B.H.K. M Fernando

AGE: 49 years | **Gender:** Female | **Coupon/UID:** NA / AOM0281

Diagnosis: Uterine Neoplasms

MEDGENOME

MGM3309: OncoTrack CGP (Liquid Biopsy)

Report Details		Specimen Information		Ordering Clinician	
Sample ID / Order ID:	9524326 / 1509783	**Specimen Site:**	NA	**Clinician:**	Dr. Mahendra Perera
Collection Date:	3rd November 2025	**Specimen Received:**	Blood in streck tube	**Affiliation:**	Aegle Omics Private Limited
Date Received:	6th November 2025	**Specimen Tested:**	Blood in streck tube	**Serviced By:**	18718
Report Date & Time:	22nd Nov 2025 16:38 PM	**Tumor Content (%):**	NA	**Report Status:**	Final

Clinical Summary: Moderately differentiated adenocarcinoma of the endocervix, T1b Nx, FIGO: 1B

Test Result Summary

Potential treatment impacts

1 Effective	0 Ineffective	0 Safety

Prognostic and Diagnostic findings

1 Prognostic	1 Diagnostic

Clinical trials

0 Trials

TMB STATUS

TMB Score: 6.00 mut/Mb

NEGATIVE | POSITIVE

MSI STATUS

STABLE | UNSTABLE

AMP Classification ^	Evidence Level ^	Treatment	Treatment Benefit	Drug Approval ^$	Clinical Trials
Genomic variants					
***PIK3R3* p.Arg306Ter (NONSENSE)** Variant Allele Frequency - 0.9%					
Tier IID	6 Clinical	-	Diagnostic	-	0
***FAT1* p.Pro2185LeufsTer28 (FRAMESHIFT-DEL)** Variant Allele Frequency - 0.6%					
Tier IID	5 Clinical	-	Prognostic	-	0
CNV variants					
***ERBB2* Copy Number Gain (CNV)**					
Tier IIC	6 Clinical	Trastuzumab Deruxtecan	Effective	Off-label	0

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.

Note: Decisions regarding treatment action plan should not be solely based on this test results. These findings are highly recommended to be correlated with the patient's clinical, pathological, and family history for decisions on diagnosis, prognosis or therapeutics.

ADDITIONAL BIOMARKERS DETECTED (Variant of Uncertain Significance)

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.

Gene	Exon	Nucleotide change	Protein change	Alternate allele Depth (x)	Allele Burden (%)	Functional predictions	Population MAF (%)
SNV table							
ZBTB7A	2	ENST00000322357.4 c.493C>T chr19:4054738:G>A	p.Gln165Ter	13x	0.5%	NA(SIFT); NA(LRT); NA(Polyphen2)	0 (1000G); 0 (gnomAD);

GLOSSARY

AMP Classification Criteria: Displays the classification of a biomarker according to the recommendations of the Association for Molecular Pathology (AMP) [PMID: 27993330].

Tier	Criteria
Tier IA	Variants of strong clinical significance. FDA-approved therapy or biomarkers included in professional guidelines.
Tier IB	Variants of strong clinical significance. Well-powered studies with consensus from experts in the field.
Tier IIC	Variants of potential clinical significance. FDA-approved therapies for different cancer types or investigational therapies. Multiple small published studies with some consensus.
Tier IID	Variants of potential clinical significance. Preclinical trials or a few case reports without consensus.
Tier III	Variants of unknown clinical significance.
Tier IV	Benign or likely benign variants.

Evidence Level: The Evidence Level, values 7-1 indicate the reliability of a biomarker to predict a specific patient outcome. This can include predictive treatment effects; in this case, the scores 7-1 apply for biomarkers associated with a single drug or drug combination. The Evidence levels are defined as follows:

Score	Definition
7, Clinically approved	Variant approved by a regulatory agency to predict a specific effect (i.e., response, resistance, or toxicity) in the patient's disease or cancer type.
6, Clinical	• Variant has not yet been approved by a regulatory agency for the patient's disease but has been observed in at least one large cohort study to predict a specific effect of the drug (i.e., to be effective, resistance) in the patient's disease. • The biomarker has been approved by a regulatory agency to predict a specific effect of the drug (response, resistance) with other diseases.

Score	Definition
5, Clinical	• Variant has not yet been approved by a regulatory agency for the patient's disease but has been observed to predict a specific effect of the drug (i.e., response, resistance) on patients with other diseases or conditions. • For variants predicting a drug to be effective or resistant, there is evidence from some patients in several cohort studies and preclinical evidence.
4, Clinical	• The variant has not yet been approved by a regulatory agency for the patient's disease. However, this variant has been observed to predict a specific effect of the drug (i.e., response, resistance) on patients with other diseases or conditions. • For variants predicting a drug to be effective or resistant, there is evidence from a few clinical case reports and additional preclinical evidence. • For variants predicting a drug to be toxic, there is evidence from a prospective study, >1 retrospective studies, or >1 cohort studies.
3, Preclinical	Variant has not yet been observed/tested in patients to predict a specific effect but has been observed in preclinical experiments. There is experimental evidence from cell lines or mouse models, for example.
2, Preclinical	Variant has not yet been observed/tested in patients or preclinical models to predict a specific effect. However, this effect can be inferred when drug-sensitivity data are available for another variant and if the two variants have the identical functional impact on the same downstream pathway
1, Preclinical	Variant has not yet been observed/tested in patients or preclinical models to predict a specific effect. However, this effect can be inferred when drug-sensitivity data are available for another variant and if both variants have the identical functional impact on the protein.

Drug approval:
The development stage of the treatment for the patient's indication as per US-FDA guidelines.

Stage	Definition
Approved	This drug is launched for the primary or a secondary patient disease
Off-Label	This drug is launched for a disease other than the primary or secondary patient diseases
Investigational	This drug is currently under clinical development in the patient disease.
Other	None of the other stages are applicable. The drug or drug class is, for example, suspended, discontinued, or withdrawn.

Medications with potential for adverse reaction or ineffectiveness: Refers to the identified treatments that are predicted to be associated with negative physiological responses to a drug therapy (i.e., drug resistance and toxicity) based on the biomedical evidence.

Potential impact: The specific drug effect predicted by the identified mutation (i.e. response, resistance, or toxicity).

Treatment: The generic name of the therapeutic agent listed on the report.

ACTIONABLE BIOMARKER DETAILS

PIK3R3(p.Arg306Ter)

Gene: *PIK3R3*	**Exon:** 7	**Variant Allele Frequency:** 0.9%
Nucleotide change: chr1:g.46521492G>A	**Protein change:** p.Arg306Ter	**Population MAF:** 0 (1000G); 0.000987 (gnomAD);
cDNA change: c.916C>T	**Variant Type:** NONSENSE	**In-silico Predictions:** NA(SIFT); NA(LRT); NA(Polyphen2)
Transcript ID : ENST00000262741.5	**Variant Allele Depth:** 19x	**Gene Function:**

Gene Summary: Phosphatidylinositol 3-kinase (PI3K) phosphorylates phosphatidylinositol and similar compounds, which then serve as second messengers in growth signaling pathways. PI3K is composed of a catalytic and a regulatory subunit. The protein encoded by *PIK3R3* represents a regulatory subunit of PI3K. The encoded protein contains two SH2 domains through which it binds activated protein tyrosine kinases to regulate their activity.

Clinical and Therapeutic Relevance: *PIK3R3* encodes a kinase involved in insulin-like growth factor 1 receptor signaling. Inactivating mutations of*PIK3R3*are found in stomach and uterine cancers. Truncating mutations in*PIK3R3* result in *PIK3R3*C-terminal truncated proteins. In a single study with lymphatic endothelial cells harboring *PIK3R3*mutations, mutant cells were demonstrated to exhibit increased phosphorylated AKT and were associated with increased sensitivity to PI3K inhibitors wortmannin and LY294 as well as mTOR inhibitor rapamycin when compared to normal endothelial cells. In a study, researchers analyzed a large cohort of 243 endometrial tumor samples, which included endometrioid grade 1–2 tumors (n=132), grade 3 endometrioid tumors (n=29), mixed endometrial tumors (n=60), and malignant mixed Müllerian tumors (MMMT, n=18). While the primary focus was on frequent mutations in PIK3R1 and PIK3R2, the study also identified truncating mutations in *PIK3R3*, a regulatory subunit of PI3K. These truncating mutations were notable because they disrupted the normal stabilizing interactions within the PI3K pathway, contributing to dysregulation of PTEN protein stability. The findings suggest that *PIK3R3* truncating mutations, though less common than PIK3R1/PIK3R2 alterations, play a role in weakening PTEN's tumor-suppressive function, thereby enhancing PI3K pathway activation and promoting oncogenesis in endometrial cancer.

PubMed References: 25424831, 27766312, 21984976

FAT1(p.Pro2185LeufsTer28)

Gene: *FAT1*	**Exon:** 10	**Variant Allele Frequency:** 0.6%
Nucleotide change: chr4:g.187541187del	**Protein change:** p.Pro2185LeufsTer28	**Population MAF:** 0 (1000G); 0 (gnomAD);
cDNA change: c.6554del	**Variant Type:** FRAMESHIFT-DEL	**In-silico Predictions:** NA(SIFT); NA(LRT); NA(Polyphen2)
Transcript ID : ENST00000441802.2	**Variant Allele Depth:** 18x	**Gene Function:** Tumor Suppressor Gene

Gene Summary: *FAT1* is an ortholog of the Drosophila fat gene, which encodes a tumor suppressor essential for controlling cell proliferation during Drosophila development. The gene product is a member of the cadherin superfamily, a group of integral membrane proteins characterized by the presence of cadherin-type repeats. In addition to containing 34 tandem cadherin-type repeats, the gene product has five epidermal growth factor (EGF)-like repeats and one laminin A-G domain. *FAT1* is expressed at high levels in a number of fetal epithelia. Its product probably functions as an adhesion molecule and/or signaling receptor, and is likely to be important in developmental processes and cell communication. Transcript variants derived from alternative splicing and/or alternative promoter usage exist, but they have not been fully described.

FAT1(p.Pro2185LeufsTer28)

Clinical and Therapeutic Relevance: *FAT1*, a tumor suppressor and transmembrane protein, is inactivated by mutation or deletion in various cancer types. Homo- and heterozygous deletion of the *FAT1*locus on chromosome 4q35 leads to the loss of *FAT1* expression and has been described in several tumors. In a study of Type I endometrial cancer, *FAT1*alterations were evaluated in relation to patient outcomes. Specifically, 72 cases of Type I endometrial carcinoma were analyzed using immunohistochemical staining to assess FAT1 protein expression alongside β-catenin. The results showed that reduced or absent FAT1 expression was significantly associated with poorer survival outcomes, suggesting that *FAT1* loss functions as a prognostic marker in uterine cancer and may contribute to tumor progression through dysregulation of cell adhesion and signaling pathways [http://www.jmatonline.com/PDF/757-763-PB-12037.pdf].

PubMed References:
21617878

ERBB2 (CNV)

Gene: *ERBB2* **CNV type:** Copy Number Gain

Gene Summary: *ERBB2* encodes a member of the epidermal growth factor (EGF) receptor family of receptor tyrosine kinases. This protein has no ligand binding domain of its own and therefore cannot bind growth factors. However, it does bind tightly to other ligand-bound EGF receptor family members to form a heterodimer, stabilizing ligand binding and enhancing kinase-mediated activation of downstream signalling pathways, such as those involving mitogen-activated protein kinase and phosphatidylinositol-3 kinase. Allelic variations at amino acid positions 654 and 655 of isoform a (positions 624 and 625 of isoform b) have been reported, with the most common allele, Ile654/Ile655, shown here. Amplification and/or overexpression of *ERBB2* has been reported in numerous cancers, including breast and ovarian tumors. Alternative splicing results in several additional transcript variants, some encoding different isoforms and others that have not been fully characterized.

Clinical and Therapeutic Relevance: The receptor tyrosine-protein kinase *ERBB2* (HER2) activates the RAS/MAPK, PI3K/AKT, and JAK/STAT signaling pathways to promote cell proliferation and survival. In preclinical studies, HER2 amplification was shown to activate the PI3K and RAS/MAPK pathways. **Trastuzumab deruxtecan is indicated for adult patients with unresectable or metastatic HER2-overexpressing (IHC 3+) solid tumors who have received prior systemic treatment and have no satisfactory alternative treatment options.** The DESTINY-PanTumor02 phase 2 trial for patients with HER2-overexpressing (IHC 3+) solid tumors (n=111) revealed an ORR of 51.4% with responses in all tumor types. In a phase 1 basket trial, one endometrial cancer patient achieved a partial response (PR) to trastuzumab deruxtecan. A randomized phase 2 trial compared carboplatin-paclitaxel with and without trastuzumab in *ERBB2*-positive patients (n=61) with advanced or recurrent uterine serous carcinoma. Progression-free survival (PFS) favors the trastuzumab arm, with a median of 12.9 months vs. 8.0 months in the control arm (p=.005). Also, overall survival (OS) was higher in the trastuzumab arm compared to the control arm, with medians of 29.6 versus 24.4 months (p=.046). One endometrial cancer patient who was treated with afatinib achieved a complete response (CR).

PubMed References: 40157574, 38457761, 37318379, 37286557, 37276871, 37246414, 36971495, 34916216, 34661865, 34156520, 33480963, 33408520, 33194604, 33167735, 33030616, 33005299, 32988996, 32469182, 32213539, 32022229, 31956604, 31825569, 31825192, 31087550, 31047804, 31011206, 30952821, 30854046, 30562681, 30552112, 30516102, 30452336, 30307354, 30206164, 29989854, 29584549, 29465762, 29409051, 29325228, 29313813, 29298799, 28868025, 28526536, 28498781, 28423702, 28343975, 28040715, 27405796, 27044931, 26874901, 26545934, 25694417, 24947902, 24868024, 23348520, 22586653, 21900593, 21115860, 20736298, 20728210, 20179222, 16782917

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 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 cfDNA 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.
- This test has been validated at MedGenome Labs and the limit of detection (LOD) of allele fraction for SNVs and short InDels is 0.5% VAF and for fusions is >3 read support. However, the report may include, at the discretion of laboratory director, the variants with lower allele burden having strong or potential clinical significance or those have been reported earlier in the patient.
- The gene rearrangements detected by DNA based sequencing may or may not result in a transcribed fusion with functional significance. Hence confirmation by other assays (RNA sequencing, IHC) on tumor tissue is recommended [PMID: 31375766, 32726941].
- TMB Score is dependent on the number of the genes in the panel and given as quantitative measurement. TMB calculations may differ for different samples from the same patient and for different detection methods for the same sample, as they depend on variables such as panel size, tumor fraction, limit of detection, filtering of mutations, and bioinformatics analysis specifications.
- MSI JS distance calculations may differ depending on variables such as number of MSI sites, tumor fraction, and type of tumor.
- **The CNV prediction (Amplification and Deletion) is based on fold change as determined for the individual genes based on gene specific cutoffs provided by Illumina.**
- Variations occurring in non-coding and deep intronic regions are not detected in this assay.
- All analysis and reporting have been performed as per the assay characteristics.

- The gene rearrangements detected by DNA based sequencing may or may not result in a transcribed fusion with functional significance. Hence confirmation by other assays (RNA sequencing, IHC) on tumor tissue is recommended [PMID:31375766 , 32726941].
- **Additional case specific disclaimer: None**

TEST DESCRIPTION

The MedGenome's Comprehensive Genomic Profiling – Liquid Biopsy test is multi-biomarker single test that determines SNVs and InDels in 523 genes, CNVs in 59 genes, Fusions in 23 genes along with TMB and MSI. The test is performed on circulating free DNA (cfDNA) isolated from blood plasma (liquid biopsy). cfDNA comprises circulating tumor DNA (ctDNA) present in blood plasma that is shed from tumor tissue and is the source of tumor genetic material. Unlike traditional biopsy, liquid biopsy is noninvasive as it requires only a peripheral blood draw in Streck tube from the cancer patient. Liquid Biopsy NGS testing is very powerful clinically as it provides- (a) real-time treatment monitoring to evaluate the drug response in cancer patients, (b) early detection of acquired resistance mutations to targeted therapy, (c) detection of recurrence at early stages before significant accumulation of tumor cell mass, (d) identification of tumor heterogeneity arising due to multiple clones and hence the disease progression.

TEST METHODOLOGY

Sample Type: Peripheral blood in Streck tube.
Extraction and Library Preparation: cfDNA isolated from blood plasma is used to perform UMI-based target enrichment and sequencing using a custom capture kit.
Sequencing: The QC passed libraries are sequenced to a minimum depth >20000X on validated Illumina sequencing platform (Pre-UMI Correction).
Data Analysis: The sequences obtained are aligned to human reference genome (GRCh37/hg19) using BWA program [PMID: 19451168,PMID: 23155063]. Somatic mutations are identified using UMI corrected Sention pipeline [PMID: 31481971]. Only non-synonymous and splice site variants found in the coding regions are used for clinical interpretation. The mutations are annotated using our in-house annotation pipeline (VariMAT).
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.
TMB Analysis: Non-synonymous and synonymous variants found in the coding regions are used for clinical tumor mutational burden interpretation. TMB metrics is generated from the annotated VCF file post small variant filtering analysis step. The TMB status will be considered as positive (>=13) or negative (<13) based on the TMB score provided as mutations/Mb as per the analysis pipeline.
MSI Analysis: The Microsatellite Instability Status (MSI) is based on Jensen–Shannon (JS) distance which is significantly higher in the test sample vs baseline normal with p-value < = 0.01 and the JS distance difference is > = 0.08, then considered as unstable. The final MSI score aggregates all JS distance across all unstable sites. The MSI status will be interpreted as stable or unstable based on the JS distance cutoff as per the analysis pipeline.
CNV Analysis: The copy number variant caller performs amplification and deletion calling for CNV genes within the assay. The software calculates fold change values for each gene, and determines the CNV status for each CNV target gene. The CNV duplication or deletion is interpretedbased on the gene specific cutoffs.
§The performance characteristics of this test is determined by MedGenome Labs Ltd.

GENES ANALYSED

SNVs, InDels and TMB									
ABL1	*ABL2*	*ACVR1*	*ACVR1B*	*AKT1*	*AKT2*	*AKT3*	*ALK*	*ALOX12B*	*ANKRD11*
ANKRD26	*APC*	*AR*	*ARAF*	*ARFRP1*	*ARID1A*	*ARID1B*	*ARID2*	*ARID5B*	*ASXL1*
ASXL2	*ATM*	*ATR*	*ATRX*	*AURKA*	*AURKB*	*AXIN1*	*AXIN2*	*AXL*	*B2M*
BAP1	*BARD1*	*BBC3*	*BCL10*	*BCL2*	*BCL2L1*	*BCL2L11*	*BCL2L2*	*BCL6*	*BCOR*
BCORL1	*BCR*	*BIRC3*	*BLM*	*BMPR1A*	*BRAF*	*BRCA1*	*BRCA2*	*BRD4*	*BRIP1*
BTG1	*BTK*	*C11orf30*	*CALR*	*CARD11*	*CASP8*	*CBFB*	*CBL*	*CCND1*	*CCND2*
CCND3	*CCNE1*	*CD274*	*CD276*	*CD74*	*CD79A*	*CD79B*	*CDC73*	*CDH1*	*CDK12*

CDK4	*CDK6*	*CDK8*	*CDKN1A*	*CDKN1B*	*CDKN2A*	*CDKN2B*	*CDKN2C*	*CEBPA*	*CENPA*
CHD2	*CHD4*	*CHEK1*	*CHEK2*	*CIC*	*CREBBP*	*CRKL*	*CRLF2*	*CSF1R*	*CSF3R*
CSNK1A1	*CTCF*	*CTLA4*	*CTNNA1*	*CTNNB1*	*CUL3*	*CUX1*	*CXCR4*	*CYLD*	*DAXX*
DCUN1D1	*DDR2*	*DDX41*	*DHX15*	*DICER1*	*DIS3*	*DNAJB1*	*DNMT1*	*DNMT3A*	*DNMT3B*
DOT1L	*E2F3*	*EED*	*EGFL7*	*EGFR*	*EIF1AX*	*EIF4A2*	*EIF4E*	*EML4*	*EP300*
EPCAM	*EPHA3*	*EPHA5*	*EPHA7*	*EPHB1*	*ERBB2*	*ERBB3*	*ERBB4*	*ERCC1*	*ERCC2*
ERCC3	*ERCC4*	*ERCC5*	*ERG*	*ERRFI1*	*ESR1*	*ETS1*	*ETV1*	*ETV4*	*ETV5*
ETV6	*EWSR1*	*EZH2*	*FAM123B*	*FAM175A*	*FAM46C*	*FANCA*	*FANCC*	*FANCD2*	*FANCE*
FANCF	*FANCG*	*FANCI*	*FANCL*	*FAS*	*FAT1*	*FBXW7*	*FGF1*	*FGF10*	*FGF14*
FGF19	*FGF2*	*FGF23*	*FGF3*	*FGF4*	*FGF5*	*FGF6*	*FGF7*	*FGF8*	*FGF9*
FGFR1	*FGFR2*	*FGFR3*	*FGFR4*	*FH*	*FLCN*	*FLI1*	*FLT1*	*FLT3*	*FLT4*
FOXA1	*FOXL2*	*FOXO1*	*FOXP1*	*FRS2*	*FUBP1*	*FYN*	*GABRA6*	*GATA1*	*GATA2*
GATA3	*GATA4*	*GATA6*	*GEN1*	*GID4*	*GLI1*	*GNA11*	*GNA13*	*GNAQ*	*GNAS*
GPR124	*GPS2*	*GREM1*	*GRIN2A*	*GRM3*	*GSK3B*	*H3F3A*	*H3F3B*	*H3F3C*	*HGF*
HIST1H1C	*HIST1H2BD*	*HIST1H3A*	*HIST1H3B*	*HIST1H3C*	*HIST1H3D*	*HIST1H3E*	*HIST1H3F*	*HIST1H3G*	*HIST1H3H*
HIST1H3I	*HIST1H3J*	*HIST2H3A*	*HIST2H3C*	*HIST2H3D*	*HIST3H3*	*HLAA*	*HLAB*	*HLAC*	*HNF1A*
HNRNPK	*HOXB13*	*HRAS*	*HSD3B1*	*HSP90AA1*	*ICOSLG*	*ID3*	*IDH1*	*IDH2*	*IFNGR1*
IGF1	*IGF1R*	*IGF2*	*IKBKE*	*IKZF1*	*IL10*	*IL7R*	*INHA*	*INHBA*	*INPP4A*
INPP4B	*INSR*	*IRF2*	*IRF4*	*IRS1*	*IRS2*	*JAK1*	*JAK2*	*JAK3*	*JUN*
KAT6A	*KDM5A*	*KDM5C*	*KDM6A*	*KDR*	*KEAP1*	*KEL*	*KIF5B*	*KIT*	*KLF4*
KLHL6	*KMT2B*	*KMT2C*	*KMT2D*	*KRAS*	*LAMP1*	*LATS1*	*LATS2*	*LMO1*	*LRP1B*
LYN	*LZTR1*	*MAGI2*	*MALT1*	*MAP2K1*	*MAP2K2*	*MAP2K4*	*MAP3K1*	*MAP3K13*	*MAP3K14*
MAP3K4	*MAPK1*	*MAPK3*	*MAX*	*MCL1*	*MDC1*	*MDM2*	*MDM4*	*MED12*	*MEF2B*
MEN1	*MET*	*MGA*	*MITF*	*MLH1*	*MLL*	*MLLT3*	*MPL*	*MRE11A*	*MSH2*
MSH3	*MSH6*	*MST1*	*MST1R*	*MTOR*	*MUTYH*	*MYB*	*MYC*	*MYCL*	*MYCN*
MYD88	*MYOD1*	*NAB2*	*NBN*	*NCOA3*	*NCOR1*	*NEGR1*	*NF1*	*NF2*	*NFE2L2*
NFKBIA	*NKX21*	*NKX31*	*NOTCH1*	*NOTCH2*	*NOTCH3*	*NOTCH4*	*NPM1*	*NRAS*	*NRG1*
NSD1	*NTRK1*	*NTRK2*	*NTRK3*	*NUP93*	*NUTM1*	*PAK1*	*PAK3*	*PAK7*	*PALB2*
PARK2	*PARP1*	*PAX3*	*PAX5*	*PAX7*	*PAX8*	*PBRM1*	*PDCD1*	*PDCD1LG2*	*PDGFRA*
PDGFRB	*PDK1*	*PDPK1*	*PGR*	*PHF6*	*PHOX2B*	*PIK3C2B*	*PIK3C2G*	*PIK3C3*	*PIK3CA*
PIK3CB	*PIK3CD*	*PIK3CG*	*PIK3R1*	*PIK3R2*	*PIK3R3*	*PIM1*	*PLCG2*	*PLK2*	*PMAIP1*
PMS1	*PMS2*	*PNRC1*	*POLD1*	*POLE*	*PPARG*	*PPM1D*	*PPP2R1A*	*PPP2R2A*	*PPP6C*
PRDM1	*PREX2*	*PRKAR1A*	*PRKCI*	*PRKDC*	*PRSS8*	*PTCH1*	*PTEN*	*PTPN11*	*PTPRD*
PTPRS	*PTPRT*	*QKI*	*RAB35*	*RAC1*	*RAD21*	*RAD50*	*RAD51*	*RAD51B*	*RAD51C*
RAD51D	*RAD52*	*RAD54L*	*RAF1*	*RANBP2*	*RARA*	*RASA1*	*RB1*	*RBM10*	*RECQL4*
REL	*RET*	*RFWD2*	*RHEB*	*RHOA*	*RICTOR*	*RIT1*	*RNF43*	*ROS1*	*RPS6KA4*
RPS6KB1	*RPS6KB2*	*RPTOR*	*RUNX1*	*RUNX1T1*	*RYBP*	*SDHA*	*SDHAF2*	*SDHB*	*SDHC*
SDHD	*SETBP1*	*SETD2*	*SF3B1*	*SH2B3*	*SH2D1A*	*SHQ1*	*SLIT2*	*SLX4*	*SMAD2*
SMAD3	*SMAD4*	*SMARCA4*	*SMARCB1*	*SMARCD1*	*SMC1A*	*SMC3*	*SMO*	*SNCAIP*	*SOCS1*
SOX10	*SOX17*	*SOX2*	*SOX9*	*SPEN*	*SPOP*	*SPTA1*	*SRC*	*SRSF2*	*STAG1*
STAG2	*STAT3*	*STAT4*	*STAT5A*	*STAT5B*	*STK11*	*STK40*	*SUFU*	*SUZ12*	*SYK*
TAF1	*TBX3*	*TCEB1*	*TCF3*	*TCF7L2*	*TERC*	*TERT*	*TET1*	*TET2*	*TFE3*
TFRC	*TGFBR1*	*TGFBR2*	*TMEM127*	*TMPRSS2*	*TNFAIP3*	*TNFRSF14*	*TOP1*	*TOP2A*	*TP53*

TP63	*TRAF2*	*TRAF7*	*TSC1*	*TSC2*	*TSHR*	*U2AF1*	*VEGFA*	*VHL*	*VTCN1*
WISP3	*WT1*	*XIAP*	*XPO1*	*XRCC2*	*YAP1*	*YES1*	*ZBTB2*	*ZBTB7A*	*ZFHX3*
ZNF217	*ZNF703*	*ZRSR2*							

CNVs									
AKT2	*ALK*	*AR*	*ATM*	*BRAF*	*BRCA1*	*BRCA2*	*CCND1*	*CCND3*	*CCNE1*
CDK4	*CDK6*	*CHEK1*	*CHEK2*	*EGFR*	*ERBB2*	*ERBB3*	*ERCC1*	*ERCC2*	*ESR1*
FGF1	*FGF10*	*FGF14*	*FGF19*	*FGF2*	*FGF23*	*FGF3*	*FGF4*	*FGF5*	*FGF6*
FGF7	*FGF8*	*FGF9*	*FGFR1*	*FGFR2*	*FGFR3*	*FGFR4*	*JAK2*	*KIT*	*KRAS*
LAMP1	*MDM2*	*MDM4*	*MET*	*MYC*	*MYCL*	*MYCN*	*NRAS*	*NRG1*	*PDGFRA*
PDGFRB	*PIK3CA*	*PIK3CB*	*PTEN*	*RAF1*	*RET*	*RICTOR*	*RPS6KB1*	*TFRC*	

Fusions									
ABL1	*ALK*	*BCR*	*BRAF*	*CD74*	*EGFR*	*ETV1*	*ETV4*	*ETV6*	*EWSR1*
FGFR2	*FGFR3*	*NAB2*	*NTRK1*	*NTRK2*	*NUTM1*	*PAX3*	*PAX8*	*PPARG*	*RET*
ROS1	*TFE3*	*TMPRSS2*							

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.

Note: The Phase 1 and Phase 2 clinical trials will be provided on request.

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