroid Cancer (TC)	<i>RET fusions</i>	RETEVMO® (selpercatinib)
Medullary Thyroid Cancer (MTC)	<i>RET SNVs, MNVs, insertions, and deletions</i>	RETEVMO® (selpercatinib)

Interpretation of Test Results

Variant results, including companion diagnostic results, tumor profiling results, Tumor Mutational Burden (TMB) score, and Microsatellite Instability (MSI) status are summarized in a TSO Comprehensive results report output to the testing laboratory and interpreted by a qualified health care professional.

Any companion diagnostic variant with an associated therapy, or any clinically significant or potentially clinically significant variant from the test, should be interpreted by board-certified clinical molecular geneticists or pathologists (or equivalent). These specialists review the results and determine which variants are most impactful for the patient in the context of tumor diagnosis, histopathology, and available laboratory data. These results are meant to be incorporated into a report produced by the laboratory and provided to clinicians who make decisions on patient management. Please refer to the Limitations of Procedure section of the Package Insert and

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Software Report for important limitations concerning assay results and incorporate those limitations into the final results report.

Additional information is available from applicable professional guidelines such as CAP/IASLC/AMP NSCLC Testing Guidelines¹ or the CAP/AMP/ASCO Interpretation and Reporting Guidelines,² or the Consensus Statement on Actionable Biomarker Testing for Thyroid Cancer Management.³



CAUTION

The assay is not intended as a primary diagnostic tool by physicians or to be used as a substitute for professional health care advice. Each laboratory is responsible for ensuring compliance with applicable international, national, and local clinical laboratory regulations and other specific accreditation requirements.

Illumina recommends that patients seek information from their oncologist and/or consult with a genetic counselor regarding their results.

What the Test Detects

TSO Comprehensive is an *in vitro* diagnostic test that uses targeted next-generation sequencing to detect variants in 517 genes using nucleic acids extracted from FFPE tumor tissue samples from previously diagnosed cancer patients with solid malignant neoplasms using the Illumina NextSeq 550Dx instrument. The test can be used to detect single nucleotide variants, multi-nucleotide variants, insertions, deletions, TMB score and MSI status from DNA, and RNA fusions in 24 genes and splice variants in one gene from RNA (EGFR).

For complete clinical and analytical performance of the test for companion diagnostics and tumor profiling indications, example reports, and interpretation of possible results, refer to the TruSight Oncology Comprehensive documentation available on the [Illumina support site](#). Refer to the FDA website for the P230011 Summary of Safety and Effectiveness Data (SSED).⁴

Companion Diagnostics

The test is intended as a companion diagnostic to identify previously diagnosed cancer patients for treatment with targeted therapies listed in [Table 1](#), in accordance with approved therapeutic product labeling. Companion diagnostics claims in [Table 1](#) are supported by analytical performance of the test for each specific biomarker and results from a clinical study establishing the link between the result of the test and patient outcomes.

Tumor Profiling

In addition to a companion diagnostic, the test is intended to provide tumor profiling information in previously diagnosed cancer patients with solid malignant neoplasms, for use by qualified health care professionals, in accordance with professional guidelines. The test is not conclusive or prescriptive for labeled use of any specific therapeutic product other than those listed in [Table 1](#).

Background

Companion Diagnostic Tests for Targeted NTRK Therapy Selection

NTRK fusions are a well-established oncogenic driver of disease across a variety of adult and pediatric cancers.⁵ The tropomyosin receptor kinase (TRK) family of receptor tyrosine kinases consists of TRKA, TRKB, and TRKC encoded by the neurotrophic tyrosine receptor kinase genes NTRK1,⁶ NTRK2,⁷ and NTRK3,⁸ respectively. Somatic inter- and intra-chromosomal rearrangements that cause an NTRK gene to fuse with another, unrelated gene have been shown to drive oncogenesis in a variety of both pediatric and adult tumors.

NTRK gene fusions have been estimated to occur in up to 1% of all solid tumors,⁹ although the frequency varies considerably between tumor types.^{5,9,10} Available evidence suggests that NTRK gene fusions are useful biomarkers to predict tumor response to TRK inhibitors. There is currently a selective tyrosine kinase inhibitor, larotrectinib (VITRAKVI), that is active against TRKs. Larotrectinib^{9,11} is a potent inhibitor of all three TRK variant types and has been authorized by the U.S. Food and Drug Administration (US FDA)¹² for the treatment of adult and pediatric patients with solid tumors that harbor NTRK fusions. For the most current information on the association between biomarkers and therapeutic outcomes, refer to the drug labeling information available on the FDA website.¹³

For the most current list of FDA-approved companion diagnostic devices for detection of NTRK fusions to select patients who may benefit from treatment with targeted therapies, refer to the FDA website.¹⁴

Companion Diagnostic Tests for Targeted RET Therapy Selection

Genomic variants in the RET gene have been estimated to occur in approximately 2% of all solid tumors,¹⁵ however, frequencies vary considerably between the type of RET abnormality and specific tumor types. Lung cancer remains the leading cause of cancer death worldwide, accounting for 2.5 million new cases (12.4% of all cancers) diagnosed in 2022 and claimed over 1.8 million lives.¹⁶ It is estimated that in the US, there will be 229,410 new cases of lung cancer with 124,990 deaths in 2026, accounting for 20% of cancer deaths in the US.¹⁷ In lung cancer, RET fusions are most prevalent in non-smokers and younger individuals.¹⁸ A number of fusion partner genes have been identified,¹⁵ with the most common fusions varying between tumor types. In non-small cell lung cancer (NSCLC), a common fusion partner is KIF5B.^{15,18} Thyroid cancer remains a significant cause of cancer worldwide, accounting for 821,173 new cases (4.1% of all cancers) diagnosed in 2022 and claiming 47,485 lives.¹⁶ RET gene fusions are generally rare.^{15,18-21} However, a higher frequency of RET rearrangements has been reported in select populations, including thyroid cancer patients with prior radiation exposure, such as survivors of the atomic bombing in Hiroshima and Nagasaki²² and the Chernobyl nuclear accident,²³ as well as thyroid cancer patients treated with radiotherapy.²¹ Medullary thyroid cancer (MTC) represents 1% to 5% of all thyroid cancers and accounts for approximately 13% of thyroid cancer-related deaths.²⁴ Somatic RET SNVs are associated with 40%-50% of all sporadic MTCs that collectively account for 75% of all MTCs.²⁴⁻²⁶ Selpercatinib (RETIVO) is a tyrosine kinase inhibitor that has significant activity against RET fusions. Selpercatinib²⁷ is a potent inhibitor of RET SNVs and fusions and has been authorized by the US FDA²⁸ for the treatment of pediatric and adult patients with NSCLC and thyroid cancer that harbor RET fusions, as well as patients with MTC that harbor DNA mutations. For the most current information on the association between biomarkers and therapeutic outcomes, refer to the drug labeling information available on the FDA website.¹³

For the most current list of FDA-approved companion diagnostic devices for detection of RET fusions to select

Limitations of the Procedure

For *in vitro* diagnostic use only.

- For prescription use only. The test must be ordered by a qualified medical professional in accordance with clinical laboratory regulations.
- Genomic findings listed in the TSO Comprehensive results report under Cancer Mutations with Evidence of Clinical Significance (Level 2) and Cancer Mutations with Potential Clinical Significance (Level 3) are not prescriptive or conclusive for labeled use of any specific therapeutic product, and clinical validation has not been performed.
- Decisions on patient care and treatment must be based on the independent medical judgment of the treating physician, taking into consideration all applicable information concerning the patient's condition, such as patient and family history, physical examinations, information from other diagnostic tests, and patient preferences, in accordance with the standard of care in a given community.
- FFPE sample quality is highly variable. Quality can be impacted by:
 - Specimens that did not undergo standard fixation procedures might not generate extracted nucleic acids that meet the assay quality control requirements.
 - High amounts of necrotic tissue ($\geq 25\%$) can interfere with the ability of the TSO Comprehensive assay to detect RNA fusions.
 - Somatic driver mutations may not be detected reliably if tumor content (by area) is less than 20%.
 - Samples with tumor content less than 40% may generate false negative results (MS-Stable) for MSI.
 - FFPE blocks that have been stored for longer than five years have demonstrated lower validity rate.
- The required input for the TSO Comprehensive assay is 40 ng RNA and/or 40 ng DNA. Lower DNA input may decrease test validity rate for MSI detection.
- The performance of TSO Comprehensive in samples obtained from patients that have had organ or tissue transplantation has not been evaluated.
- Accuracy of small DNA tumor profiling variants below 5% variant allele frequency (VAF) has not been established.
- Accuracy for the EGFRvIII splice variant from RNA has only been established in brain tissue. Accuracy for EGFRvIII in other tissue types has not been established.
- Detection of MSI-High status in endometrial/uterine cancer specimens is reduced relative to other tumor types. The observed positive percent agreement (PPA) in the MSI Tumor Profiling Accuracy study for endometrial/uterine cancers was 81% (13/16) for specimens with tumor content $\geq 40\%$.
- The Limit of Detection (LoD) for insertions of 1-2 base pairs with dinucleotide repeats has been established with cell lines.
- Contamination detection can be impacted by:
 - In highly rearranged genomes with deletions and loss of heterozygosity, TSO Comprehensive software can erroneously classify a DNA sample as contaminated (CONTAMINATION_SCORE > 3106 and p-value

> 0.049).

- Contamination during the procedure can result from nucleic acid from previous sample processing steps. Good laboratory practice and following all precautions and guidelines in this Package Insert will help avoid cross-contamination between samples.
- TSO Comprehensive does not have a metric to detect contamination for RNA libraries. If the same fusion is detected in multiple samples in the same run, repeating TSO Comprehensive testing is suggested.
- A negative result does not rule out the presence of a mutation below the limits of detection (LoD) of the assay. Alterations at allele frequencies below the established LoD may not be detected consistently.
- The overall negative percent agreement for NTRK fusions between drug trial enrollment assays and TSO Comprehensive was 96.3% (95%CI: 93.1%, 98.3%). False positive results for gene fusions such as NTRK may be due to non-specific detection.
- The sensitivity for detection of small DNA variants can be impacted by:
 - Low complexity genomic context.
 - Increasing variant length.
 - Phasing: MNVs (more than 3 base pairs), delins (one or more nucleotides replaced by one or more nucleotides and is not an SNV or MNV), and certain deletions require a phasing approach to be detected. A predefined set of MNVs, delins, and deletions are detected for the EGFR and RET genes using a phasing approach (refer to Appendix D, MNVs, delins, and Deletions in EGFR and RET Detectable by Phased Variant Caller in the *Local Run Manager TruSight Oncology Comprehensive (US) Analysis Module Workflow Guide (document # 200061834)*). The phasing approach for small variant calling is limited to only these variants. The detection rate of these variants (for example, RET E632_V637delinsAA) may not be as high as observed for tumor profiling variants that do not require phasing.
- The clinical significance of TMB measurement has not been established. TMB is reported as mutations per megabase (Muts/Mb). TMB is a function of characteristics of a patient's specimen and testing parameters; therefore, TMB values may differ across specimens (eg, primary vs metastatic, tumor content) and targeted assay panels. The TMB values will increase or decrease dependent on the variables listed below:
 - The size and specific region to calculate TMB may result in different values.
 - The percentage of tumor in the sample.
 - Methods of germline filtering.
 - The read depth threshold and other bioinformatic test specifications.
- TMB scores can be inaccurate in the following contexts:
 - As tumor content reaches levels where germline and somatic variant allele frequencies (VAFs) converge.
 - In ethnic populations not well represented in public databases.
- TSO Comprehensive performance has been demonstrated for insertions and deletions up to 24 bp.
- Fusion calling algorithms in the TSO Comprehensive assay software may not consider evidence from reads that extend outside of annotated gene boundaries.
- The sensitivity for detection of fusions can be impacted by:

- Low library complexity resulting in decreased supporting reads due to deviations in the assay workflow.
- A single gene spanning both breakpoints.
- Cases where several fusions breakpoints are in close proximity to each other with one or multiple partners, the multiple breakpoints and partners might be reported as a single breakpoint and partner.
- Small median insert sizes. A minimum median insert size of 80 bp is required, but sensitivity decreases in the 80–100 bp range.
- Low sequence complexity or homologous genomic context around fusion breakpoints.
- Non-canonical junctions (such as in intronic regions).
- Resolution of the genes involved in a fusion can be impacted when fusion breakpoints occur in genomic regions containing overlapping genes. The assay will report all genes, delimited by semicolons, if multiple genes are overlapping a breakpoint.
 - RNA fusions where both gene partners (GeneA/GeneB) are overlapping cannot be distinguished as either true fusion events or alternative splicing artifacts.
- Inconsistent coverage in the TERT Promoter region can result in a low depth outcome in that region for small DNA variants.
- Annotation or Knowledge Base errors can cause a false positive or false negative result, including listing a variant in the wrong cancer mutation level (between Level 2 and 3), or the annotation information in the report could be incorrect. Annotation or Knowledge Base errors do not impact variants reported in the CDx Results. The possibility of error exists from the following three sources:
 - TSO Comprehensive variant annotation. There is an error rate of approximately 0.0027% based on an analysis of 2,448,350 variants from COSMIC v92, therefore there is a low possibility for errors.
 - Knowledge Base error due to the curation or leveling process.
 - The report reflects the knowledge at the time when the Knowledge Base version was curated.
- TSO Comprehensive is designed to report somatic variants and is not intended to report germline variants, except for BRCA1/2 and RET germline variants with therapeutic, diagnostic, and prognostic associations with an established solid malignant neoplasm. When reporting variants with evidence of clinical significance or variants with potential clinical significance, as a tumor-only test, germline (inherited) variants may be inadvertently reported since TSO Comprehensive uses a Knowledge Base to report somatic variants without explicitly annotating germline or somatic origin. However, if a reported alteration is suspected to be germline, confirmatory testing should be considered in the appropriate clinical context.
- The Knowledge Base only includes therapeutic, diagnostic, and prognostic associations that are relevant for variants present within an established solid malignant neoplasm. Susceptibility or cancer risk associations are not included in the Knowledge Base.
- A C95 value was not directly evaluated for a RET insertion in FFPE tissue. Based on observed C95 values for RET insertions evaluated in FFPE cell line materials and for a RET MNV assessed in FFPE tissue-all within Exon 11, where activating insertions are predominantly observed, the estimated C95 for a RET insertion in FFPE tissue is expected to fall within the range of 0.022 to 0.071.
- One RET delins variant (E632_V637delinsAA) was not reliably detected by TSO Comp software and therefore, its C95 could not be determined.

References

1. Lindeman N, Cagle P, Aisner D, et al. Updated Molecular Testing Guideline for the Selection of Lung Cancer Patients for Treatment With Targeted Tyrosine Kinase Inhibitors Guideline From the College of American Pathologists, the International Association for the Study of Lung Cancer, and the Association for Molecular Pathology. *Arch Pathol Lab Med*. 2018;142(3):321-346. doi:10.5858/arpa.2017-0388-CP
2. Li M, Datto M, Duncavage E, et al. Standards and Guidelines for the Interpretation and Reporting of Sequence Variants in Cancer: A Joint Consensus Recommendation of the Association for Molecular Pathology, American Society of Clinical Oncology, and College of American Pathologists. *J Mol Diagn*. 2017;19(1):4-23. doi:10.1016/j.jmoldx.2016.10.002
3. Mete O, Boucher A, Schrader KA, et al. Consensus Statement: Recommendations on Actionable Biomarker Testing for Thyroid Cancer Management. *Endocr Pathol*. 2024; 35:293-308. doi: 10.1007/s12022-024-09836-x
4. U.S. Food & Drug Administration. List of Cleared or Approved Companion Diagnostic Devices. Premarket Approval. Accessed August 22, 2025. <https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfpma/pma.cfm?id=P230011>
5. Cocco E, Scaltriti M, Drilon A. NTRK fusion-positive cancers and TRK inhibitor therapy. *Nat Rev Clin Oncol*. 2018;15(12):731-747. doi:10.1038/s41571-018-0113-0
6. Weier HU, Rhein AP, Shadravan F, Collins C, Polikoff D. Rapid physical mapping of the human trk protooncogene (NTRK1) to human chromosome 1q21-q22 by P1 clone selection, fluorescence in situ hybridization (FISH), and computer-assisted microscopy. *Genomics*. 1995;26(2):390-3. doi:10.1016/0888-7543(95)80226-C
7. Nakagawara A, Liu XG, Ikegaki N, et al. Cloning and chromosomal localization of the human TRK-B tyrosine kinase receptor gene (NTRK2). *Genomics*. 1995;25(2):538-46. doi:10.1016/0888-7543(95)80055-Q
8. Valent A, Danglot G, Bernheim A. Mapping of the tyrosine kinase receptors trkA (NTRK1), trkB (NTRK2) and trkC(NTRK3) to human chromosomes 1q22, 9q22 and 15q25 by fluorescence in situ hybridization. *Eur J Hum Genetics*. 1997;5(2):102-4. doi:10.1159/000484742
9. Drilon A, Laetsch TW, Kummar S, et al. Efficacy of Larotrectinib in TRK Fusion-Positive Cancers in Adults and Children. *N Engl J Med*. 2018;378(8):731-739. doi:10.1056/NEJMoa1714448
10. Amatu A, Sartore-Bianchi A, Bencardino K, Pizzutilo EG, Tosi F, Siena S. Tropomyosin receptor kinase (TRK) biology and the role of NTRK gene fusions in cancer. *Ann Oncol*. 2019;30(Suppl_8):viii5-viii15. doi:10.1093/annonc/mdz383
11. Burris HA, Shaw AT, Bauer TM, et al. Abstract 4529: Pharmacokinetics (PK) of LOXO-101 during the first-in-human Phase I study in patients with advanced solid tumors: Interim update. *Cancer Res*. 2015;75(15 Supplement):4529. doi:10.1158/1538-7445.AM2015-4529
12. VITRAKVI (larotrectinib). Highlights of Prescribing Information. Bayer; 2018. Accessed May 5, 2023. <https://www.accessdata.fda.gov/scripts/cder/daf/index.cfm>
13. U.S. Food & Drug Administration. Drugs@FDA: FDA-Approved Drugs. Updated April 9, 2024. Accessed August 22, 2025. <https://www.accessdata.fda.gov/scripts/cder/daf/index.cfm>

14. U.S. Food & Drug Administration. List of Cleared or Approved Companion Diagnostic Devices (In Vitro and Imaging Tools). Updated March 05, 2025. Accessed August 22, 2025. <https://www.fda.gov/medical-devices/in-vitro-diagnostics/list-cleared-or-approved-companion-diagnostic-devices-in-vitro-and-imaging-tools>
15. Kato S, Subbiah V, Marchlik E, et al. RET Aberrations in Diverse Cancers: Next-Generation Sequencing of 4,871 Patients. *Clin Cancer Res*. 2017;23(8):1988. doi:10.1158/1078-0432.CCR-16-1679
16. Bray F, Laversanne M, Sung H, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin*. 2024;74(3):229-263. doi:10.3322/caac.21834
17. Siegel RL, Kratzer TB, Wagle NS, Sung H, Jemal A. Cancer statistics, 2026. *CA Cancer J Clin*. 2026;76 (1):e70043. doi:10.3322/caac.70043
18. Drilon A, Hu ZI, Lai GGY, Tan DSW. Targeting RET-driven cancers: lessons from evolving preclinical and clinical landscapes. *Nat Rev Clinical Oncol*. 2018;15(3):151-167. doi:10.1038/nrclinonc.2017.175
19. Wang R, Hu H, Pan Y, et al. RET fusions define a unique molecular and clinicopathologic subtype of non small-cell lung cancer. *J Clin Oncol* 2012;30(35):4352-9. doi:10.1200/JCO.2012.44.1477
20. Agrawal N, Akbani R, Aksoy BA, et al. Integrated genomic characterization of papillary thyroid carcinoma. *Cell*. 2014;159(3):676-690. doi:10.1016/j.cell.2014.09.050
21. Bounacer A, Wicker R, Caillou B, et al. High prevalence of activating ret proto-oncogene rearrangements, in thyroid tumors from patients who had received external radiation. *Oncogene*. 1997;15(11):1263-1273. doi:10.1038/sj.onc.1200206
22. Hamatani K, Eguchi H, Ito R, et al. RET/PTC rearrangements preferentially occurred in papillary thyroid cancer among atomic bomb survivors exposed to high radiation dose. *Cancer Res*. 2008;68(17):7176-82. doi:10.1158/0008-5472.CAN-08-0293
23. Leeman-Neill RJ, Brenner AV, Little MP, et al. RET/PTC and PAX8/PPAR γ chromosomal rearrangements in post-Chernobyl thyroid cancer and their association with iodine-131 radiation dose and other characteristics. *Cancer*. 2013;119(10):1792-1799. doi:10.1002/cncr.27893
24. Santillan VR, Master SR, Menon G, et al. Medullary Thyroid Cancer. *STATPearls*. Updated November 10, 2024. Accessed Mar 17 2025. <https://www.ncbi.nlm.nih.gov/books/NBK459354/>
25. Elisei R, Cosci B, Romei C, et al. Prognostic significance of somatic RET oncogene mutations in sporadic medullary thyroid cancer: a 10-year follow-up study. *J Clin Endocrinol Metab*. 2008;93(3):682-687. doi:10.1210/jc.2007-1714
26. Arighi E, Borrello MG, Sariola H. RET tyrosine kinase signaling in development and cancer. *Cytokine Growth Factor Rev*. 2005;16(4):441-467. doi:10.1016/j.cytogfr.2005.05.010
27. Subbiah V, Velcheti V, Tuch BB, et al. Selective RET kinase inhibition for patients with RET-altered cancers. *Ann Oncol* 2018;29(8):1869-1876. doi:10.1093/annonc/mdy137
28. RETEVMO (selpercatinib). Highlights of Prescribing Information. Eli Lilly and Company; 2020. Accessed August 22, 2025. <https://www.accessdata.fda.gov/scripts/cder/daf/index.cfm>

Revision History

Document	Date	Description of Change
Document # 200061833 v02	August 2026	<Summary of Changes to be added after finishing labeling negotiations with FDA>
Document # 200061833 v01	July 2026	Added - MSI information - Limitations of the Procedure to align with Package Insert - Reference to US FDA premarket approval Updated - List of references - Language for editorial consistency
Document # 200061833 v00	October 2024	Initial Release

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Product Labeling

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