Benedict De Zilwa

AGE: 72 years | **Gender:** Male | **Coupon/UID:** NA **/** AOMG0231

Diagnosis: Carcinoma, Non-Small-Cell Lung

MEDGENOME

MGM1496 : Lung Cancer gene Panel by NGS (SNVs, InDels & Fusions)+ PD-L1 (SP263) by IHC + Microsatellite Instability (MSI) test

Report Details		Specimen Information		Ordering Clinician	
Sample ID / Order ID:	9186003 / 1325595	**Specimen Site:**	Lymph node	**Clinician:**	Dr. Mahendra Perera
Collection Date:	NA	**Specimen Received:**	FFPE Tissue Blocks [3]	**Affiliation:**	Aegle Omics Private Limited
Date Received:	7th June 2025	**Specimen Tested:**	SK 9791 (A)	**Serviced By:**	18718
Report Date & Time:	26th Jun 2025 17:12 PM	**Tumor Content (%):**	65	**Report Status:**	Final

Clinical Summary: Lymph node biopsy - Metastatic pulmonary Adenocarcinoma

TEST RESULT SUMMARY

Microsatellite Instability (MSI) Test	PD-L1 IHC (SP263)
Status - MSS	TPS - 0 %

Kindly refer to the complete MSI and PD-L1 IHC reports below.

Next Generation Sequencing (NGS) Results

POSITIVE

Gene	Findings	Gene	Findings
ALK	Not Detected	*BRAF*	Not Detected
EGFR	Not Detected	*ERBB2*	Not Detected
KRAS	**Q61H**	*MET*	Not Detected
NTRK1	Not Detected	*NTRK2*	Not Detected
NTRK3	Not Detected	*RET*	Not Detected
ROS1	Not Detected		

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
***KRAS* p.Gln61His (MISSENSE)** Variant Allele Frequency - 79.47%				
Tier II	c.183A>C (ENST00000311936.8)	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.

Please refer to the appendix section for the complete list of genes covered in this assay.

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

KRAS (p.Gln61His) - MISSENSE

Gene: *KRAS*	**Exon:** 3	**Variant Allele Frequency:** 79.47%
Nucleotide change: chr12:g.25227341T>G	**Protein change:** p.Gln61His	**Population MAF:** 0 (1000G);0(gnomAD);
cDNA change: c.183A>C	**Variant Type:** MISSENSE	**In-silico Predictions:** D_lc(SIFT); D(LRT); NA(Polyphen2)
Transcript ID: ENST00000311936.8	**Variant Allele Depth/Total depth:** 2326/2927x	**Gene Function:** Oncogene

Gene Summary: *KRAS*, a Kirsten ras oncogene homolog from the mammalian ras gene family, encodes a protein that is a member of the small GTPase superfamily. A single amino acid substitution is responsible for an activating mutation. The transforming protein that results is implicated in various malignancies, including lung adenocarcinoma, mucinous adenoma, ductal carcinoma of the pancreas and colorectal carcinoma. Alternative splicing leads to variants encoding two isoforms that differ in the C-terminal region.

Clinical and Therapeutic Relevance: The small GTPase *KRAS* activates the RAS/MAPK signaling pathway to promote cell proliferation and survival. Variants at codon 61 of *KRAS* have been shown to promote transformation due to enhanced downstream signaling. *KRAS* mutations at codon 61 are seen in a subset of lung cancer (<3%). In a phase I study in non-squamous, non-small cell lung cancer, a tumor with this variant responded to binimetinib combined with chemotherapy. Single clinical cases of *ALK*-rearranged lung cancer and this variant showed resistance to ALK inhibitors crizotinib or brigatinib. Preclinical models were sensitive to the combination treatment of MDM2 inhibitor navtemadlin (AMG-232) and MEK inhibitor trametinib. A lung cancer cell line with *PIK3CA*.E545K mutation and this variant was sensitive to MEK inhibitors binimetinib or trametinib.

PubMed References: 34537440, 34052705, 33415011, 27422710, 27338794, 26898615, 22169769, 35797463, 33579957, 31088841, 30194935

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 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 on FDA 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 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 (WHO, 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 lung cancer panel is a high throughput next-generation sequencing based single assay that may provide treatment benefit to the patients. This next-generation sequencing based multi-gene lung cancer test is developed to sequence and identify genomic alterations associated with genes having therapeutic, prognostic and diagnostic implications. This panel covers key lung cancer genes for the assessment of various classes of genomic alterations (SNVs, InDels, CNVs and Fusions). *MET* gene is primarily tested for variations that lead to exon 14 skipping.

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					
BRAF	*EGFR*	*ERBB2*	*KRAS*	*MET*	*RET*

Note: *MET* exon 14 skipping mutations included.

CNVs		
EGFR	*ERBB2*	*MET*

FUSIONS					
ALK	*NTRK1*	*NTRK2*	*NTRK3*	*RET*	*ROS1*

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

Benedict De Zilwa

AGE: 72 years | **Gender:** Male | **Coupon/UID:** NA / AOMG0231

Diagnosis: Carcinoma, Non-Small-Cell Lung

MEDGENOME

MGM527: Microsatellite Instability (MSI) by fragment analysis

Report Details		Specimen Information		Ordering Clinician	
Sample ID / Order ID:	9186003 / 1325595	**Specimen Site:**	Lymph node	**Clinician:**	Dr. Mahendra Perera
Collection Date:	NA	**Specimen Received:**	FFPE Tissue Blocks [3]	**Affiliation:**	Aegle Omics Private Limited
Date Received:	7th June 2025	**Specimen Tested:**	SK 9791 (A)	**Serviced By:**	18718
Report Date & Time:	14th Jun 2025 15:12 PM	**Tumor Content (%):**	65	**Report Status:**	Final

Clinical Summary: Lymph node biopsy - Metastatic pulmonary Adenocarcinoma

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 23-06-2025 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	8
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-16, ABI-20A, ABI-20B, BAT-26* and *BAT-40*" had failed in amplification. Hence, the MSI status of this subject has been interpreted based on the status of 8 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
Molecular Pathologist
KMC Registration No. 71468

END OF REPORT

Medgenome Labs Ltd
Sy No 94/1C and 94/2, Tower-1, Veerasandra Village,
Attibele Hobli, Electronic City, Phase-1, Bangalore,
Karnataka, 560100.

PD-L1 (SP263) - IHC Test Report

Patient Name	Benedict De Zilwa	Order ID	1325595
Age / Gender	72 Years / Male	Sample ID	9186003
Physician	Dr. Mahendra Perera	Collection Date	NA
Customer	MCC18718-Aegle Omics Private Limited	Sample Received Date	07-06-2025 11:56 AM
Report Date	13-06-2025 05:26 PM	Report Status	Final

Lab/Biopsy No : MBI-1745-25

Clinical Details : Lymph node biopsy - Metastatic pulmonary Adenocarcinoma.

Specimen received : Three blocks

Gross Examination : Three blocks labelled as SK-9791-A, B & C. Test done on SK-9791-A block

Test interpretation/Result:

IHC Markers	Tumor cell proportion score (TPS)	Result
PD-L1 (SP263) IHC	0%	Negative. No membranous staining evident in tumor cells.

Note:

A sample from this individual was referred to our laboratory for "Combo Test" (Two different tests were performed & two different reports shall be sent). Results of these two reports have to be interpreted while making a clinical decision.
Report 1 of 2 (Report 2 of 2 is due for release).

Comments:

1. PD-L1 testing done by ventana PD-L1 (SP263) assay using rabbit anti-human PD-L1/CD274 monoclonal antibody (clone SP 263) on Ventana benchmark autostainer with optiview DAB IHC detection kit.

2. PD-L1 staining / expression is defined as complete or partial circumferential linear plasma membrane staining at any intensity that can be differentiated from background and diffuse cytoplasmic staining. Only cytoplasmic staining is not considered significant.

3. Roche's Ventana PD-L1 (SP263) assay is CE (European Conformity) labelled to inform treatment decisions in lung cancer patients being considered for keytruda (pembrolizumab) immunotherapy as a first line of treatment for high PD-L1 expressors.

4. Recommended positive cut off for PD-L1 (clone SP 263) in lung cancer(NSCLC) : > or = 50% of tumor cells. Studies showed superior progression free survival and overall survival in first-line treatment of mNSCLC with PD-L1 expression > or = 50% of tumor cells. There is also high degree of concordance between SP 263 (CE marked) and 22c3 assays (FDA approved) if a 50 % cut off point is applied in both cases.

5. Recommended positive cut off of PD-L1 (clone SP 263) for metastatic urothelial carcinoma is > or = 25% of tumor cells.

6. Clinical utility of this PD-L1 clone SP 263 assay needs to be verified in clinical studies for tumors other than NSCLC and urothelial carcinoma.

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PD-L1 (SP263) - IHC Test Report

Patient Name	Benedict De Zilwa	Order ID	1325595
Age / Gender	72 Years / Male	Sample ID	9186003
Physician	Dr. Mahendra Perera	Collection Date	NA
Customer	MCC18718-Aegle Omics Private Limited	Sample Received Date	07-06-2025 11:56 AM
Report Date	13-06-2025 05:26 PM	Report Status	Final

Note:

System level Controls (internal & or external) run with the test are satisfactory. Reagents used are the complimentary diagnostic assay consisting of primary antibody PD-L1 clone SP 263 and Optiview DAB detection on a Ventana Benchmark autostainer. This assay has not been validated on decalcified tissue and result should be interpreted with caution given the likelihood of false negativity of decalcified specimen. Specimen should be processed by routine tissue processing method. Inappropriate fixation (nonformalin) and processing may give erroneous result.

The performance characteristics of this assay has been determined by MedGenome. Performance characteristics refer to the analytical performance of the test.

Please correlate the block# given with that of its HPE report.

References:

1.Kerr K. M., Nicolson. M.C.; Non-small cell lung cancer, PDL-1 and the Pathologist. Arch Pathol Lab Med. 2016;140:249-254.

2.Fred Hirsch, McElhinny A, Dave Stanforth D. PD-L1 Immunohistochemistry Assays for Lung Cancer: Results from Phase 1 of the Blueprint PD-L1 IHC Assay Comparison Project. Journal of Thoracic Oncology. 2017;12:208-22.

3.Scholl L.M. et al. 2016. Programmed Death Ligand-1 Immunohistochemistry—A New Challenge for Pathologists. A Perspective From Members of the Pulmonary Pathology Society Arch Pathol Lab Med. 140:341-344.

4.Ratcliffe et al. Agreement between Programmed Cell Death Ligand-1 Diagnostic Assays across Multiple Protein Expression Cutoffs in Non–Small Cell Lung Cancer. Clin Cancer Res July 15 2017 (23)(14)3585-3591; DOI: 10.1158/1078-0432.

Enclosed : Three blocks

Verified By

Dr. Rumana Tasneem
Junior Pathologist, MBBS, MD
KMC Reg No. 96079

Approved By

Dr. Syed Muqlisur Rehman, MD (Path)
Molecular Pathologist
KMC Reg No. - 71468

*****End of Report*****

Medgenome Labs Ltd
Sy No 94/1C and 94/2, Tower-1, Veerasandra Village,
Attibele Hobli, Electronic City, Phase-1, Bangalore,
Karnataka, 560100.

CONDITIONS OF LABORATORY TESTING AND REPORTING

Medgenome Labs Ltd, Bangalore, Karnataka, India

- Laboratory results should be used with other clinical information to determine a final diagnosis.
- In case of unexpected test results please contact the laboratory. We will investigate and repeat analysis if possible.
- The medical report must be viewed and reproduced as a whole
- This medical report is not intended for medico-legal purposes.
- The medical report is to be interpreted and used by medical personnel only
- Assays are performed and reported in accordance with the stated schedule.
- There may be circumstances beyond our control that delay results, e.g., invalid assay run.
- The results of a laboratory test are dependent on the quality of the sample as well as the assay procedure.
- A requested test may not be carried out if:
- Sample is insufficient or inappropriate
- Sample quality is unsatisfactory
- Request for testing is withdrawn by the ordering doctor or patient
- There is discord between the labelling of the sample container and the name on the test requisition.
- For any query contact customer support : +91(0)8067154932/33
