MI PROFILE™
MOLECULAR INTELLIGENCE TUMOR REPORT

CARIS® LIFE SCIENCES

FINAL REPORT

PATIENT

Name: Patient, Test
Date of Birth: XX-Mon-1952
Sex: Female
Case Number: TN15-111111
Diagnosis: Leiomyosarcoma, NOS

SPECIMEN INFORMATION

Primary Tumor Site: Uterus, NOS
Specimen Site: Peritoneum, NOS
Specimen ID: ABC-12345-YZ
Specimen Collected: XX-Mon-2015
Completion of Testing: XX-Mon-2015

ORDERED BY

Ordering Physician, MD
The Cancer Center
12345 Main Street
Springfield, YZ
(123) 456-7890

Bold Therapies = On NCCN Compendium® Therapies

✔ THERAPIES WITH **POTENTIAL BENEFIT** (PAGE 3)

dacarbazine, **temozolomide**	MGMT★
doxorubicin, **epirubicin**, **liposomal-doxorubicin**	Her2/Neu, TOP2A, PGP

★ **Indicates Clinical Trial Opportunity • 8 Chemotherapy Trials • 3 Targeted Therapy Trials** *(See Clinical Trials Connector™ on page 6 for details.)*

✘ THERAPIES WITH **POTENTIAL LACK OF BENEFIT** (PAGE 4)

anastrozole, **exemestane**, **letrozole**, **megestrol acetate**	PR, ER
docetaxel	PGP, TUBB3, TLE3
gemcitabine	RRM1
abarelix, degarelix, goserelin, leuprolide, triptorelin	ER, PR, Androgen Receptor
abiraterone, bicalutamide, enzalutamide, flutamide	Androgen Receptor
capecitabine, fluorouracil, pemetrexed	TS
dabrafenib, vemurafenib	BRAF
fulvestrant, tamoxifen, toremifene	PR, ER
irinotecan, topotecan	TOPO1
paclitaxel	PGP, TUBB3, TLE3

? THERAPIES WITH **INDETERMINATE BENEFIT** (PAGE 5)

ado-trastuzumab emtansine (T-DM1)†, pertuzumab†, trastuzumab†	imatinib	nab-paclitaxel
carboplatin, cisplatin, oxaliplatin	lapatinib†	vandetanib
everolimus, temsirolimus		

†Association to Benefit was not indicated due to assay failure.

Therapies associated with potential benefit or lack of benefit, as indicated above, are based on biomarker results provided in this report and are based on published medical evidence. This evidence may have been obtained from studies performed in the cancer type present in the tested patient's sample or derived from another tumor type. The selection of any, all, or none of the matched therapies resides solely with the discretion of the treating physician. Decisions on patient care and treatment must be based on the independent medical judgment of the treating physician, taking into consideration all available information in addition to this report concerning the patient's condition in accordance with the applicable standard of care.

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SUMMARY OF BIOMARKER RESULTS (SEE APPENDIX FOR FULL DETAILS)

Biomarker	Method	Result
ABL1	NGS	Mutation Not Detected
AKT1	NGS	Mutation Not Detected
ALK	NGS	Mutation Not Detected
Androgen Receptor	IHC	Negative
APC	NGS	Mutation Not Detected
ATM	NGS	Mutation Not Detected
BRAF	NGS	Mutation Not Detected
BRCA1	NGS	Mutation Not Detected
BRCA2	NGS	Mutation Not Detected
c-KIT	NGS	Mutation Not Detected
cMET	CISH	No Hybridization
cMET	IHC	Negative
cMET	NGS	Mutation Not Detected
CSF1R	NGS	Mutation Not Detected
CTNNB1	NGS	Mutation Not Detected
EGFR	NGS	Mutation Not Detected
EGFR	IHC	Negative
ER	IHC	Negative
FGFR1	NGS	Mutation Not Detected
FGFR2	NGS	Mutation Not Detected
FLT3	NGS	Mutation Not Detected
GNA11	NGS	Mutation Not Detected
GNAQ	NGS	Mutation Not Detected
GNAS	NGS	Mutation Not Detected
Her2/Neu	CISH	No Hybridization
Her2/Neu	IHC	Negative
Her2/Neu (ERBB2)	NGS	Mutation Not Detected
HRAS	NGS	Quantity Not Sufficient
IDH1	NGS	Mutation Not Detected
JAK2	NGS	Mutation Not Detected
KDR (VEGFR2)	NGS	Mutation Not Detected
KRAS	NGS	Mutation Not Detected
MGMT	IHC	Negative
MPL	NGS	Mutation Not Detected
NOTCH1	NGS	Mutation Not Detected
NRAS	NGS	Mutation Not Detected
PD-1	IHC	Negative
PDGFRA	NGS	Mutation Not Detected
PD-L1	IHC	Negative
PGP	IHC	Negative
PIK3CA	NGS	Mutation Not Detected
PR	IHC	Negative
PTEN	NGS	Mutation Not Detected
PTEN	IHC	Positive
RET	NGS	Mutation Not Detected
RRM1	IHC	Positive
SMO	NGS	Quantity Not Sufficient
SPARC Polyclonal	IHC	Negative
TLE3	IHC	Negative
TOP2A	IHC	Positive
TOPO1	IHC	Negative
TP53	NGS	Mutated | R337H
TS	IHC	Positive
TUBB3	IHC	Positive
VHL	NGS	Mutation Not Detected

IHC: Immunohistochemistry **CISH:** Chromogenic *in situ* hybridization **NGS:** Next-Generation Sequencing

For Next-Generation Sequencing, a total of 35 genes were analyzed. The results above include genes most commonly associated with cancer and any additional mutations identified. No alterations were identified in 32 genes. For a complete list of genes tested, visit www.CarisMolecularIntelligence.com/profilemenu.

See the Appendix section for a detailed overview of the biomarker test results for each technology.

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✔ THERAPIES WITH **POTENTIAL BENEFIT**

Therapies	Test	Method	Result	Value†	Clinical Association				
					Potential Benefit	Decreased Potential Benefit	Lack of Potential Benefit	Highest Level of Evidence*	Reference
dacarbazine, temozolomide	MGMT	IHC	Negative	1+ 5%	✔			II-2 / Good	34, 35
doxorubicin, epirubicin, liposomal-doxorubicin	Her2/Neu	CISH	No Hybridization						
	PGP	IHC	Negative	0+ 100%	✔			II-1 / Fair	45, 46
	TOP2A	IHC	Positive	2+ 20%	✔			I / Good	43, 44

* The level of evidence for all references is assigned according to the Literature Level of Evidence Framework consistent with the US Preventive Services Task Force described further in the Appendix of this report. The data level of each biomarker-drug interaction is the highest level of evidence based on the body of evidence, overall clinical utility, competing biomarker interactions and tumor type from which the evidence was gathered.

†Refer to Appendix for detailed Result and Value information for each biomarker, including appropriate cutoffs, unit of measure, etc.

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✘ THERAPIES WITH **POTENTIAL LACK OF BENEFIT**

Therapies	Test	Method	Result	Value†	Clinical Association				
					Potential Benefit	Decreased Potential Benefit	Lack of Potential Benefit	Highest Level of Evidence*	Reference
abarelix, degarelix, goserelin, leuprolide, triptorelin	Androgen Receptor	IHC	Negative	0+ 100%			✔	II-3 / Good	2
	ER	IHC	Negative	0+ 100%			✔	I / Good	1
	PR	IHC	Negative	0+ 100%			✔	I / Good	1
abiraterone, bicalutamide, enzalutamide, flutamide	Androgen Receptor	IHC	Negative	0+ 100%			✔	I / Good	2, 3, 4, 5
anastrozole, exemestane, fulvestrant, letrozole, megestrol acetate, tamoxifen, toremifene	ER	IHC	Negative	0+ 100%			✔	I / Good	13, 16, 17, 18, 19, 20, 21, 22
	PR	IHC	Negative	0+ 100%			✔	I / Good	13, 14, 15, 16, 17, 18, 19, 20
capecitabine, fluorouracil, pemetrexed	TS	IHC	Positive	1+ 10%			✔	I / Good	23, 24, 25
dabrafenib, vemurafenib	BRAF	Next Gen SEQ	Mutation Not Detected				✔	I / Good	30, 31, 32, 33
docetaxel, paclitaxel	PGP	IHC	Negative	0+ 100%	✔			II-3 / Fair	36, 37
	TLE3	IHC	Negative	1+ 5%			✔	II-2 / Good	42
	TUBB3	IHC	Positive	2+ 30%			✔	I / Good	38, 39, 40, 41
gemcitabine	RRM1	IHC	Positive	2+ 50%			✔	I / Good	50
irinotecan, topotecan	TOPO1	IHC	Negative	1+ 60%			✔	II-1 / Good	56, 57, 58

* The level of evidence for all references is assigned according to the Literature Level of Evidence Framework consistent with the US Preventive Services Task Force described further in the Appendix of this report. The data level of each biomarker-drug interaction is the highest level of evidence based on the body of evidence, overall clinical utility, competing biomarker interactions and tumor type from which the evidence was gathered.

†Refer to Appendix for detailed Result and Value information for each biomarker, including appropriate cutoffs, unit of measure, etc.

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? THERAPIES WITH **INDETERMINATE BENEFIT**

(Biomarker results do not impact potential benefit or lack of potential benefit)

Therapies	Test	Method	Result	Value†	Clinical Association				
					Potential Benefit	Decreased Potential Benefit	Lack of Potential Benefit	Highest Level of Evidence*	Reference
ado-trastuzumab emtansine (T-DM1), pertuzumab, trastuzumab	Her2/Neu	CISH	No Hybridization						
	Her2/Neu	IHC	Negative	0+ 100%			✔	I / Good	6, 7, 8, 9, 10, 11, 12
carboplatin, cisplatin, oxaliplatin	BRCA1	Next Gen SEQ	Mutation Not Detected				✔	II-2 / Good	26, 27, 28, 29
	BRCA2	Next Gen SEQ	Mutation Not Detected				✔	II-2 / Good	26, 27, 28
everolimus, temsirolimus	PIK3CA	Next Gen SEQ	Mutation Not Detected			✔		II-2 / Good	47, 48, 49
imatinib	c-KIT	Next Gen SEQ	Mutation Not Detected				✔	II-2 / Good	51, 52
	PDGFRA	Next Gen SEQ	Mutation Not Detected				✔	II-3 / Good	53, 54, 55
lapatinib	Her2/Neu	CISH	No Hybridization						
	Her2/Neu	IHC	Negative	0+ 100%			✔	I / Good	59, 60, 61
nab-paclitaxel	SPARC Polyclonal	IHC	Negative	1+ 80%			✔	II-2 / Good	62, 63
vandetanib	RET	Next Gen SEQ	Mutation Not Detected					I / Good	64

* The level of evidence for all references is assigned according to the Literature Level of Evidence Framework consistent with the US Preventive Services Task Force described further in the Appendix of this report. The data level of each biomarker-drug interaction is the highest level of evidence based on the body of evidence, overall clinical utility, competing biomarker interactions and tumor type from which the evidence was gathered.

†Refer to Appendix for detailed Result and Value information for each biomarker, including appropriate cutoffs, unit of measure, etc.

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CLINICAL TRIALS CONNECTOR™

For a complete list of open, enrolling clinical trials visit MI Portal to access the **Clinical Trials Connector** . This personalized, real-time web-based service provides additional clinical trial information and enhanced searching capabilities, including, but not limited to:

- Location: filter by geographic area
- Biomarker(s): identify specific biomarkers associated with open clinical trials to choose from
- Drug(s): search for specific therapies
- Trial Sponsor: locate trials based on the organization supporting the trial(s)

Visit www.CarisMolecularIntelligence.com to view all matched trials.

CHEMOTHERAPY CLINICAL TRIALS (8)			
Drug Class	**Biomarker**	**Method**	**Investigational Agent(s)**
Alkylating agents (8)	MGMT	IHC	temozolomide

TARGETED THERAPY CLINICAL TRIALS (3)			
Drug Class	**Biomarker**	**Method**	**Investigational Agent(s)**
Cell cycle inhibitors (3)	TP53	Next Gen SEQ	MK-1775

( ) = represents the total number of clinical trials identified by the Clinical Trials Connector for the provided drug class or table.

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** See Appendix page 6 for Level of Evidence description.*

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** See Appendix page 6 for Level of Evidence description.*

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SOURCE	LEVEL OF EVIDENCE*
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** See Appendix page 6 for Level of Evidence description.*

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SPECIMEN(S) RECEIVED (GROSS DESCRIPTION)

Specimen ID: ABC-12345-YZ **Specimen Collected:** XX-Mon-2015

The specimen(s) consist of: 1 (A) Tissue Biopsy Formalin Vial - Client ID(ABC-12345-YZ) with the corresponding surgical pathology report labeled "ABC-12345-YZ". Received is a single formalin filled specimen container labeled with "sarcoma nodule peritoneal space". It contains a single portion of white firm tissue measuring 2.5 x 1.8 x 14.5 cm. The specimen is serially sectioned and entirely submitted in cassettes A1-A5.

Clinical History: Per the submitted documents, the patient is a 62 year-old female with leiomyosarcoma.

Pathologic Diagnosis: Sarcoma nodule peritoneal space: Leiomyosarcoma, consistent with clinical history of uterine leiomyosarcoma.

Interpretation (Caris Life Sciences Microscopic Diagnosis):

Soft tissue, peritoneal nodule: Leiomyosarcoma.

Electronic Signature XX-Mon-2015

By my electronic signature, I as the attending pathologist affirm that I have personally reviewed and examined microscopically the prepared slide(s) and that the above diagnosis has been made or confirmed by me.

Disclaimer

All of the individual assays that are available through Caris Molecular Intelligence™ were developed and validated by Caris MPI, Inc. d/b/a Caris Life Sciences® and their test performance characteristics were determined and validated by Caris Life Sciences pursuant to the Clinical Laboratory Improvements Amendments and accompanying regulations ("CLIA"). Some of the assays that are part of Caris Molecular Intelligence have been approved by the U.S. Food and Drug Administration (FDA). For any remaining assays, Caris MPI, Inc. is certified under CLIA to perform high complexity testing, including all of the assays that comprise the Caris Molecular Intelligence.

The CLIA certification number of Caris MPI, Inc. laboratory performing testing in connection with Caris Molecular Intelligence can be found at the bottom of each page. This report includes information about therapies that appear to be associated with clinical benefit based on NCCN Compendium® guidelines, relevance of tumor lineage, level of published evidence and strength of biomarker results. This report, neither ranks biomarkers listed nor therapies associated with such biomarkers, in order of potential or predicted efficacy, and such therapies may or may not be suitable for administration to a particular patient. A determination of biomarker results do not necessarily indicate pharmacologic effectiveness or lack thereof. This report does not guarantee or suggest that any particular agent will be effective with the treatment of any particular condition. Caris Life Sciences expressly disclaims and makes no representation or warranty whatsoever relating, directly or indirectly, to review of identified scientific literature, the conclusions drawn from such review or any of the information set forth in this report that is derived from such review, including information and conclusions relating to therapies that are included or omitted from this report.

Decisions regarding care and treatment should not be based on a single test such as this test or the information contained in this report. The decision to select any, all or none of the listed therapies resides solely within the discretion of the treating physician. 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, including but not limited to, patient and family history, physical examinations, information from other diagnostic tests, and patient preferences, in accordance with the applicable standard of care.

The information presented in the Clinical Trials Connector™ section of this report (if applicable) is compiled from sources believed to be reliable and current. We have used our best efforts to make this information as accurate as possible. However, the accuracy and completeness of this information cannot be guaranteed. The contents are to be used for clinical trial guidance and may not include all relevant trials. Current enrollment status for these trials is unknown. The clinical trials information present in the biomarker description was compiled from www.clinicaltrials.gov. The contents are to be used only as a guide, and health care providers should employ their judgment in interpreting this information for a particular patient. Specific eligibility criteria for each clinical trial should be reviewed as additional inclusion criteria may apply. Caris Life Sciences makes no promises or guarantees that a healthcare provider, insurer or other third party private or government payor, will provide reimbursement for any of the tests performed.

The next-generation sequencing assay performed by Caris Life Sciences examines nucleic acids obtained from tumor tissue only and does not examine normal tissue such as tumor adjacent tissue or whole or peripheral blood. As such, the origin of any mutation detected may be a somatic mutation (not inherited) or a germline mutation (inherited) and will not be distinguishable by this assay. It is recommended that results be considered within the patient's clinical and health history. If a germline inheritance pattern is suspected then counseling by a board certified genetic counselor is recommended.

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A laboratory technician harvested targeted tissues for extraction from the marked areas using a dissection microscope. The areas marked and extracted were microscopically reexamined on post-scraped slides and adequacy of scraping was verified by a board certified Pathologist.

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MI PROFILE™
MOLECULAR INTELLIGENCE TUMOR REPORT

CARIS® LIFE SCIENCES

MUTATIONAL ANALYSIS BY **NEXT-GENERATION SEQUENCING (NGS)**

GENES TESTED WITH ALTERATIONS

Gene	Alteration	Frequency (%)	Exon	Result
TP53	R337H	85	10	Mutated, Pathogenic

Interpretation: A TP53 mutation was detected in this sample. This mutation affects the tetramerization of the TP53 therefore renders the protein nonfunctional. Mutations at the Arginine residue 337 (R337) have been reported for various tumor types. It should be noted that mutations at residue 337 have been observed more frequently as germline mutations (causing Li-Fraumeni Syndrome) than somatic mutations.

TP53, or p53, plays a central role in modulating response to cellular stress through transcriptional regulation of genes involved in cell-cycle arrest, DNA repair, apoptosis, and senescence. Inactivation of the p53 pathway is essential for the formation of the majority of human tumors. Mutation in p53 (TP53) remains one of the most commonly described genetic events in human neoplasia, estimated to occur in 30-50% of all cancers. Generally, presence of a disruptive p53 mutation is associated with a poor prognosis in all types of cancers, and diminished sensitivity to radiation and chemotherapy. In addition, various clinical trials (on www.clinicaltrials.gov) investigating agents which target p53's downstream or upstream effectors may have clinical utility depending on the p53 status. Germline p53 mutations are associated with the Li-Fraumeni syndrome (LFS) which may lead to early-onset of several forms of cancer currently known to occur in the syndrome, including sarcomas of the bone and soft tissues, carcinomas of the breast and adrenal cortex (hereditary adrenocortical carcinoma), brain tumors and acute leukemias.

GENES TESTED WITH NO MUTATIONS DETECTED

ABL1	BRAF	EGFR	GNA11	KDR	PDGFRA
AKT1	c-KIT	ERBB2	GNAQ	KRAS	PIK3CA
ALK	cMET	FGFR1	GNAS	MPL	PTEN
APC	CSF1R	FGFR2	IDH1	NOTCH1	RET
ATM	CTNNB1	FLT3	JAK2	NRAS	VHL

GENES TESTED WITH QNS RESULTS (QUANTITY NOT SUFFICIENT)

HRAS	SMO

For Next-Generation Sequencing, a total of 35 genes were analyzed. The results above include genes most commonly associated with cancer and any additional mutations identified. No alterations were identified in 32 genes. For a complete list of genes tested, visit www.CarisMolecularIntelligence.com/profilemenu.

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NGS Methods

Direct sequence analysis was performed on genomic DNA isolated from a formalin-fixed paraffin-embedded tumor sample using the Illumina MiSeq platform. Specific regions of the genome were amplified using the Illumina TruSeq Amplicon Cancer Hotspot panel. This panel only sequences selected regions of 44 genes and the amino acids sequenced by this assay can be found at www.carislifesciences.com. All variants reported by this are detected with >99% confidence based on the frequency of the mutation present and the amplicon coverage. This test is not designed to distinguish between germ line inheritance of a variant or acquired somatic mutation. This test has a sensitivity to detect as low as approximately 10% population of cells containing a mutation a sequenced amplicon. This test has not been cleared or approved by the United States Food and Drug Administration (FDA) as such approval is not necessary. All performance characteristics were determined by Caris Life Sciences. Insertions or deletions larger than 27 bp will not be detected by this assay. Benign and non-coding variants are not included in this report but are available upon request.

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MUTATIONAL ANALYSIS BY **NEXT-GENERATION SEQUENCING (NGS)**

GENES TESTED WITH NO MUTATIONS DETECTED	
BRCA1	BRCA2

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BRCA1 Sequencing Methods

Direct sequence analysis was performed on genomic DNA isolated from a formalin-fixed paraffin-embedded tumor sample using the Illumina MiSeq platform. Specific regions of BRCA1 were amplified using primers flanking coding regions of this gene. All variants reported by this are detected with >99% confidence based on the frequency of the mutation present and the amplicon coverage. This test is not designed to distinguish between germ line inheritance of a variant or acquired somatic mutation. This test has a sensitivity to detect as low as approximately 20% population of cells containing a mutation a sequenced amplicon. This test has not been cleared or approved by the United States Food and Drug Administration (FDA) as such approval is not necessary. All performance characteristics were determined by Caris Life Sciences. Insertions or deletions larger than 27 bp will not be detected by this assay. Benign and non-coding variants are not included in this report but are available upon request.

BRCA2 Sequencing Methods

Direct sequence analysis was performed on genomic DNA isolated from a formalin-fixed paraffin-embedded tumor sample using the Illumina MiSeq platform. Specific regions of BRCA2 were amplified using primers flanking coding regions of this gene. All variants reported by this are detected with >99% confidence based on the frequency of the mutation present and the amplicon coverage. This test is not designed to distinguish between germ line inheritance of a variant or acquired somatic mutation. This test has a sensitivity to detect as low as approximately 20% population of cells containing a mutation a sequenced amplicon. This test has not been cleared or approved by the United States Food and Drug Administration (FDA) as such approval is not necessary. All performance characteristics were determined by Caris Life Sciences. Insertions or deletions larger than 27 bp will not be detected by this assay. Benign and non-coding variants are not included in this report but are available upon request.

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PROTEIN EXPRESSION BY **IMMUNOHISTOCHEMISTRY (IHC)**

Biomarker	Patient Tumor			Threshold * Biomarker Intensity/Percentage
	Staining Intensity	Percent Staining	Result	
SPARC Polyclonal	1	80	Negative	<30% or <2+ or ≥2+ and ≥30%
ER	0	100	Negative	=0+ or <10% or ≥1+ and ≥10%
PR	0	100	Negative	=0+ or <10% or ≥1+ and ≥10%
Androgen Receptor	0	100	Negative	=0+ or <10% or ≥1+ and ≥10%
TOPO1	1	60	Negative	=0+ or <30% or <2+ or ≥2+ and ≥30%
TOP2A	2	20	Positive	=0+ or <10% or ≥1+ and ≥10%
TLE3	1	5	Negative	<30% or <2+ or ≥2+ and ≥30%
TUBB3	2	30	Positive	<30% or <2+ or ≥2+ and ≥30%
PGP	0	100	Negative	=0+ or <10% or ≥1+ and ≥10%
EGFR	0	100	Negative	=0+ or <10% or ≥1+ and ≥10%
Her2/Neu	0	100	Negative	≤1+ or =2+ and ≤10% or ≥3+ and >10%
cMET	0	100	Negative	<50% or <2+ or ≥2+ and ≥50%
MGMT	1	5	Negative	=0+ or ≤35% or ≥1+ and >35%
PD-L1	1	5	Negative	<5% or <2+ or ≥2+ and ≥5%
PTEN	3	100	Positive	=0+ or ≤50% or ≥1+ and >50%
RRM1	2	50	Positive	=0+ or <50% or <2+ or ≥2+ and ≥50%
TS	1	10	Positive	=0+ or ≤3+ and <10% or ≥1+ and ≥10%

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IHC Methods

These tests were developed and their performance characteristics determined by Caris MPI, Inc. d/b/a Caris Life Sciences®

* Caris Life Sciences has defined threshold levels of reactivity of IHC to establish cutoff points based on published evidence. Polymer detection systems are used for each IHC.

* Please note that PD-L1 staining is read from the cytoplasmic or membrane staining of cancer cells.

Clones used: SPARC Polyclonal(Polyclonal), ER(SP1), PR(1E2), Androgen Receptor(AR27), TOPO1(1D6), TOP2A(3F6), TLE3(Polyclonal), TUBB3(Polyclonal), PGP(C494), EGFR(H11), Her2/Neu(4B5), cMET(SP44), MGMT(MT23.2), PD-L1(130021), PTEN(6H2.1), RRM1(Polyclonal), TS(TS106/4H4B1).

Additional IHC results continued on the next page. >

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PROTEIN EXPRESSION BY **IMMUNOHISTOCHEMISTRY (IHC)**

Biomarker	TIL Count/HPF w/40X Objective	Result	Threshold *
PD-1	0/HPF	Negative	=0+ or ≥1+

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IHC Methods

These tests were developed and their performance characteristics determined by Caris MPI, Inc. d/b/a Caris Life Sciences®

* Please note that PD1 staining is read from the tumor infiltrating lymphocytes (TIL).Clones used: PD-1(MRQ-22).

Clones used: PD-1(MRQ-22).

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AMPLIFICATION BY **CHROMOGENIC *IN SITU* HYBRIDIZATION (CISH)**

Gene / ISCN	Cells Counted	Result	Total/Avg Gene Copy Number	Total/Avg Control Copy Number	Cells with ≥4 Copies	Cells with ≥15 Copies	Ratio Calculation	Ratio
Her2/Neu nuc ish (D17Z1x1-2,HER2x1-2)[/30]		No Hybridization			N/A	N/A	Her2/neu/ Chromosome 17	
	Reference Range: Her2/Neu:CEP 17 signal ratio of >= 2.0; and non-amplification as <2.0 per Ventana INFORM HER2 CISH Package insert.							
cMET nuc ish (D7Z1x1-2,cMETx1-2)[100/100]		No Hybridization			N/A	N/A		
	Reference Range: Positivity for increased gene copy number for cMET CISH has been defined as >= 5 copies of mean MET gene copy number per cell in NSCLC based on cMET FISH evidence (Cappuzzo et al 2009). The gene copy number threshold for other tumor types has not been determined.							

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CISH Methods

HER2 CISH test was carried out using the INFORM DUAL HER2 ISH Assay (Ventana Medical Systems, Inc.), which has been cleared by the US Food and Drug Administration (FDA) for enumerating the ratio of HER2/Chr 17 in Breast Cancer samples. The HER2 CISH testing for cancer lineages other than breast has been developed and their performance characteristics determined by Caris MPI, Inc. (d/b/a Caris Life Sciences), and have not been cleared or approved by the FDA.

cMET CISH was carried out using a probe specific for cMET and a probe for the pericentromeric region of chromosome 7 (Ventana). The cMET CISH testing for cancer lineages has been developed and its performance characteristics determined by Caris MPI, Inc. (d/b/a Caris Life Sciences), and has not been cleared or approved by the FDA.

TOP2A CISH was carried out using a probe specific for TOP2 and a probe for the pericentromeric region of chromosome 17 (Ventana). The TOP2A CISH testing for cancer lineages has been developed and its performance characteristics determined by Caris MPI, Inc. (d/b/a Caris Life Sciences), and has not been cleared or approved by the FDA.

The FDA has determined that such clearance or approval is not currently necessary. These tests should not be regarded as investigational or research as they are used for clinical purpose and determined to be medically necessary by the ordering physician, who is not employed by Caris MPI, Inc. or its affiliates. This laboratory is certified under Clinical Laboratory Improvement Amendment of 1988 (CLIA-88) and is qualified to perform high complexity testing. CLIA 03D1019490

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LITERATURE LEVEL OF EVIDENCE ASSESSMENT FRAMEWORK*

STUDY DESIGN	
Hierarchy of Design	**Criteria**
I	Evidence obtained from at least one properly designed **randomized controlled trial**.
II-1	Evidence obtained from well-designed controlled trials **without randomization**.
II-2	Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group.
II-3	Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled trials might also be regarded as this type of evidence.
III	Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled trials might also be regarded as this type of evidence.

STUDY VALIDITY	
Grade	**Criteria**
Good	The study is judged to be valid and relevant as regards results, statistical analysis, and conclusions and shows no significant flaws.
Fair	The study is judged to be valid and relevant as regards results, statistical analysis, and conclusions, but contains at least one significant but not fatal flaw.
Poor	The study is judged to have a fatal flaw such that the conclusions are not valid for the purposes of this test.

* Adapted from Harris, T., D. Atkins, et al. (2001). "Current Methods of the U.S. Preventive Services Task Force." Am J Prev Med 20(3S)

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