Mitochondrial Disorders Panel (mtDNA and Nuclear Genes)

Patient: [redacted] DOB: [redacted] Age: 0 Sex: F **Patient Identifiers:** [redacted] **Visit Number (FIN):** [redacted]	Client: Physician:	ARUP Test Code: 3001959 Collection Date: 04/20/2023 Received in lab: 04/22/2023 Completion Date: 06/23/2023

Test Information

Test performed at GeneDx.

Patient Report

Patient's results from GeneDx continue on following page(s).

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Jonathan R. Genzen, MD, PhD, Laboratory Director

Chart continues on following page(s)

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Submitter Patient ID(s) [redacted]

Sample	Testing	Provider
Source: Blood in EDTA Date Collected: 20-APR-2023 Date Received: 25-APR-2023	Date Started: 11-MAY-2023 Date Reported: 22-JUN-2023	Account #: A.R.U.P Laboratories

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 188 Nuclear Genes

Result: Positive

Gene	Mode of Inheritance	Variant	Zygosity	Classification
PDHA1	**X-Linked**	**c.904 C>T p.(R302C)**	**Heterozygous**	**Pathogenic Variant**
SCO2	Autosomal dominant, Autosomal recessive	c.737 C>T p.(S246L)	Heterozygous	Variant of Uncertain Significance

Variant(s) of uncertain significance that do not establish a molecular diagnosis are listed in the table below.

Interpretation

This individual is heterozygous for a pathogenic variant in the PDHA1 gene, which is consistent with pyruvate dehydrogenase deficiency.

This individual is heterozygous for a variant of uncertain significance in the SCO2 gene, which does not establish a molecular diagnosis in this individual.

Recommendation(s)

- Genetic counseling is recommended to discuss the implications of these results.
- Targeted testing of the parents of this individual will help determine if the variant in the PDHA1 gene was inherited or arose de novo.
- Molecular prenatal diagnosis may be considered for the pathogenic variant.
- Please contact GeneDx to discuss testing requirements prior to submitting fetal samples.
- Targeted testing of family members for the variant in the SCO2 gene may be considered to determine if the variant is segregating with the phenotype in this family. If desired, please contact GeneDx to discuss which family members may be the most informative for testing.

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.

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PDHA1

GENE SUMMARY

The X-linked PDHA1 gene encodes the E1-alpha subunit of the pyruvate dehydrogenase (PDH) complex, which is located in the mitochondrial matrix and catalyzes the irreversible oxidative decarboxylation of pyruvate to acetyl-CoA (MIM 312170). The majority (>80%) of PDH complex deficiencies result from pathogenic variants in the PDHA1 gene, which cause a spectrum of clinical disorders that vary in age-of-onset, symptoms, and severity (PMID: 16904023, 20002461). At the severe end of the spectrum, patients may present with neonatal severe lactic acidosis leading to early death. In other cases, individuals may have hypotonia, lethargy, seizures, intellectual disability, and spasticity. Some individuals are diagnosed with Leigh syndrome, while others have a milder presentation with intermittent lactic acidosis and cerebellar ataxia, or there may be neurologic dysfunction with or without structural brain abnormalities (MIM 312170). Heterozygous females exhibit a broad range of presentations, from severely affected to unaffected.

p.(Arg302Cys) (CGT>TGT): c.904 C>T in exon 10 of the PDHA1 gene (NM_000284.3)

- Observed in multiple unrelated female patients from different ethnic backgrounds with pyruvate dehydrogenase complex (PDHc) deficiency (Quintana et al., 2010; Glushakova et al., 2011; Dahl et al. 1992) and in a male patient who was mosaic for the variant (Quintana et al., 2010); has not been observed in controls
- De novo variant with confirmed parentage in a patient with clinical features consistent with pyruvate dehydrogenase deficiency previously tested at GeneDx
- Published functional studies demonstrate that colonies harboring the R302C variant had no functional enzyme activity (Drakulic et al., 2018)
- In silico analysis supports that this missense variant has a deleterious effect on protein structure/function
- Not observed at significant frequency in large population cohorts (gnomAD)

We interpret this as a Pathogenic Variant.

SCO2

GENE SUMMARY

The SCO2 gene encodes a metallochaperone involved in the delivery of copper to Complex IV (COX) of the respiratory chain (PMID: 10749987). Pathogenic variants in the SCO2 gene are most commonly associated with an autosomal recessive form of early-onset cardioencephalopathy characterized by early mortality (PMID: 10749987). Affected individuals typically develop respiratory insufficiency, hypotonia, lactic acidosis, seizures and hypertrophic cardiomyopathy, with severe COX deficiency observed in heart and skeletal muscle (PMID: 10749987). Nearly all reported patients carry at least one copy of the common c.1541 G>A (E140K) pathogenic variant (PMID: 10749987, 16326995). Patients homozygous for E140K have a delayed onset of disease and a more prolonged survival, while patients with one copy of E140K in combination with a more deleterious pathogenic variant follow a severe clinical course, which sometimes mimicks spinal muscular atrophy type I (Werdnig-Hoffman disease) (PMID: 14994243, 16326995). Patients with bi-allelic variants in SCO2 and axonal neuropathy (Charcot Marie Tooth disease type 4) with decreased copper levels were recently described; neither have developed cardiomyopathy (PMID: 29351582). In addition, heterozygous variants in SCO2 have been reported in association with early-onset high-grade myopia (PMID: 23643385, 25525168).

p.(Ser246Leu) (TCG>TTG): c.737 C>T in exon 2 of the SCO2 gene (NM_005138.2)

- Has not been previously published as pathogenic or benign to our knowledge
- Not observed at significant frequency in large population cohorts (gnomAD)
- In silico analysis supports that this missense variant has a deleterious effect on protein structure/function

We interpret this as a Variant of Uncertain Significance.

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Additional Comments

This individual's haplogroup and a table of observed variants 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.

Additional Variants of Uncertain Significance (See Below)

Gene	Mode of Inheritance	Variant	Zygosity	Classification
COASY	Autosomal recessive	c.1183 G>A p.(A395T)	Heterozygous	Variant of Uncertain Significance

At this time, the above variants are classified as variants of uncertain significance as they do not meet criteria to be classified otherwise. This table may include single heterozygous variants of uncertain significance (VUS) in autosomal recessive genes, VUS in genes associated with dual inheritance that are unlikely to be related to the referring phenotype, VUS in candidate genes that have been suggested to be associated with autosomal recessive or dual inheritance human disease, and VUS or unclassified variants in the mitochondrial DNA observed at low levels of heteroplasmy. For variants identified in the nuclear genome, information on population data and in-silico analysis can be found in the supplemental variant information tables at the end of the report.

Genes Evaluated

AARS2, ABCB7, ACAD9, ACO2, ADCK4, AFG3L2, AGK, AIFM1, ALAS2, APOPT1, ATP5F1A, ATP5E, ATP7B, ATPAF2, AUH, BCS1L, BOLA3, C10ORF2, C12ORF65, C19ORF12, C20ORF7, C8ORF38, CARS2, CLPB, COA6, COASY, COQ2, COQ4, COQ6, COQ7, COQ8A, COQ9, COX10, COX15, COX20, COX6A1, COX6B1, CYC1, DARS2, DGUOK, DLAT, DLD, DNA2, DNAJC19, DNM1L, EARS2, ECHS1, ELAC2, ETFA, ETFB, ETFDH, ETHE1, FARS2, FASTKD2, FBXL4, FDX2, FH, FLAD1, FOXRED1, GARS, GCDH, GFER, GFM1, GFM2, GLRX5, GTPBP3, HARS2, HMGCL, HTRA2, IARS2, IBA57, ISCA2, ISCU, LAMP2, LARS, LARS2, LIAS, LIPT1, LRPPRC, LYRM4, LYRM7, MARS2, MFF, MFN2, MGME1, MICU1, MPC1, MPV17, MRPL3, MRPL44, MRPS16, MRPS22, 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, NDUFA12, NDUFA2, NDUFA9, NDUFAF1, NDUFAF2, NDUFAF3, NDUFAF4, NDUFB11, NDUFB3, NDUFS1, NDUFS2, NDUFS3, NDUFS4, NDUFS6, NDUFS7, NDUFS8, NDUFV1, NDUFV2, NFU1, NR2F1, NUBPL, OPA1, OPA3, OTC, PARS2, PC, PCCA, PCCB, PDHA1, PDHB, PDHX, PDP1, PDSS1, PDSS2, PET100, PNPT1, POLG, POLG2, PRKAG2, PUS1, QARS, RARS1, 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, TIMM8A, TK2, TMEM126A, TMEM126B, TMEM70, TPK1, TRIT1, TRMT10C, TRMU, TRNT1, TSFM, TTC19, TUFM, TWNK, TYMP, UQCC2, UQCRB, UQCRC2, VARS2, WDR45, WFS1, YARS2

Methods

Genomic DNA was extracted directly from the submitted specimen or, if applicable, from cultured fibroblasts. For the nuclear genome, 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. Alternative sequencing or copy number detection methods were used to analyze or confirm 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 amplified and sequenced using next-generation sequencing. DNA sequence was assembled and analyzed in comparison with the revised Cambridge Reference Sequence (rCRS GeneBank number NC_012920) and the reported variants listed in the MITOMAP database (http://www.mitomap.org). Next generation sequencing may not detect large-scale mtDNA deletions present at 5% heteroplasmy or lower or mtDNA point variants present at 1.5% heteroplasmy or lower. Alternative sequencing or other detection methods may be used to analyze or confirm mtDNA variants. Reportable variants in both the nuclear and mitochondrial genome 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. Available evidence for variant classification may change over time and variant(s) in nuclear genes may be reclassified according to the ACMG/AMP Standards and Guidelines (PMID: 25741868), while the reported variant(s) in mtDNA may be reclassified according to our mitochondrial variant classification guidelines aligned with the ACMG/AMP Standards and Guidelines which may lead to issuing a revised report. If included in this test, the

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following gene specific information applies. Gene specific exclusions for exon-level deletion/duplication testing for this panel are: SCO2 and SDHA, no copy number testing; COX6A1, GTPBP3, NDUFAF4, NDUFB3, NR2F1, SLC25A26, TAZ, and TYMP genes, only whole gene deletions or duplications may be detected.

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. Unless indicated in the test methods, the following may not be detected: mosaic variants, pure heterodisomy, balanced chromosomal aberrations, nucleotide repeat expansion/contraction, abnormal DNA methylation, and variants located in regions not evaluated by this test or that cannot be detected by the methodology used. Regions of certain genes have technical limitations and inherent sequence properties that yield suboptimal data, potentially impairing accuracy of the results (for example: repetitive DNA, homology or pseudogene regions, high GC content). Unless otherwise indicated, sequence analysis cannot reliably detect deletions of 20bp to 500bp in size, or insertions of 10bp to 500bp in size; deletions/insertions of less than 500 bp cannot be reliably detected by exon-level array. Rarely, incidental findings of large chromosomal rearrangements outside the gene(s) of interest may be identified. The zygosity reported reflects the presumed germline status of this individual, but may be limited by depth of read coverage and/or parental genotype data at the time of reporting. The chance of a false positive or false negative result due to laboratory errors incurred during any phase of testing cannot be completely excluded. 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. False negative results may also occur in the setting of bone marrow transplantation, recent blood transfusion, or suboptimal DNA quality. DNA extracted using external methodologies may negatively affect test performance. In individuals with active or chronic hematologic neoplasms or conditions, there is a possibility that testing may detect an acquired somatic variant, leading to a false positive result. The clinical sensitivity of this test depends in part on the patient's clinical phenotype, and is expected to be highest for individuals with clearly defined disease and/or family history of disease. Interpretations are made with the assumption that any clinical information provided, including family relationships, is 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);Richards et al. (2015) Genetics In Medicine: 17 (5):405-24 (PMID: 25741868);Jakesch M et al. (2000) Hum Mol Genet. 9 (5):795-801 (PMID: 10749987);Tarnopolsky MA et al. (2004) American journal of medical genetics. Part A. 125A (3):310-4 (PMID: 14994243);Bohm M et al. (2006) Pediatric research. 59 (1):21-6 (PMID: 16326995);Tran-Viet KN et al. (2013) American journal of human genetics. 92 (5):820-6 (PMID: 23643385);Jiang D et al. (2015) Investigative ophthalmology & visual science. 56 (1):339-45 (PMID: 25525168);Rebelo AP et al. (2018) Brain. 141 (3):662-672 (PMID: 29351582);Brown RM et al. (2006) Dev Med Child Neurol. 48 (9):756-60 (PMID: 16904023);Quintana E et al. (2010) Clin Genet. 77 (5):474-82 (PMID: 20002461); Drakulic et al. (2018) Cell. Mol. Life Sci. 75 (16):3009-3026 (PMID: 29445841); Dahl et al. (1992) J. Inherit. Metab. Dis. 15 (6):835-47 (PMID: 1293379); Glushakova et al. (2011) Molecular Genetics And Metabolism 104 (3):255-60 (PMID: 21846590); Quintana et al. (2010) Clin. Genet. 77 (5):474-82 (PMID: 20002461); Stenson et al. (2014) Hum. Genet. 133 (1):1-9 (PMID: 24077912)

Report Electronically Signed By

Hong Cui PhD, FACMG
Director, Clinical Genetics

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Variant Table

Gene: Coding DNA	PDHA1: c.904 C>T	COASY: c.1183 G>A	SCO2: c.737 C>T
Variant (Protein)	p.(Arg302Cys) ((R302C))	p.(Ala395Thr) ((A395T))	p.(Ser246Leu) ((S246L))
Classification	Pathogenic Variant	Variant of Uncertain Significance	Variant of Uncertain Significance
Zygosity	Heterozygous	Heterozygous	Heterozygous
Chr: Position	X: 19377038	17: 40716862	22: 50962104
dbSNP	rs137853252	rs139263701	rs139003628
gnomAD_Freq		0.0006	0.0000
gnomAD_AMR		0.00012749	0.00000000
gnomAD_NFE		0.00110818	0.00001760
gnomAD_AFR		0.00024372	0.00000000
gnomAD_EAS		0.00000000	0.00000000
gnomAD_FIN		0.00000000	0.00000000
gnomAD_Other		0.00015480	0.00000000
gnomAD_SAS		0.00000000	0.00000000
gnomAD_ASJ		0.00000000	0.00000000
gnomAD_Hom		0	0
Provean	-7.77 (D)	-1.46 (N)	-2.98 (D)
ClinVar	Pathogenic	Uncertain 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 at the classification. This information is subject to change 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 were available at time of analysis.

- dbSNP - NCBI repository for single base nucleotide substitutions and short deletion and insertion polymorphisms https://www.ncbi.nlm.nih.gov/snp/
- The 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, including individuals recruited for disease-specific studies such as cancer and cardiovascular diseases. (PMID 32461654).
- gnomAD_Freq - variant allele frequency (in percent) from approximately 15,000 genomes and 123,000 exomes in the Genome Aggregation Database. Select ancestries include: gnomAD_AMR (Admixed American/Latino); gnomAD_AFR (African); gnomAD_EAS (East Asian); gnomAD_FIN (Finnish of European ancestry); gnomAD_NFE (non-Finnish of European ancestry); gnomAD_SAS (South Asian); gnomAD_ASJ (Ashkenazi Jewish). gnomAD_Hom - number of individuals homozygous for the variant.
- gnomAD_AMR- variant frequency (in percent) for individuals of Latino ancestry
- PROVEAN (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 (≤ or = -2.5, predicted deleterious; >-2.5, predicted neutral).
- Other published in silico algorithms, including those that predict splicing impact, may be considered for variant analysis. In silico scores may change. In silico models use algorithms that predict the effect a variant may have on the protein. Thus, predictions should be interpreted with caution and only be used in combination with other available evidence to support the classification of any variant (PMID 23056405).
- ClinVar - Classification of variant in ClinVar database, an NCBI archive of human variants with supporting evidence of phenotypic association. https://www.ncbi.nlm.nih.gov/clinvar/ (PMID 26582918).




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MTDNA BENIGN/LIKELY BENIGN VARIANTS

Functional Location	Variant Change	Amino Acid Change	Frequency (Gen. Pop)
MT-DLOOP	m.73 A>G	non-coding	37469/49135
MT-DLOOP	m.119 T>C	non-coding	75/50175
MT-DLOOP	m.189 A>G	non-coding	2719/49135
MT-DLOOP	m.195 T>C	non-coding	9537/49135
MT-DLOOP	m.204 T>C	non-coding	3142/49135
MT-DLOOP	m.207 G>A	non-coding	2295/49135
MT-DLOOP	m.227 A>G	non-coding	165/50175
MT-DLOOP	m.263 A>G	non-coding	46799/49135
MT-DLOOP	m.503 A>G	non-coding	19/51673
MT-RNR1	m.709 G>A	rRNA	6395/49135
MT-RNR1	m.750 A>G	rRNA	48276/49135
MT-RNR1	m.1243 T>C	rRNA	776/49135
MT-RNR1	m.1438 A>G	rRNA	46721/49135
MT-RNR2	m.2706 A>G	rRNA	38981/49135
MT-RNR2	m.3106del	In Frame	common
MT-ND1	m.3505 A>G	Missense	701/49135
MT-ND2	m.4769 A>G	Synonymous	55528/56895
MT-ND2	m.5046 G>A	Missense	868/49135
MT-ND2	m.5460 G>A	Missense	3179/49135
MT-CO1	m.7028 C>T	Synonymous	39824/49135
MT-CO2	m.7864 C>T	Synonymous	211/50175
MT-CO2	m.8251 G>A	Synonymous	2823/49135
MT-ATP6	m.8860 A>G	Missense	48479/49135
MT-ATP6	m.8994 G>A	Synonymous	808/49135
MT-ND4	m.11204 T>C	Missense	147/49135
MT-ND4	m.11674 C>T	Synonymous	561/49135
MT-ND4	m.11719 G>A	Synonymous	38205/49135
MT-ND4	m.11914 G>A	Synonymous	5367/49135

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MT-ND4	m.11947 A>G	Synonymous	533/49135
MT-ND5	m.12414 T>C	Synonymous	670/49135
MT-ND5	m.12648 A>G	Synonymous	11/50175
MT-ND5	m.12705 C>T	Synonymous	20517/49135
MT-ND5	m.14148 A>G	Extended Protein	315/49135
MT-CYB	m.14766 C>T	Missense	37907/49135
MT-CYB	m.15326 A>G	Missense	48493/49135
MT-CYB	m.15884 G>C	Missense	535/49135
MT-DLOOP	m.16223 C>T	non-coding	19551/49135
MT-DLOOP	m.16292 C>T	non-coding	1218/49135
MT-DLOOP	m.16519 T>C	non-coding	30962/49135
Haplogroup (HG): W1c1			

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