Germline Cancer Predisposition

Test Description

The MolQ Germline Cancer Predisposition Panel include genes associated with both common and rare hereditary cancers.

Clinician

Clinician Name: Dr Amit Verma
Medical Facility: Dr AV Institute of Personalized Therapy and Cancer Research (IPTCR)
Pathologist: Not Provided

Patient Demographic

Name: Ms. Sakshi Gupta
Sex: Female
Date of Birth/Age: 37 years
Disease: Asymptomatic

Specimen

Booking ID: 012407130127
Site: NA
Sample Type: Blood
Date of Collection:13-07-2024
Date of Booking:13-07-2024

CLINICAL SYNOPSIS

Sakshi Gupta is an asymptomatic individual with a family history of cancer. Her mother has endometrial carcinoma. She has been evaluated for pathogenic variations in the genes listed in Appendix 2.

RESULTS

Variants of uncertain significance related to the clinical phenotype are detected.

Gene (Transcript)#	Location	Variant	Zygosity	Disease (OMIM/Ref¶)	Inheritance	Classification$
SDHA (+) (NM_004168.4)	Exon 8	c.977T>A (p.Phe326Tyr)	Heterozygous	Paragangliomas 5	Autosomal Dominant	#Uncertain significance (PM2, PP2, PP3)
FANCA (-) (NM_000135.4)	Intron 1	c.79+5G>C (Splice region variant)	Heterozygous	Fanconi anemia complementation group A	Autosomal Recessive	#Uncertain significance (PM2)

$Genetic test results are reported based on the recommendations of American College of Medical Genetics.
#Due to lack of clinical evidence for this variant, it is classified as variant of uncertain significance.

CLINICAL CORRELATION AND VARIANT INTERPRETATION

SDHA **p.Phe326Tyr**

Variant description: The submitted sample shows heterozygous variant in Exon 8 of gene *SDHA* (chr5: g.233558T>A). A missense variant "T" to "A" detected at nucleotide position 977 leading to change in amino acid sequences from Phenyl Alanine to Tyrosine at codon 326. *In silico predictions*: The p.Phe326Tyr missense variant is predicted to be damaging by both SIFT and PolyPhen2. The phenylalanine residue at codon 326 of *SDHA* is conserved in all mammalian species. The nucleotide c.977 in *SDHA* is predicted conserved by GERP++ and PhyloP across 100 vertebrates. *Population frequency and Internal database*: The p.Phe326Tyr variant is novel (not in any individuals) in gnomAD All. The p.Phe326Tyr variant is novel (not in any individuals) in 1kG All. This variant is absent in reference laboratory internal database.

OMIM Phenotype: The p.Phe326Tyr variant is novel (not in any individuals) in gnomAD All. The p.Phe326Tyr variant is novel (not in any individuals) in 1kG All. Pheochromocytoma/paraganglioma syndrome-5 (PPGL5) is an autosomal dominant disorder characterized by the development of neuroendocrine tumors, usually in adulthood. Paragangliomas are tumors derived from paraganglia located throughout the body. Nonchromaffin types primarily serve as chemoreceptors and are located in the head and neck region (i.e., carotid body, jugular, vagal and tympanic regions), whereas chromaffin types have endocrine activity, conventionally referred to as 'pheochromocytomas,' and are usually located below the head and neck (i.e., adrenal medulla and pre- and para-vertebral thoracoabdominal regions). PPGL can manifest as nonchromaffin head and neck tumors only, adrenal and/or extraadrenal pheochromocytomas only, or a combination of the 2 types of tumors (Baysal, 2002; Neumann *et al*., 2004).

Variant Evidence

Variant Evidence for 2400144758.targeted — GRCh37 GRCh38

Chromosome: chr5

Position: 233,558

NC_000005.10 (GRCh38 Chr5): g.233558T>A

Allele	DP	%
T	6	66.67
A	3	33.33

Depth:9

Genotype: Heterozygous

Phred Quality Score: 26.00 — 1 in 100 probability of FP

Gene Impact

RefSeq Genes 110, NCBI — Transcripts (3)

Gene: *SDHA*

Transcript: NM_004168.4 — LRG_315t1, Complete, Forward

Effect: Missense — missense_variant

Protein: p.Phe326Tyr — NP_004159.2 (AA 326 of 665)

Exon: Exon 8 of 15

Coding: c.977T>A — Coding 976 of 1995 (49%)

Based on the above evidence$, ***this variant (SDHA: c.977T>A) is classified as Uncertain significance variant.***

FANCA **c.79+5G>C**

Variant description: The submitted sample shows heterozygous variant in Intron 1 of gene *FANCA* (chr16: g.89816532C>G). A splice region variant "G" to "C" detected at nucleotide position 79+5. *In silico predictions*: Combined Annotation Dependent Depletion (CADD) Score is Uncertain. *Population frequency and Internal database*: The c.79+5G>C variant is novel (not in any individuals) in gnomAD All. The c.79+5G>C variant is novel (not in any individuals) in 1kG All. This variant is absent in reference laboratory internal database.

OMIM Phenotype: Fanconi anemia of complementation group A (FANCA) is caused by homozygous or compound heterozygous mutation in the *FANCA* gene (607139) on chromosome 16q24. Fanconi anemia is a clinically and genetically heterogeneous disorder that causes genomic instability. Characteristic clinical features include developmental abnormalities in major organ systems, early-onset bone marrow failure, and a high predisposition to cancer. The cellular hallmark of FA is hypersensitivity to DNA crosslinking agents and high frequency of chromosomal aberrations pointing to a defect in DNA repair (summary by Deakyne and Mazin, 2011).

Variant Evidence

Variant Evidence for 2400144758.targeted — GRCh37 GRCh38

Chromosome: chr16

Position: 89,816,532

NC_000016.10 (GRCh38 Chr16): g.89816532C>G

Allele	DP	%
C	23	65.71
G	12	34.29

Depth:35

Genotype: Heterozygous

Phred Quality Score: 32.00 — 1 in 1,000 probability of FP

Gene Impact

RefSeq Genes 110, NCBI — Transcripts (4)

Gene: *FANCA*

Transcript: NM_000135.4 — Complete, Reverse

Effect: Intron — splice_region_variant

Protein: N/A — NP_000126.2 N/A

Intron: Intron 1 of 42 — Between Exons 1 and 2 of 43

Coding: c.79+5G>C — N/A

Based on the above evidence$, ***this variant (FANCA: c.79+5G>C) is classified as uncertain significance variant. Second variant of FANCA gene has not been detected and alternate method such as deletion/duplication analysis is recommended.***

ADDITIONAL FINDINGS

Gene (Transcript)#	Location	Variant	Zygosity/ Inheritance	Depth/ VAF	OMIM Phenotype	Classification$
HBB (-) (NM_000518.5) (chr11:g.5226925C>G)	Intron 1	c.92+5G>C (Splice region variant)	Heterozygous/ Autosomal Recessive	43x/ 46%	Beta-thalassemia	Pathogenic (PP3, PP5)

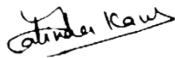
$Genetic test results are reported based on the recommendations of American College of Medical Genetics.

Beta-thalassemia

Beta-thalassemia can be caused by homozygous or compound heterozygous mutation in the beta-globin gene (*HBB*; 141900) on chromosome 11p15. A form of thalassemia. Thalassemia's are common monogenic diseases occurring mostly in Mediterranean and Southeast Asian populations. The hallmark of beta-thalassemia is an imbalance in globin-chain production in the adult HbA molecule. Absence of beta chain causes beta(0)-thalassemia, while reduced amounts of detectable beta globin causes beta(+)-thalassemia. In the severe forms of beta-thalassemia, the excess alpha globin chains accumulate in the developing erythroid precursors in the marrow. Their deposition leads to a vast increase in erythroid apoptosis that in turn causes ineffective erythropoiesis and severe microcytic hypochromic anemia. Clinically, beta-thalassemia is divided into thalassemia major which is transfusion dependent, thalassemia intermedia (of intermediate severity), and thalassemia minor that is asymptomatic.

RECOMMENDATIONS

- Segregation analysis of the variant by Sanger sequencing is recommended in affected and unaffected members of the family.
- Validation of the variant(s) by Sanger sequencing is recommended to rule out false positives.
- Genetic counselling is advised for interpretation on the consequences of the variant(s).
- The significance/classification of the variants might change based on parental and family members genetic testing.
- For questions about this report, or for assistance in locating nearby genetic counseling services, please contact the laboratory: contact@molq.in.
- If results obtained do not match the clinical findings, additional testing should be considered as per referring clinician's recommendations.



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APPENDIX 1: TEST METHODOLOGY

Method

Next Generation Sequencing:[1-5] DNA extracted from blood, saliva, amniotic fluid, CVS or any other standard source is used for targeted capture-based Library preparation. Targeted capture provides an efficient and sensitive means for sequencing specific genomic regions in a high-throughput manner. The libraries were sequenced to mean >85-100x coverage on Illumina Novaseq 6000 sequencing platform with Paired End 2x150 chemistry.
Illumina DRAGEN Bio-IT Platform was followed for identification of variants in the sample. The sequences obtained are assembled and aligned to reference sequences based on NCBI RefSeq transcripts and human genome build (GRCh38). Haplotype caller has been used to identify variants which are relevant to the clinical indication.
In addition to SNVs and small Indels, copy number variants (CNVs) are detected from targeted sequence data using the commercially available algorithm. This algorithm detects rare CNVs based on comparison of the read-depths of the test data with the matched aggregate reference dataset. Clinically relevant mutations were annotated using published variants in literature, Commercial datasets and a set of diseases databases.
Common variants are filtered based on allele frequency in 1000Genome, ExAC, gnomAD, dbSNP and reference laboratory's internal database.
Non-synonymous variants effect is calculated using multiple algorithms such as PolyPhen-2, SIFT, MutationTaster2. Based on annotations data, ACMG rules-based classification performed for classification of variants identified through next generation sequencing study.

Average Sequencing Depth (x)	Percentage Target Base Pairs Covered		
	1x	≥5x	≥20x
55.98	98.91	98.67	96.58

Title	Data
Total data generated (Gb)	6.14
Total reads aligned (%)	100.00
Reads that passed alignment (%)	99.51
Data ≥ Q30 (%)	94.05

$The classification of the variations is done based on American College of Medical Genetics as described below[6]

Variant	A change in a gene. This could be disease causing (pathogenic) or not disease causing (benign).
Benign	A variant which is known not to be responsible for disease has been detected. Generally, no further action is warranted on such variants when detected.
Likely Benign	A variant which is very unlikely to contribute to the development of disease however, the scientific evidence is currently insufficient to prove this conclusively. Additional evidence is expected to confirm this assertion of Pathogenicity.
Pathogenic	A disease-causing variation in a gene which can explain the patient's symptoms has been detected. This usually means that a suspected disorder for which testing had been requested has been confirmed.
Likely Pathogenic	A variant which is very likely to contribute to the development of disease however, the scientific evidence is currently insufficient to prove this conclusively. Additional evidence is expected to confirm this assertion of pathogenicity.
Variant of Uncertain Significance	A variant has been detected, but it is difficult to classify it as either pathogenic (disease causing) or benign (non-disease causing) based on current available scientific evidence. Further testing of the patient or family members as recommended by your clinician may be needed. It is probable that their significance can be assessed only with time, subject to availability of scientific evidence.

DISCLAIMER

- Interpretation of variants in this report is performed to the best knowledge of the laboratory based on the information available at the time of reporting. The classification of variants can change over time and MolQ cannot be held responsible

for this. Please feel free to contact MolQ Laboratory (contact@molq.in) in the future to determine if there have been any changes in the classification of any variations. Re-analysis of variants in previously issued reports in light of new evidence is not routinely performed, but may be considered upon request.

- The sensitivity of this assay to detect large deletions/duplications of more than 10 bp or copy number variations (CNV) is 70-75%. The CNVs detected with this assay have to be confirmed by alternate method such as MLPA & Microarray.
- Due to inherent technology limitations, coverage is not uniform across all regions. Hence pathogenic variants present in areas of insufficient coverage as well as those variants which currently do not corelate with the provided phenotype may not be analyzed/ reported. Additionally, it may not be possible to fully resolve certain details about variants, such as mosaicism, phasing, or mapping ambiguity.
- Genes with pseudogenes, paralog genes and genes with low complexity may have decreased sensitivity and specificity of variant detection and interpretation due to inability of the data and analysis tools to unambiguously determine the origin of the sequence data in such regions.
- The mutations have not been validated/confirmed by Sanger sequencing.
- Incidental or secondary findings (if any) that meet the ACMG guidelines[2] can be given upon request.
- Sequence and copy number variants are reported according to the Human Genome Variation Society (HGVS).
- The transcript used for clinical reporting generally represents the canonical transcript, 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.
- The report shall be generated within turnaround time (TAT), however, such TAT may vary depending upon the complexity of test(s) requested. MolQ Laboratory under no circumstances will be liable for any delay beyond afore mentioned TAT.
- It is hereby clarified that the report(s) generated from the test(s) do not provide any diagnosis or opinion or recommends any cure in any manner. MolQ Laboratory hereby recommends the patient and/or the guardians of the patients, as the case may be, to take assistance of the clinician or a certified physician or doctor, to interpret the report(s) thus generated. MolQ Laboratory hereby disclaims all liability arising in connection with the report(s).
- In a very few cases genetic test may not show the correct results, e.g. because of the quality of the material provided to MolQ Laboratory. In case where any test provided by MolQ Laboratory fails for unforeseeable or unknown reasons that cannot be influenced by MolQ Laboratory in advance, MolQ Laboratory shall not be responsible for the incomplete, potentially misleading or even wrong result of any testing if such could not be recognized by MolQ Laboratory in advance.
- This is a laboratory developed test and the development and the performance characteristics of this test was determined by reference laboratory.

LIMITATIONS

- Genetic testing is an important part of the diagnostic process. However, genetic tests may not always give a definitive answer. In some cases, testing may not identify a genetic variant even though one exists. This may be due to limitations in current medical knowledge or testing technology. Accurate interpretation of test results may require knowing the true biological relationships in a family. Failing to accurately state the biological relationships in {my/my child's} family may result in incorrect interpretation of results, incorrect diagnoses, and/or inconclusive test results.
- Test results are interpreted in the context of clinical findings, family history and other laboratory data. Only variations in genes potentially related to the proband's medical condition are reported. Rare polymorphisms may lead to false negative or positive results. Misinterpretation of results may occur if the information provided is inaccurate or incomplete.
- Triplet repeat expansions, translocations, large deletion or duplications and chromosomal rearrangements events are currently not reliably detected by next generation sequencing.
- This assay is not meant to interrogate most promoter regions, deep intronic regions, or other regulatory elements, other gene rearrangements like inversion or translocation and does not detect single or multiexon deletions or duplications.
- Genetic testing is highly accurate. Rarely, inaccurate results may occur for various reasons. These reasons include, but are not limited to: mislabelled samples, inaccurate reporting of clinical/medical information, rare technical errors or unusual circumstances such as bone marrow transplantation, blood transfusion; or the presence of change(s) in such a small percentage of cells that may not be detectable by the test (mosaicism).
- It should be noted that this test does not sequence all bases in a human genome, not all variants have been identified or interpreted, and this report is limited only to variants with evidence for causing or contributing to disease/clinical details provided to MolQ Laboratory.
- Testing has been performed assuming that the sample received belongs to the above named individual and any stated relationships between individuals are accepted as true.
- The results should be interpreted in the context of the patient’s medical evaluation, family history and racial/ethnic

background. Please note that variant classification and/or interpretation may change over time if more information available. Reinterpretation of multi gene next generation sequencing data is recommended on an annual basis and may be requested by a medical provider.

REFERENCES

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3. 1000 Genomes Project Consortium et al., A global reference for human genetic variation. Nature, 526(7571): 68-74, 2015.
4. Lek M. et al., Analysis of protein-coding genetic variation in 60,706 humans. Nature, 536(7616):285-91, 2016.
5. McLaren W. et al., Deriving the consequences of genomic variants with the Ensembl API and SNP Effect Predictor. Bioinformatics., 26(16):2069-70, 2010.
6. Richards S. *et al*., Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology, Genet Med., 17(5):405-24, 2015.
7. Kalia S.S. et al., Recommendations for reporting of secondary findings in clinical exome and genome sequencing, 2016 update (ACMG SF v2.0): a policy statement of the American College of Medical Genetics and Genomics. Genet Med., 19(2):249-255, 2017.

APPENDIX 2: GENE LIST WITH COVERAGE

Gene	Percentage of coding region covered	Gene	Percentage of coding region covered	Gene	Percentage of coding region covered	Gene	Percentage of coding region covered
ABRAXAS1	100.00	*EP300*	100.00	*MEN1*	100.00	*RHOH*	100.00
ACD	100.00	*EPCAM*	100.00	*MET*	100.00	*RINT1*	100.00
ACVRL1	100.00	*ERCC1*	100.00	*MITF*	100.00	*RIT1*	100.00
ADA	100.00	*ERCC2*	100.00	*MLH1*	100.00	*RMRP*	0.00
AIP	100.00	*ERCC3*	100.00	*MLH3*	100.00	*RNASEL*	100.00
AKT1	100.00	*ERCC4*	100.00	*MRE11*	100.00	*RNF43*	100.00
ALK	100.00	*ERCC5*	100.00	*MSH2*	100.00	*RPS20*	100.00
ANKRD26	100.00	*ETV6*	100.00	*MSH3*	100.00	*RRAS*	100.00
APC	100.00	*EXO1*	100.00	*MSH6*	100.00	*RUNX1*	100.00
APCS	100.00	*EXT1*	100.00	*MSR1*	100.00	*SAMD9*	100.00
AR	100.00	*EXT2*	100.00	*MUTYH*	100.00	*SAMD9L*	100.00
ATM	100.00	*EZH2*	100.00	*MXI1*	100.00	*SBDS*	100.00
AXIN1	100.00	*FADD*	100.00	*MYC*	100.00	*SDHA*	100.00
AXIN2	100.00	*FAM111B*	100.00	*MYD88*	100.00	*SDHAF2*	100.00
B2M	100.00	*FAN1*	100.00	*MYSM1*	100.00	*SDHB*	100.00
BAP1	100.00	*FANCA*	100.00	*NBN*	100.00	*SDHC*	100.00
BARD1	100.00	*FANCB*	100.00	*NF1*	100.00	*SDHD*	100.00
BCL2	100.00	*FANCC*	100.00	*NF2*	100.00	*SH2D1A*	100.00
BLM	100.00	*FANCD2*	100.00	*NHEJ1*	100.00	*SHOC2*	100.00
BMPR1A	100.00	*FANCE*	100.00	*NOTCH1*	100.00	*SLX4*	100.00
BRAF	100.00	*FANCF*	100.00	*NOTCH2*	100.00	*SMAD4*	100.00
BRCA1	100.00	*FANCG*	100.00	*NPAT*	100.00	*SMARCA2*	100.00
BRCA2	100.00	*FANCI*	100.00	*NRAS*	100.00	*SMARCA4*	100.00
BRIP1	100.00	*FANCL*	100.00	*NSD1*	100.00	*SMARCB1*	100.00
BTNL2	100.00	*FANCM*	100.00	*NSUN2*	100.00	*SMARCE1*	100.00
BUB1B	100.00	*FAS*	100.00	*NTHL1*	100.00	*SOCS1*	100.00
CARD11	100.00	*FASLG*	100.00	*PALB2*	100.00	*SOS1*	100.00
CARMIL2	100.00	*FCHO1*	100.00	*PAX5*	100.00	*SOS2*	100.00
CASP10	100.00	*FGFR2*	100.00	*PDGFRA*	100.00	*SPINK1*	100.00
CASP8	100.00	*FH*	100.00	*PHOX2B*	100.00	*SPRED1*	100.00
CASR	100.00	*FLCN*	100.00	*PIK3CA*	100.00	*SRP72*	96.83
CBL	100.00	*FOXO1*	100.00	*PIK3CD*	100.00	*STAT3*	100.00
CD27	100.00	*GALNT12*	100.00	*PIK3R1*	100.00	*STAT6*	100.00
CD70	100.00	*GATA2*	100.00	*PIM1*	100.00	*STK11*	100.00
CD79A	100.00	*GNA13*	100.00	*PMS1*	100.00	*STK4*	100.00
CD79B	100.00	*GPC3*	100.00	*PMS2*	100.00	*STXBP2*	100.00
CD82	100.00	*GPR101*	100.00	*POLD1*	100.00	*SUFU*	100.00
CDC73	100.00	*GREM1*	100.00	*POLE*	100.00	*TERC*	0.00
CDH1	100.00	*HAVCR2*	100.00	*POLH*	100.00	*TERF2IP*	100.00
CDK4	100.00	*HBB*	100.00	*POT1*	100.00	*TERT*	100.00
CDKN1B	100.00	*HNF1A*	100.00	*PPM1D*	100.00	*TET2*	100.00
CDKN1C	100.00	*HNF1B*	100.00	*PRF1*	100.00	*TGFB1*	100.00
CDKN2A	100.00	*HOXB13*	100.00	*PRKAR1A*	100.00	*TGFBR2*	100.00
CDKN2B	100.00	*HRAS*	100.00	*PRKCD*	100.00	*TINF2*	100.00

CEBPA	100.00	*IKZF1*	100.00	*PRSS1*	100.00	*TMEM127*	100.00
CEP57	100.00	*IL2RA*	100.00	*PTCH1*	100.00	*TNFAIP3*	100.00
CFTR	100.00	*IL2RB*	100.00	*PTCH2*	100.00	*TNFRSF13B*	100.00
CHEK2	100.00	*ITK*	100.00	*PTEN*	100.00	*TNFRSF14*	100.00
CPA1	100.00	*KIF1B*	100.00	*PTPN11*	100.00	*TOX3*	96.65
CREBBP	100.00	*KIT*	100.00	*RAC2*	100.00	*TP53*	100.00
CTLA4	100.00	*KITLG*	100.00	*RAD50*	100.00	*TPP2*	100.00
CTNNA1	100.00	*KLHDC8B*	100.00	*RAD51*	100.00	*TRIP13*	100.00
CTPS1	100.00	*KMT2D*	100.00	*RAD51C*	100.00	*TSC1*	100.00
CYLD	100.00	*KRAS*	100.00	*RAD51D*	100.00	*TSC2*	100.00
DDB2	100.00	*LIG4*	100.00	*RAF1*	100.00	*UBE2T*	100.00
DDX41	100.00	*LSP1*	79.12	*RASA2*	100.00	*VHL*	100.00
DICER1	100.00	*LZTR1*	100.00	*RASAL1*	100.00	*VPS13B*	100.00
DIS3L2	100.00	*MAD2L2*	100.00	*RASGRP1*	100.00	*WAS*	100.00
DKC1	100.00	*MAGT1*	100.00	*RB1*	100.00	*WRN*	100.00
DOCK8	100.00	*MAP2K1*	100.00	*RECQL*	100.00	*WT1*	100.00
EFL1	100.00	*MAP2K2*	100.00	*RECQL4*	100.00	*XIAP*	100.00
EGFR	100.00	*MAP3K1*	100.00	*REST*	100.00	*XPA*	100.00
ELAC2	100.00	*MAX*	100.00	*RET*	100.00	*XPC*	100.00
ELANE	100.00	*MC1R*	100.00	*RFWD3*	100.00	*XRCC2*	100.00
ENG	100.00	*MCM4*	100.00	*RHBDF2*	100.00	*XRCC3*	100.00

MTDNA GENES LIST WITH COVERAGE

Gene	Percentage of coding region covered	Gene	Percentage of coding region covered	Gene	Percentage of coding region covered
MT-ATP6	100.00	*MT-ND6*	100.00	*MT-TL1*	100.00
MT-ATP8	100.00	*MT-RNR1*	100.00	*MT-TL2*	100.00
MT-CO1	100.00	*MT-RNR2*	100.00	*MT-TM*	100.00
MT-CO2	100.00	*MT-TA*	100.00	*MT-TN*	100.00
MT-CO3	100.00	*MT-TC*	100.00	*MT-TP*	100.00
MT-CYB	100.00	*MT-TD*	100.00	*MT-TQ*	100.00
MT-ND1	100.00	*MT-TE*	100.00	*MT-TR*	100.00
MT-ND2	100.00	*MT-TF*	100.00	*MT-TS1*	100.00
MT-ND3	100.00	*MT-TG*	100.00	*MT-TS2*	100.00
MT-ND4	100.00	*MT-TH*	100.00	*MT-TT*	100.00
MT-ND4L	100.00	*MT-TI*	100.00	*MT-TV*	100.00
MT-ND5	100.00	*MT-TK*	100.00	*MT-TW*	100.00
MT-TY	100.00				