L J Corea

AGE: 61 years | **Gender:** Male | **Coupon/UID:** NA **/** AOM0286

Diagnosis: Colorectal Neoplasms

MEDGENOME

MGM2529 : Colorectal advanced panel by NGS & Microsatellite Instability (MSI) by fragment analysis

Report Details		Specimen Information		Ordering Clinician	
Sample ID / Order ID:	9564955 / 1528738	**Specimen Site:**	Colon	**Clinician:**	Dr. Mahendra Perera
Collection Date:	20th November 2025	**Specimen Received:**	FFPE Tissue Blocks [1]	**Affiliation:**	Aegle Omics Private Limited
Date Received:	20th November 2025	**Specimen Tested:**	JH1813 C	**Serviced By:**	18718
Report Date & Time:	6th Dec 2025 15:41 PM	**Tumor Content (%):**	70	**Report Status:**	Final

Clinical Summary:

R/S Hemicolectomy specimen: well differentiated adenocarcinoma; pT4 pN0.

TEST RESULT SUMMARY

Microsatellite Instability (MSI) Test

Status - Stable

Kindly refer to the complete MSI reports below.

Next Generation Sequencing (NGS) Results

POSITIVE

Gene	Findings	Gene	Findings
AKT1	Not Detected	*APC*	Not Detected
BRAF	**V600E**	*ERBB2*	Not Detected
HRAS	Not Detected	*KRAS*	Not Detected
MET	Not Detected	*NRAS*	Not Detected
NTRK1	Not Detected	*NTRK2*	Not Detected
NTRK3	Not Detected	*PIK3CA*	Not Detected
PIK3R1	Not Detected	*POLD1*	Not Detected
POLE	Not Detected	*PTEN*	Not Detected
RET	Not Detected	*SMAD4*	Not Detected
TP53	**Frameshift insertion**		

Please refer to the complete variant details in the result table in page 2.

Next Generation Sequencing (NGS) Test Result

Result - POSITIVE
CLINICALLY RELEVANT VARIANT/S DETECTED

AMP Classification ^	CDS variant details	Interpretation	Treatment Recommendations	$Treatment Response
***BRAF* p.Val600Glu (MISSENSE)** Variant Allele Frequency - 16.61%				
Tier I	c.1799T>A (ENST00000646891.2)	Oncogenic	Sensitive to BRAF inhibitors	Effective
***TP53* p.Cys135TrpfsTer14 (FRAMESHIFT-INS)** Variant Allele Frequency - 32.49%				
Tier II	c.404dup (ENST00000269305.9)	Oncogenic	NA	Diagnostic

No clinically significant fusion has been detected in this sample

^Refer to Glossary section for the classification criteria details.

$Drug Approvals are based on US-FDA Guidelines. Kindly refer to local guidelines if required.

ADDITIONAL BIOMARKERS DETECTED

This section provides information about variants that do not have any therapeutic value. However, these variants may or may not have a likely oncogenic effect.

No other biomarkers that warrants to be reported was detected

ACTIONABLE BIOMARKER DETAILS

BRAF (p.Val600Glu) - MISSENSE

Gene: *BRAF*	**Exon:** 15	**Variant Allele Frequency:** 16.61%
Nucleotide change: chr7:g.140753336A>T	**Protein change:** p.Val600Glu	**Population MAF:** 0 (1000G);0(gnomAD);
cDNA change: c.1799T>A	**Variant Type:** MISSENSE	**In-silico Predictions:** D_lc(SIFT); D(LRT); PrD(Polyphen2)
Transcript ID: ENST00000646891.2	**Variant Allele Depth/Total depth:** 52/313x	**Gene Function:** Oncogene

Gene Summary: *BRAF* encodes a protein belonging to the RAF family of serine/threonine protein kinases. This protein plays a role in regulating the MAP kinase/ERK signaling pathway, which affects cell division, differentiation, and secretion. Mutations in *BRAF*, most commonly the V600E mutation, are the most frequently identified cancer-causing mutations in melanoma, and have been identified in various other cancers as well, including non-Hodgkin lymphoma, colorectal cancer, thyroid carcinoma, non-small cell lung carcinoma, hairy cell leukemia and adenocarcinoma of lung. Mutations in *BRAF* are also associated with cardiofaciocutaneous, Noonan, and Costello syndromes, which exhibit overlapping phenotypes. A pseudogene of *BRAF* has been identified on the X chromosome.

Clinical and Therapeutic Relevance: The serine/threonine-protein kinase BRAF activates the RAS/MAPK signaling pathway to promote cell proliferation and survival. This mutation, located in the catalytic domain, results in constitutive activation of the RAS/RAF/MEK signaling pathway. **Encorafenib, in combination with cetuximab alone or in triple combination with mFOLFOX6, is approved for the treatment of patients with metastatic colorectal cancer (CRC) harboring this mutation. In a phase III study (BEACON CRC), the triple combination of encorafenib, binimetinib, and cetuximab resulted in an overall response rate (ORR) of 26% in CRC patients with *BRAF*.V600E.**A phase I study in CRC patients with this mutation has shown that encorafenib, in combination with cetuximab and the PI3Kα-inhibitor alpelisib, demonstrated an ORR of 18%. Some tumor responses have been reported with the combination treatment of panitumumab with vemurafenib. Also, the combination of panitumumab and encorafenib has resulted in a PR and SD in two patients, respectively, but preclinical confirmation of this sensitivity is missing. Furthermore, in a phase II basket trial, the treatment of *BRAF*-mutant CRC patients (n=31) with trametinib, dabrafenib, and spartalizumab resulted in a confirmed ORR of 25% and a disease control rate (DCR) of 75%. The combination of vemurafenib and cetuximab with FOLFIRI achieved an ORR of 81% (17/21 patients) with 15 PRs and 2 complete responses (CRs). In a small cohort study, 7 of 19 patients achieved a PR and 11 patients achieved SD with this treatment combination. Furthermore, the triple combination of vemurafenib, cetuximab, and camrelizumab resulted in CR in one case. Also, in a small cohort study (n=31) the combination of vemurafenib and erlotinib resulted in an ORR of 16% (5/31) with median progression-free survival (PFS) of 3.9 months and median overall survival (OS) of 6.3 months. However, vemurafenib monotherapy was found to be ineffective in CRC, with an ORR of about only 5% for BRAF-mutated tumors. Nonetheless, one case report described a patient who was treated for 10 months with triple therapy of dabrafenib, trametinib, and cetuximab and achieved CR. Due to severe skin toxicity, cetuximab was discontinued, and the double therapy of dabrafenib and trametinib was continued for 41 months. CRC cell lines harboring a *BRAF*.V600E mutation have shown sensitivity to lifirafenib, plixorafenib (PLX8394), and to combination treatments with trametinib and dabrafenib; trametinib and dabrafenib and cetuximab; cetuximab and dabrafenib; vemurafenib and cetuximab; cetuximab and selumetinib; encorafenib with cetuximab and alpelisib, as well as to the combination treatment encorafenib and cetuximab. The cells were resistant to treatment with panitumumab or cetuximab. Dabrafenib is not indicated for CRC patients; also, in combination with trametinib, the response rate (12%) was very low in CRC patients because of intrinsic resistance to BRAF inhibitors.

BRAF (p.Val600Glu) - MISSENSE

PubMed References: 37213293, 36713544, 36702949, 36638198, 36409971, 35970034, 35074651, 33356422, 33204026, 31566309, 30220966, 29431699, 28363909, 28179366, 27834212, 27729313, 27312529, 26460303, 26208524, 25589621, 19001320, 38178783, 27864688, 20023270, 19614767

TP53 (p.Cys135TrpfsTer14) - FRAMESHIFT-INS

Gene: *TP53*	**Exon:** 5	**Variant Allele Frequency:** 32.49%
Nucleotide change: chr17:g.7675208dup	**Protein change:** p.Cys135TrpfsTer14	**Population MAF:** 0 (1000G);0(gnomAD);
cDNA change: c.404dup	**Variant Type:** FRAMESHIFT-INS	**In-silico Predictions:** NA(SIFT); NA(LRT); NA(Polyphen2)
Transcript ID: ENST00000269305.9	**Variant Allele Depth/Total depth:** 167/514x	**Gene Function:** Tumor Suppressor Gene

Gene Summary: *TP53* encodes a tumor suppressor protein containing transcriptional activation, DNA binding, and oligomerization domains. The encoded protein responds to diverse cellular stresses to regulate expression of target genes, thereby inducing cell cycle arrest, apoptosis, senescence, DNA repair, or changes in metabolism. Mutations in *TP53* are associated with a variety of human cancers, including hereditary cancers such as Li-Fraumeni syndrome. Alternative splicing of *TP53* and the use of alternate promoters result in multiple transcript variants and isoforms. Additional isoforms have also been shown to result from the use of alternate translation initiation codons from identical transcript variants (PMIDs: 12032546, 20937277).

Clinical and Therapeutic Relevance: *TP53* is a well-characterized tumor suppressor gene that is involved in regulating cell cycle, apoptosis, and DNA repair and damage response. Frameshift and nonsense mutations in the TP53 gene are likely to confer loss of its tumor suppressor activity. Some *TP53* mutants were associated with a higher frequency of platinum and taxane-based chemoresistance and distant metastasis in ovarian carcinoma. Preclinical data suggests loss of TP53 tumor suppressor activity confers sensitivity towards the Wee1-inhibitor adavosertib, which may be further enhanced by combinational treatment with DNA-damaging agents. Early-phase clinical trials in *TP53*-mutant ovarian cancer show a slight but significant improved response to chemotherapy in combination with adavosertib compared to placebo. In early-phase clinical studies, treatment with the VEGF-targeted antiangiogenic agent bevacizumab in combination with chemotherapy resulted in significantly prolonged PFS and OS in patients with *TP53* mutated advanced solid tumors compared to treatment with temsirolimus and chemotherapy. This difference was not seen in *TP53* wild-type tumors, suggesting mutated T*P53* as a potential biomarker for sensitivity towards bevacizumab. *TP53* germline mutations are found in about 70% of families with Li-Fraumeni syndrome, a cancer predisposition syndrome which greatly increases the risk of developing several types of cancer, particularly in children and young adults. About 22% of the cancers develop in the childhood phase (0-15 years), especially, adrenal cortical carcinoma, rhabdomyosarcoma, and medulloblastoma. During early adulthood (16–50 years) about 51% of cancers arise, including breast cancer, osteosarcoma, soft tissue sarcomas, leukemia, astrocytoma, glioblastoma, colorectal, and lung cancer. In the stage of late adulthood (51–80 years), 27% of cancers develop, especially pancreatic and prostate cancers. Somatic mutations in this gene are among the most common genetic changes found in human cancer. *TP53* mutations have been identified in diverse tumors such as breast, ovarian, lung, bladder cancer, and tumors of the central nervous system.

PubMed References: 36081565, 32611648, 32047167, 31366114, 27998224, 25385265, 40525786, 39133932, 39133921, 37505914, 37483562, 36892252, 36287260, 35797463, 35732829, 33091559, 29540348, 28819011, 26837699, 24501221, 23243274, 21720382, 20421449, 16461462, 37505914, 36892252, 36287260, 35954327, 33240819, 25584008, 25201186, 23175693, 20308654

AMP-ASCO-CAP CLASSIFICATION CRITERIA

Genetic test results are reported based on the somatic variant classification recommendations of College of American Pathologists (CAP) /American society for Clinical Oncology (ASCO)/Association of Molecular Pathologists (AMP) [PMID: 27993330] as described in the table below:

Tier	Criteria
Tier I	Variants of strong clinical significance.
Tier II	Variants of potential clinical significance.
Tier III	Variants of unknown clinical significance
Tier IV	Benign or likely benign variants

DISCLAIMER

- **Decisions regarding treatment action plans should not be solely based on these test results. These findings are highly recommended to be correlated with the patient's clinical, pathological, radiological and family history for decisions on diagnosis, prognosis, or therapeutics.**
- The therapy information provided in this report is based on FDA approved drugs data, NCCN guidelines, peer reviewed published literature, standard clinical databases, and strength of biomarker results till date. These therapies may or may not be suitable/beneficial to a particular patient. This clinical report summarises potentially effective medications, potentially ineffective medications, and medications that may pose a higher risk of adverse reactions by mapping the patient's genetic alterations to the biomedical reference information. The report may also provide prognostic and diagnostic biomarkers detected or shown for the given disease context.The treatment recommendations for the variants classified in Tier II are not provided.
- The clinical trials information provided in this report is compiled from www.clinicaltrials.gov as per currently available data, however completeness of information provided herein cannot be guaranteed. This information should only be used as a guide and specific eligibility criteria should be reviewed thoroughly for the concerned patient. MedGenome Labs does not guarantee or promise an enrolment in any clinical trials.
- The identification of a genomic biomarker does not necessarily imply pharmacological effectiveness or ineffectiveness. The medications identified by the treating physician may or may not be suitable for use on a particular patient. Thus, the clinical report does not guarantee that any particular agent will be effective in the treatment of any particular condition. Also, the absence of a treatment option does not determine the effectiveness or predict an ineffective or safety-relevant effect of a medication selected by the treating physician.
- The classification and clinically relevant information for the reported variants is based on peer-reviewed publications, public clinical databases, medical guidelines (NCCN, ASCO, AMP) or other publicly available information and it has been ensured that the information provided is up to date at the time of report generated, however continuous updates may happen in public domains. Also, the classification of variants can change based on the updated literature evidence. Re-analysis of the results can be requested at additional cost.
- This test is performed on the patient's tumor sample without a paired blood sample; therefore, it may include variations which may be of germline origin. However, this test is designed and validated for the detection and reporting of somatic genomic variants only and does not discriminate between germline and somatic variants. If clinically warranted, appropriate germline testing and genetic counselling for the patient should be considered for further evaluation.
- Due to poor quality of FFPE tissue blocks, the QC parameters for extracted RNA may not pass to proceed further with the testing, therefore there is a possibility of assay failure at various steps (RNA QC, Library QC, Bioinformatics QC) or compromised results that include low gene coverage and low variant depth. However, sample status in such scenarios shall be sent through mail to the ordering clinician.
- This test has been validated at MedGenome Labs and the limit of detection (LOD) of allele fraction for SNVs and InDels is ≥5% and for fusions is ≥10 spanning reads. However, the report may include, at the discretion of laboratory director, the variants with lower allele burden (3-5%) having strong or potential clinical significance or those have been reported earlier in the patient. Variants with <1% allele

fraction and variants of uncertain significance with <5% allele fraction are not routinely reported. However, possibility of false negative or false positive below the limit of detection of this assay cannot be ruled out.

- Large deletions and deep intronic variations are not detected in this assay.
- Copy Number Variations (CNVs) are based on the RNA expression data using a CNV prediction model developed with control samples. Hence, the chromosome coordinates and size of the CNV can not be determined. It is recommended to confirm the CNVs by alternate methods, such as FISH as the sensitivity of NGS for detecting CNVs is not 100%.
- **Additional case specific disclaimer : None**

TEST DESCRIPTION

The MedGenome's Colorectal panel is a high throughput next-generation sequencing assay covering key genes to detect SNVs, InDels, CNVs and fusions and aids in diagnosis, prognosis and therapeutics of the colorectal cancer patients.

TEST METHODOLOGY

Sample type: FFPE Specimen; A histopathologic review is performed to determine the tumor content in the FFPE block/curls.

Extraction and Library Preparation: Tumor nucleic acid is extracted from FFPE (Formalin fixed) tissue block and used to perform targeted gene capture using a custom hybrid capture kit.

Sequencing: The QC passed libraries are sequenced to a minimum depth of 250X on validated Illumina sequencing platform.

Data Analysis: The sequences are processed using a customized and validated analysis pipeline designed to accurately detect all classes of genomic alterations (SNVs, InDels, CNVs and Fusions).

Variant Annotation and Reporting: The variants are annotated using our in-house annotation pipeline. Reportable genomic alterations and fusions are prioritized, classified, and reported based on AMP-ASCO-CAP guidelines [PMID: 27993330] and NCCN guidelines.

Limit of Detection (LOD): The LOD for SNVs and InDels is 5% Variant allele Frequency (VAF) and for fusions is >10 spanning reads.

The transcript used for clinical reporting generally represents the canonical transcript (according to Ensembl release 99 human gene model), which is usually the longest coding transcript with strong/multiple supporting evidence. However, clinically relevant variants annotated in alternate complete coding transcripts could also be reported. Variants annotated on incomplete, and nonsense mediated decay transcripts are not reported.

§This test is developed, and its performance characteristics is determined by MedGenome Labs Ltd.

GENES ANALYSED

SNVs/InDels							
AKT1	*APC*	*BRAF*	*ERBB2*	*HRAS*	*KRAS*	*MET*	*NRAS*
POLE	*POLD1*	*PIK3R1*	*PIK3CA*	*PTEN*	*SMAD4*	*TP53*	

Note: *MET* exon 14 skipping mutations included.

CNVs	
ERBB2	*MET*

FUSIONS				
MET	*NTRK1*	*NTRK2*	*NTRK3*	*RET*

CLINICAL TRIALS

The following trials are potentially best suited for your patient's indication, considering all reported treatment recommendations. See https://clinicaltrials.gov (clinical trials from NCT) or https://trialsearch.who.int (clinical trials from other registries) for more information.

Clinical trials in total : 0 **Trial countries :** IN-India, US-United States

S.No	Title	Phase and ID	Intervention	Disease	Age & Sex
No Clinical Trials.					

Aparna Natarajan, Ph.D
Lead - Genome Analyst (Oncology)

Dr. Syed Muqlisur Rehman, MD Path
Molecular Pathologist
KMC Registration No. 71468

END OF REPORT

L J Corea

AGE: 61 years | **Gender:** Male | **Coupon/UID:** NA / AOM0286

Diagnosis: Colorectal Neoplasms

MEDGENOME

MGM527: Microsatellite Instability (MSI) by fragment analysis

Report Details		Specimen Information		Ordering Clinician	
Sample ID / Order ID:	9564955 / 1528738	**Specimen Site:**	Colon	**Clinician:**	Dr. Mahendra Perera
Collection Date:	20th November 2025	**Specimen Received:**	FFPE Tissue Blocks [1]	**Affiliation:**	Aegle Omics Private Limited
Date Received:	20th November 2025	**Specimen Tested:**	JH1813 C	**Serviced By:**	18718
Report Date & Time:	28th Nov 2025 15:26 PM	**Tumor Content (%):**	70	**Report Status:**	Final

Clinical Summary:

Adenocarcinoma colon

Kindly note that this is the MSI report. The final NGS report including the status of SNVs & Indels, Fusions and CNVs will be released on or before 06-12-2025 based on the QC status.

TEST RESULT SUMMARY

Microsatellite Instability (MSI) Status - Stable

Summary of Markers	
Count of markers reported Unstable	0
Count of markers reported Stable	12
Reported Unstable Rate	0.00%
Unstable Markers	None

CLINICAL SIGNIFICANCE

- MSI screening has long been recognized as important in the care of patients with colorectal cancer (CRC) or endometrial cancer (EC).
- High-frequency MSI (MSI-H) is also recognized as a potential marker for germline mutations in certain DNA mismatch repair (MMR) genes associated with Lynch syndrome [PMID: 15872200].
- MSI has been found in several cancer types, including non-small cell lung cancer, melanoma, breast cancer, urothelial cancer, pancreatic ductal adenocarcinoma and brain cancer. The expansion of MSI clinical trials into other cancers may elucidate the prognostic and predictive value of MSI for non-colorectal [PMID: 35955855].
- NCCN® guidelines recommend universal screening for 15+ different cancer types by MSI and/or IHC analysis [www.nccn.org]
- MSI-H status is predictive of a positive response to immunotherapies such as immune checkpoint blockade inhibitors [PMID: 26028255]
- The 2015 paper by Le et al. reported the extended analysis on the efficacy of PD-1 blockade in patients with advanced mismatch repair-deficient cancers of both colorectal cancer and non-colorectal origins. Following 41 patients, the study found that patients with mismatch repair deficient tumors, experienced an objective response rate of 40% and a progression-free survival rate of 78%. In contrast, the objective response rate was 0% and the progression-free survival rate was 11% for mismatch repair-proficient

- The College of American Pathologists (CAP),in collaboration with the Association of Molecular Pathology (AMP),American Society of Clinical Oncology (ASCO), and patient advocacy group Fight Colorectal Cancer (Fight CRC) convened a multidisciplinary expert and advisory panel to develop evidence-based guidelines to identify the optimal clinical laboratory test to identify defects in DNA mismatch repair (dMMR) in patients with solid tumor malignancies who are being considered for immune checkpoint inhibitor (ICI) therapy. MSI by PCR was recommended for colorectal cancer, patients with gastroesophageal and small bowel cancer and other solid malignancies [PMID: 35920830].
- On June 29, 2020, the Food and Drug Administration approved pembrolizumab (KEYTRUDA, Merck & Co.) for the first-line treatment of patients with unresectable or metastatic microsatellite instability-high (MSI-H) or mismatch repair deficient (dMMR) colorectal cancer [www.fda.gov].
- The FDA approved pembrolizumab on May 23, 2017, for the treatment of adult and pediatric patients with unresectable or metastatic, microsatellite instability-high (MSI-H), or mismatch repair deficient (dMMR) solid tumors that have progressed following prior treatment and who have no satisfactory alternative treatment options and for the treatment of unresectable or metastatic MSI-H or dMMR colorectal cancer that has progressed following treatment with a fluoropyrimidine, oxaliplatin, and irinotecan [www.fda.gov].

DISCLAIMER

- **Decisions regarding treatment action plan should not be solely based on these test results. These findings are highly recommended to be correlated with the patient's clinical, pathological, radiological and family history for decisions on diagnosis, prognosis, or therapeutics.**
- The therapy information provided in this report is based onFDA approved drugs data, NCCN guidelines, peer-reviewed published literature, standard clinical databases, and strength of biomarker results. These therapies may or may not be suitable/beneficial to a particular patient. This clinical report summarizes potentially effective medications, potentially ineffective medications, and medications that may pose a higher risk of adverse reactions by mapping the patient's genetic alterations to the biomedical reference information. The report may also provide prognostic and diagnostic biomarkers detected or shown for the given disease context.
- The identification of a genomic biomarker does not necessarily imply pharmacological effectiveness or ineffectiveness. The medications identified by the treating physician may or may not be suitable for use on a particular patient. Thus, the clinical report does not guarantee that any particular agent will be effective in the treatment of any particular condition. Also, the absence of a treatment option does not determine the effectiveness or predict an ineffective or safety-relevant effect of a medication selected by the treating physician.
- Due to poor quality of FFPE tissue blocks, the QC parameters for extracted DNA may not pass to proceed further with the testing, therefore there is a possibility of assay failure or compromised. However, sample status in such scenarios shall be sent through mail to the ordering clinician.
- This test has been validated at MedGenome Labs as per the CAP guidelines with 100% sensitivity and specificity.
- The results of this test are dependent on the tumor content in the tissue sample provided. A minimum of >10% tumour content is required for a successful testing.
- In case of MSI negative or MSS patients, if there is a co-existing strong personal or family history of HNPCC related cancers for this patient, consider microsatellite instability and IHC testing on a different tumor block to further evaluate the possible role of defective DNA mismatch repair.
- **Additional case specific disclaimer: In this case, the markers "ABI-20A" had failed in amplification. Hence, the MSI status of this subject has been interpreted based on the status of 12 out of 13 markers. Kindly correlate clinically.**

TEST METHODOLOGY

This assay detects the presence of microsatellite instability (MSI) in DNA samples through multiplex PCR [1] and fragment analysis and screens for 13 mononucleotide markers listed in table below. Mononucleotide markers like BAT-25, BAT-26 and BAT-40 markers are selected as per the NCI guidelines. A revised guidelines suggests mononucleotide marker panel is more sensitive for MSI-H tumors than other microsatellite markers. Dinucleotide markers are less sensitive, and if only dinucleotide markers are positive, it is mandatory to test additional mononucleotide markers to rule out MSI-L [PMID: 14970275].This kit contains 13 mononucleotide markers for higher resolution and two STR sequences that can be used to

track sample identity [PMID: 35884597][PMID: 35982978].

ABI-16	ABI-19	ABI-20B	BAT-26	CAT-25	NR-22	NR-27
ABI-17	ABI-20A	BAT-25	BAT-40	NR-21	NR-24	

The primers are fluorophore tagged at the 5' end and the end-point PCR product is analyzed by Fluorophore Capillary Electrophoresis. The tumor tissue is classified as MSS/MSI-L/MSI-H as mentioned in the table below.

MSI Result	**Interpretation**[PMID: 35884597]
MSI-High	Unstable marker rate:- 30% - 100%
MSI-Low	Unstable marker rate:- 5% - 29.99%
MSS(Microsatellite Stable)	Unstable marker rate:- 0%

RECOMMENDATION

Test results should be interpreted in context of clinical findings, family history, and other laboratory data. If results obtained do not match other clinical or laboratory findings, please contact the laboratory for possible interpretation. Misinterpretation of results may occur if the information provided is inaccurate or incomplete.

REFERENCE

1. Application note: TrueMark MSI Assay—a simplified solution for analyzing microsatellite instability in FFPE tumor samples, 2020.

Dr Syed Muqlisur Rehman, MD
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END OF REPORT