Client: Example Client ABC123
123 Test Drive
Salt Lake City, UT 84108
UNITED STATES

Physician: Doctor, Example

Patient: Patient, Example

DOB	1/15/1990
Gender:	Male
Patient Identifiers:	01234567890ABCD, 012345
Visit Number (FIN):	01234567890ABCD
Collection Date:	00/00/0000 00:00

Mitochondrial Disorders Panel (mtDNA and Nuclear Genes)

ARUP test code 3001959

Ordering Physician Name	[redacted] Performed by: GeneDx 207 Perry Parkway Gaithersburg, MD 20877 Anne Maddalena, Ph.D.,FACMG,
Ordering Physician Phone Number	NOT PROVIDED Performed by: GeneDx 207 Perry Parkway Gaithersburg, MD 20877 Anne Maddalena, Ph.D.,FACMG,
EER Mito Disorders, mtDNA/Nuclear Genes	See Note
Mito Disorders, mtDNA and Nuclear Genes	*

POSITIVE: See Interpretation

Date Test(s) Started: 3/10/2020 09:50:00
Test(s) Requested Combined Mito Genome Plus Mito Focused Nuclear Gene Panel / Sequencing and Deletion Analysis of the Mitochondrial Genome and Sequencing and Deletion/Duplication Analysis of 202 Nuclear Genes
Clinical Indication Not provided
Result(s): POSITIVE: See Interpretation
GeneMode of InheritanceVariantHeteroplasmy (%)Classification
MT-ND4Maternal
m.11778 G>A
p.R340H
HomoplasmicPathogenic Variant
GeneMode of InheritanceVariantZygosityClassification
TMEM126B
Autosomal Recessivec.635 G>T
p.G212VHeterozygousPathogenic Variant
This individual is also heterozygous for variant(s) of uncertain significance in genes associated with autosomal recessive disorders (see table below). A second reportable variant, as expected for an autosomal recessive disorder, was not identified in these genes. At present, the finding of single variant(s) of

H=High, L=Low, *=Abnormal, C=Critical

uncertain clinical significance does not establish a molecular diagnosis.
This individual's mitochondrial haplogroup and a table of observed variants in the mitochondrial genome are also provided.* The observed variants have not been reported to be associated with a disorder of mitochondrial metabolism when present in association with this individual's specific haplogroup.
Interpretation This individual is homoplasmic for them.11778 G>A pathogenic variant in the MT-ND4 gene, which is commonly associated with Leber's hereditary optic neuropathy (LHON). LHON-causing mtDNA variants have markedly reduced penetrance as approximately 50% of males and 90% of females who harbor a primary LHON-causing mtDNA pathogenic variant do not develop blindness (Yu-Wai-Man, P. and Chinnery, P. 2001).
This individual is heterozygous for a pathogenic variant in theTMEM126B gene. As this gene is associated with an autosomal recessive disorder and no second variant was identified, this finding does not establish a molecular diagnosis. Of note, exon-level deletion/duplication analysis of this gene is not available as part of this test.
Recommendation(s) Clinical correlation and genetic counseling is recommended to discuss the implications of this test result. There are no recommendations for ongoing surveillance of asymptomatic individuals who harbor LHON-pathogenic variants; however, if any visual problems arise, these individuals should seek medical attention immediately (Yu-Wai-Man, P. and Chinnery, P. 2001).Mitochondrial DNA disorders are maternally inherited. Testing appropriate matrilineal relatives for them.11778 G>A variant is available.
This result permits targeted carrier testing for the pathogenic variant in theTMEM126B gene for family members.
The level of variant heteroplasmy may differ among tissues so that mtDNA variants may be detected in some tissues, but not others. Therefore it is usually best to test an involved tissue, such as muscle or liver. If clinically indicated, full sequence analysis and deletion testing of the mitochondrial genome can be repeated on a muscle biopsy (approximately 50mg).The MitoXpanded panel, which includes concurrent patient and parent sequencing of approximately 1800 genes associated with mitochondrial disorders or a similar phenotype is also available. Whole exome sequencing could also be considered. Please visit our website for additional information: http: //www.genedx.com.
Resources MyGene2 is a portal through which families with rare genetic conditions who are interested in sharing their health and genetic information can connect with other families, clinicians, and researchers. If you are interested in learning more and/or participating, please visit www.mygene2.org.
GenomeConnect is an NIH initiative created to enable individuals and families with the same genetic variant or medical history to connect and share de-identified information. If you are interested in participating, please visit www.genomeconnect.org.
MT-ND4 Genome Summary:
The mitochondrial genome spans 16.5 kb and contains 37 genes, all of which are essential for normal mitochondrial function. Thirteen of these genes encode proteins that are subunits of the electron transport chain, two of the genes encode rRNA molecules (12S and 16S) and the remaining twenty-two genes encode tRNAs that are involved in the translation process of the mitochondrial genome. It is estimated that approximately 29% of patients (approximately 15-20% pediatric and approximately 40% adult) with primary mitochondrial disorders are caused by pathogenic variants in the mitochondrial genome (Chinnery, 2003; Dimauro and Davidzon, 2005; Jaksch et al., 2001; Swalwell et al., 2011). Mitochondrial disorders are clinically heterogeneous and result from dysfunction of the mitochondrial respiratory chain. Mitochondrial disorders may affect a single organ, but many involve multiple organ systems particularly those that are highly dependent on aerobic metabolism (brain, skeletal muscle, heart, kidney and endocrine system), and patients may present at any age. Some affected individuals exhibit clinical features that fall into a discrete clinical syndrome; however, often the

clinical features are highly variable and non-specific and many affected persons do not fit into one particular category. Variants in mtDNA arise de novo or are maternally inherited. Usually, mtDNA variants affect only a fraction of the mtDNA; the coexistence of normal and mutant mtDNA is called heteroplasmy. When the percentage of mutant mtDNA (variant load) reaches a certain threshold that varies by tissue type, age, and specific mtDNA variant the function of that tissue may become impaired (Longo, N., 2003). As the mutation load varies within and between tissues, the manifestation of mitochondrial disease may reflect tissue-specific variant load (Tarnopolsky, M and Raha, S., 2005). Many factors can affect the percent heteroplasmy these include physiologic processes that are affected by the mtDNA variant, the function of the tissue, and the rate of cell division in that tissue. Variants in mtDNA may only be identified in specific tissues, particularly those with a lower rate of cell division such as skeletal muscle, heart and brain (Longo, N., 2003).
m.11778 G>A: p.Arg340His (R340H) (CGC>CAC) in the MT-ND4 gene (NC_012920.1)Commonly associated with Leber's hereditary optic neuropathy (LHON) at near homoplasmic levels in blood (Wallace et al., 1988); however, approximately 50% of males and 90% of females who harbor a primary LHON-causing mtDNA pathogenic variant do not develop blindness (Yu-Wai-Man, P. and Chinnery, P. 2016)Most affected individuals harbor this variant at greater than 70% heteroplasmy in blood (Yu-Wai-Man, P. and Chinnery, P. 2016)Published functional studies showed oxygen consumption was significantly decreased in cybrid clones containing m.11778 G>A variant (Vergani et al., 1995)In-silico analyses, including protein predictors and evolutionary conservation, support a deleterious effectSmoking has been reported to have a consistent role in increasing disease penetrance in LHON. Asymptomatic LHON carriers are advised to avoid smoking and to moderate alcohol consumption (Kirkman et al., 2009)
We interpret this as a Pathogenic Variant.
TMEM126B Gene Summary: The TMEM126B gene encodes a mitochondrial protein that assists in the assembly of mitochondrial complex I from its 44 subunits (Andrews et al., 2013). Biallelic pathogenic variants in TMEM126B result in a complex I deficiency (Alston et al., 2016; Sanchez-Caballero et al., 2017). Most affected individuals presented with myalgia and exercise intolerance with onset generally in late childhood, but ranging from 3 to 38 years. One described patient had a notably more severe course, with onset at 2 months of age and multiorgan involvement including respiratory failure, cardiomyopathy, renal acidosis, growth failure, and renal insufficiency (Alston et al., 2016). p.Gly212Val (G212V) (GGT>GTT): c.635 G>T in exon 5 of the TMEM126B gene (NM_018480.4) Observed as homozygous or heterozygous with a pathogenic variant on the opposite allele (in trans) in multiple individuals with mitochondrial complex I deficiency (Alston et al., 2016; Sanchez-Caballero et al., 2016; Theunissen et al., 2017) Observed in 0.1072% (295/275284 alleles) in large population cohorts, and no individuals were reported to be homozygous (Lek et al., 2016) In silico analysis, which includes protein predictors and evolutionary conservation, supports a deleterious effect
We interpret this as a Pathogenic Variant.
Additional Comments At this time, the variants listed below are classified as variants of uncertain significance as they do not meet criteria to be classified otherwise (http: //www.ncbi.nlm.nih.gov/clinvar/submitters/26957). Information on population data and in-silico analysis can be found in the supplemental variant information tables at the end of the report.
* Reported in an individual with pontocerebellar hypoplasia who did not have a second variant in the RARS2 gene identified by whole genome sequencing (Reuter et al., 2018) ; Functional studies found that this variant did not affect mitochondrial localization of mitochondrial aminoacyl-tRNA synthetases (Gonzalez-Serrano et al. 2018)
Additional Variants of Uncertain Significance (See Above)
GeneMode of InheritanceVariantZygosityClassification
GFM2Autosomal Recessivec.1387 C>G

H=High, L=Low, *=Abnormal, C=Critical

p.R463GHeterozygousVariant of Uncertain Significance
RARS2Autosomal Recessivec.997 C>G*
p.R333G
HeterozygousVariant of Uncertain Significance
TARS2Autosomal Recessivec.3 G>A
p.M1?HeterozygousVariant of Uncertain Significance
Genes Evaluated AARS2, ABCB7, ACAD9, ACO2, ADCK4, AFG3L2, AGK, AIFM1, ALAS2, APOPT1, ATP5A1, ATP5E, ATP7B, ATPAF2, AUH, BCS1L, BOLA3, C10ORF2, C12ORF65, C19ORF12, C20ORF7, C8ORF38, CARS2, CLPB, COA5, COA6, COASY, COQ2, COQ4, COQ6, COQ7, COQ8A, COQ9, COX10, COX14, COX15, COX20, COX6A1, COX6B1, COX8A, CYC1, DARS2, DGUOK, DLAT, DLD, DNA2, DNAJC19, DNM1L, EARS2, ECHS1, ELAC2, ETFA, ETFB, ETFDH, ETHE1, FARS2, FASTKD2, FBXL4, FDX1L, FH, FLAD1, FOXRED1, GARS, GCDH, GFER, GFM1, GFM2, GLRX5, GTPBP3, GYG2, HARS2, HMGCL, HTRA2, IARS2, IBA57, ISCA2, ISCU, LAMP2, LARS, LARS2, LIAS, LIPT1, LRPPRC, LYRM4, LYRM7, MARS2, MFF, MFN2, MGME1, MICU1, MPC1, MPV17, MRPL12, MRPL3, MRPL44, MRPS16, MRPS22, MRPS7, MT-ATP6, MT-ATP8, MT-CO1, MT-CO2, MT-CO3, MT-CYB, MT-DLOOP, MTFMT, MT-ND1, MT-ND2, MT-ND3, MT-ND4, MT-ND4L, MT-ND5, MT-ND6, MTO1, MTPAP, MT-RNR1, MT-RNR2, MT-TA, MT-TC, MT-TD, MT-TE, MT-TF, MT-TG, MT-TH, MT-TI, MT-TK, MT-TL1, MT-TL2, MT-TM, MT-TN, MT-TP, MT-TQ, MT-TR, MT-TS1, MT- TS2, MT-TT, MT-TV, MT-TW, MT-TY, NARS2, NDUFA1, NDUFA10, NDUFA11, NDUFA12, NDUFA2, NDUFA4, NDUFA9, NDUFAF1, NDUFAF2, NDUFAF3, NDUFAF4, NDUFAF7, NDUFB11, NDUFB3, NDUFB9, NDUFS1, NDUFS2, NDUFS3, NDUFS4, NDUFS6, NDUFS7, NDUFS8, NDUFV1, NDUFV2, NFS1, NFU1, NR2F1, NUBPL, OPA1, OPA3, OTC, PARS2, PC, PCCA, PCCB, PDHA1, PDHB, PDHX, PDP1, PDSS1, PDSS2, PET100, PNPT1, POLG, POLG2, PRKAG2, PUS1, QARS, RARS, RARS2, RMND1, RNASEH1, RRM2B, SARS2, SCO1, SCO2, SDHA, SDHAF1, SERAC1, SFXN4, SLC19A2, SLC19A3, SLC22A5, SLC25A26, SLC25A3, SLC25A38, SLC25A4, SLC25A46, SPAST, SPG7, SUCLA2, SUCLG1, SURF1, TACO1, TARS2, TAZ, TFAM, TIMM8A, TK2, TMEM126A, TMEM126B, TMEM70, TPK1, TRIT1, TRMT10C, TRMU, TRNT1, TSFM, TTC19, TUFM, TWNK, TYMP, UQCC2, UQCC3, UQCRB, UQCRC2, UQCRQ, VARS2, WDR45, WFS1, YARS2
Methods Genomic DNA was extracted from the submitted specimen. For skin punch biopsies, fibroblasts were cultured and used for DNA extraction. The DNA was enriched for the complete coding regions and splice junctions of the genes on this panel using a proprietary targeted capture system developed by GeneDx for next-generation sequencing with CNV calling (NGS-CNV). The enriched targets were simultaneously sequenced with paired-end reads on an Illumina platform. Bi-directional sequence reads were assembled and aligned to reference sequences based on NCBI RefSeq transcripts and human genome build GRCh37/UCSC hg19. After gene specific filtering, data were analyzed to identify sequence variants and most deletions and duplications involving coding exons at the exon-level; however, technical limitations and inherent sequence properties effectively reduce this resolution for some genes. Due to the presence of non-functional pseudogenes, regions of the GYG2, NR2F1, PDSS1 and TSFM genes were not fully sequenced by this method. For the COQ7, COX8A, HTRA2, NDUFB11, RNASEH1, SCO2, SDHA, SLC25A26, SLC25A46, TFAM, TMEM126B and TRMT10C gene(s), sequencing but not deletion/duplication analysis, was performed. Alternative sequencing or copy number detection methods were used to analyze regions with inadequate sequence or copy number data by NGS. Sequence variants are reported according to the Human Genome Variation Society (HGVS) guidelines. Copy number variants are reported based on the probe coordinates, the coordinates of the exons involved, or precise breakpoints when known. The entire mitochondrial genome from the submitted sample was also amplified and sequenced using Next Generation sequencing. DNA sequence was assembled and analyzed in comparison with the revised Cambridge Reference Sequence (rCRS) and the reported variants listed in the MITOMAP database (http://www.mitomap.org). Reported clinically significant variants were confirmed by an appropriate method. Reportable variants include pathogenic variants, likely pathogenic variants and variants of uncertain significance. Likely benign and benign variants, if present, are not routinely reported but are available upon request.

H=High, L=Low, *=Abnormal, C=Critical

Disclaimer Genetic testing using the methods applied at GeneDx is expected to be highly accurate. Normal findings do not rule out the diagnosis of a genetic disorder since some genetic abnormalities may be undetectable by this test. The methods used cannot reliably detect deletions of 20bp to 500bp in size, or insertions of 10bp to 500 bp in size. Sequencing cannot detect low-level mosaicism. The copy number assessment methods used with this test cannot reliably detect mosaicism and cannot identify balanced chromosome aberrations. Rarely, incidental findings of large chromosomal rearrangements outside the gene of interest may be identified. Regions of certain genes have inherent sequence properties (for example: repeat, homology, or pseudogene regions, high GC content, rare polymorphisms) that yield suboptimal data, potentially impairing accuracy of the results. For mitochondrial DNA (mtDNA) deletions, this test will detect almost all disease-associated heteroplasmy reported to date; levels of heteroplasmy of 5% or lower may not be detected and the standard deviation for heteroplasmy of large-scale deletions is estimated to be 5%. For mtDNA point mutations, novel mutations with a heteroplasmy lower than 5% may not be detected. False negative results may also occur in the setting of bone marrow transplantation, recent blood transfusion, or suboptimal DNA quality. As the ability to detect genetic variants and naming conventions can differ among laboratories, rare false negative results may occur when no positive control is provided for testing of a specific variant identified at another laboratory. The chance of a false positive or false negative result due to laboratory errors incurred during any phase of testing cannot be completely excluded. Interpretations are made with the assumption that any clinical information provided, including family relationships, are accurate. Consultation with a genetics professional is recommended for interpretation of results.
This test was developed and its performance characteristics determined by GeneDx. This test has not been cleared or approved by the U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary. The test is used for clinical purposes and should not be regarded as investigational or for research. The laboratory is certified under the Clinical Laboratory Improvement Amendments of 1988 (CLIA) as qualified to perform high-complexity clinical testing.
References Lek et al. (2016) Nature 536 (7616): 285-91 (PMID: 27535533);Stenson et al. (2014) Human genetics 133 (1): 1-9 (PMID: 24077912);Landrum et al. (2016) Nucleic Acids Res. 44 (D1): D862-8 (PMID: 26582918);Lott et al. (2013) Curr Protoc Bioinformatics 44 : 1.23.1-26 (PMID: 25489354);Sanchez-Caballero et al. (2016) Am. J. Hum. Genet. 99 (1): 208-16 (PMID: 27374773);Alston et al. (2016) Am. J. Hum. Genet. 99 (1): 217-27 (PMID: 27374774); Chinnery et al. (2003) Trends Genet. 19 (2): 60-2 (PMID: 12547509).; Dimauro et al. (2005) Ann. Med. 37 (3): 222-32 (PMID: 16019721); Jaksch et al. (2001) Journal Of Medical Genetics 38 (10): 665-73 (PMID: 11584044); Swalwell et al. (2011) Eur. J. Hum. Genet. 19 (7): 769-75 (PMID: 21364701); Longo et al. (2003) Neurol Clin 21 (4): 817-31 (PMID: 14743651); Tarnopolsky et al. (2005) Med Sci Sports Exerc 37 (12): 2086-93 (PMID: 16331134); Wallace et al., (1998) Science 242: 1427-30. Yu-Wai-Man, P. and Chinnery, P. (Updated [Sept 19, 2013]). Leber's Hereditary Optic Neuropathy. In: GeneReviews at Genetests: Medical Genetics Information Resource (database online). Copyright, University of Washington, Seattle. 1997-2014. Available at http: //www.genetests.org. Horvath et al. (2000) Journal of neurology 247 (1): 65-7; Wallace et al. (1988) Science 242 (4884): 1427-30 (PMID: 3201231); Yu-Wai-Man et al. Leber Hereditary Optic Neuropathy. 2000 Oct 26 [Updated 2016 Jun 23]. In: Adam MP, Ardinger HH, Pagon RA, et al., editors. GeneReviews [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2019. (Available from: https: //www.ncbi.nlm.nih.gov/books/NBK1174/): (PMID: 20301353); Vergani et al. (1995) Biochem. Biophys. Res. Commun. 210 (3): 880-8 (PMID: 7763260); Kirkman et al. (2009) Brain 132 (Pt 9): 2317-26 (PMID: 19525327) ; Alston et al. (2016) Am. J. Hum. Genet. 99 (1): 217-27 (PMID: 27374774) Sanchez-Caballero et al.

H=High, L=Low, *=Abnormal, C=Critical

(2016) Am. J. Hum. Genet. 99 (1): 208-16 (PMID: 27374773); Theunissen et al. (2017) Front Mol Neurosci 10 : 336 (PMID: 29093663) ; Reuter et al. (2018) CMAJ 190 (5): E126-E136 (PMID: 29431110); Gonzalez-Serrano et al. (2018) J. Biol. Chem. 293 (35): 13604-13615 (PMID: 30006346);
###
Gene: Coding DNATMEM126B: c.635 G>TGFM2: c.1387 C>GRARS2: c.997 C>GTARS2: c.3 G>A
Variant (Protein)p.Gly212Val (G212V)p.Arg463Gly (R463G)p.Arg333Gly (R333G)p.Met1? (M1?)
ClassificationPathogenic VariantVariant of Uncertain SignificanceVariant of Uncertain SignificanceVariant of Uncertain Significance
ZygosityHeterozygousHeterozygousHeterozygousHeterozygous
Chr: Position11: 853472155: 740327446: 882312201: 150459929
dbSNPrs141542003rs377321462
gnomAD_Freq0.00110.00000.00000.0000
gnomAD_AMR0.000151210.000000000.000000000.00000000
gnomAD_NFE0.001896650.000061790.000035240.00006483
gnomAD_AFR0.000161130.000000000.000000000.00000000
gnomAD_EAS0.000000000.000000000.000000000.00000000
gnomAD_FIN0.001328610.000000000.000000000.00000000
gnomAD_Other0.001562060.000000000.000000000.00000000
gnomAD_SAS0.000034560.000000000.00000000
gnomAD_ASJ0.000000000.000000000.000000000.00000000
gnomAD_Hom0000
Provean-6.17 (D)-1.13 (N)-6.92 (D)-1.31 (N)
ClinVarUncertain significance
This supplement provides evidence to support the classification of each reportable variant in the attached result report. This information is provided as a resource. It is not inclusive of all available information used by GeneDx for variant classification, and individual data elements may be weighted differently to derive the classification. This information is subject to change over time and may differ from what is currently available. Results should always be interpreted in the context of the patient's clinical presentation.Blank fields indicate that no data was available at time of analysis.dbSNP - NCBI repository for single base nucleotide substitutions and short deletion and insertion polymorphismsThe Genome Aggregation Database (gnomAD) combines exome and genome sequencing data from a variety of large-scale sequencing projects, including approximately 15,000 genomes and 123,000 exomes (Lek et al., 2016).The gnomAD set integrates data from the 1000 Genomes project as well as individuals recruited for disease-specific studies, including cancer and cardiovascular diseases. Genotype quality metrics and site quality metrics for a specific variant are available at http: //gnomad.broadinstitute.org/.gnomAD_Freq - variant allele frequency (in percent) from approximately 15,000 genomes and 123,000 exomes in the Genome Aggregation DatabasegnomAD_AMR- variant frequency (in percent) for individuals of Latino ancestrygnomAD_NFE - variant frequency (in percent) for non-Finnish individuals of European ancestrygnomAD_AFR - variant frequency (in percent) for individuals of African ancestrygnomAD_EAS - variant frequency (in percent) for individuals of East Asian ancestrygnomAD_FIN - variant frequency (in percent) for Finnish individuals of European ancestrygnomAD_Other - variant frequency (in percent) for individuals of other ancestrygnomAD_SAS - variant frequency (in percent) for individuals of South Asian ancestrygnomAD_ASJ- variant frequency (in percent) for individuals of Ashkenazi Jewish ancestrygnomAD_Hom - The number of individuals who are homozygous for the variantPROVEAN (Protein Variation Effect Analyzer) - predicts whether an amino acid substitution or indel affects the biological function of a protein using a delta alignment score from -14 to +14 (more negative=more damaging) with a predefined threshold of -2.5. If the PROVEAN score is equal to or below -2.5, the variant is predicted to have a deleterious effect. If the PROVEAN score is greater than -2.5, the variant is predicted to have a neutral effect.Note that other published in silico algorithms, including those that predict splicing impact, may be considered for variant analysis.

In silico scores used by GeneDx are precomputed and may change over time. In silico models use algorithms that predict the effect a variant may have on the protein, but they do not provide direct evidence regarding the actual impact on protein structure or function. In silico models should be interpreted with caution and only be used in combination with other available evidence to support the classification of any variant.ClinVar - Classification of variant in ClinVar database, an NCBI archive of human variants with supporting evidence of phenotypic association.REFERENCES:
1. gnomAD: Lek et al. (2016) Nature 536 (7616): 285-91 (PMID: 27535533). 2. PROVEAN: Choi et al. (2012) PLoS ONE 7 (10): e46688 (PMID: 23056405). 3. ClinVar: Landrum et al. (2014) Nucleic Acids Res. 42 (1): D980-5 (PMID: 24234437).
Report electronically signed by: Renkui Bai M.D., Ph.D., FACMG
Performed by: GeneDx
207 Perry Parkway
Gaithersburg, MD 20877

Anne Maddalena, Ph.D.,FACMG,

VERIFIED/REPORTED DATES

Procedure	Accession	Collected	Received	Verified/Reported
Ordering Physician Name	20-064-401312	00/00/0000 00:00	00/00/0000 00:00	00/00/0000 00:00
Ordering Physician Phone Number	20-064-401312	00/00/0000 00:00	00/00/0000 00:00	00/00/0000 00:00
EER Mito Disorders, mtDNA/Nuclear Genes	20-064-401312	00/00/0000 00:00	00/00/0000 00:00	00/00/0000 00:00
Mito Disorders, mtDNA and Nuclear Genes	20-064-401312	00/00/0000 00:00	00/00/0000 00:00	00/00/0000 00:00

END OF CHART

H=High, L=Low, *=Abnormal, C=Critical