15 Crawford St., STE 100
Needham, MA 02494
(p) 626-350-0537
(f) 626-454-1667
Lab Director: Arash Radfar M.D.
CLIA: 22D0957540

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Patient Information:
13186, donor
DOB: [redacted]
Sex: M
MR#:
Patient#: FT-PT8878938

Partner Information:
Not Tested

Physician:
Kuan, James
ATTN: SSB Genetics, Dept
Seattle Sperm Bank
4915 25th Avenue NE, Ste 204W
Seattle, WA 98105
Phone: (206) 588-1484

Laboratory:
Fulgent Therapeutics LLC
CAP#: 8042697
CLIA#: 05D2043189
Laboratory Director:
Lawrence M. Weiss, MD
Report Date: **Nov 08,2024**

Accession:
FT-6945987
Test#: FT-TS14986034
Specimen Type: Blood (EDTA)
Collected: Oct 21,2024

Accession:
N/A

FINAL RESULTS

4 Carrier for genetic conditions in **multiple** genes.
Genetic counseling is recommended.

TEST PERFORMED

Beacon Preconception Carrier Screening - 515 Genes (without X-linked Disorders)
(515 Gene Panel; gene sequencing with deletion and duplication analysis)

Condition and Gene	Inheritance	13186, donor	Partner
Schindler disease types 1 and 3 *NAGA*	AR	⊕ Carrier c.973G>T (p.Glu325*)	N/A
Retinitis pigmentosa 28 *FAM161A*	AR	⊕ Carrier c.1837G>T (p.Glu613*)	N/A
Osteogenesis imperfecta, type XIII *BMP1*	AR	⊕ Carrier c.584dup (p.Gln197Profs*10)	N/A
Smith-Lemli-Opitz syndrome *DHCR7*	AR	⊕ Carrier c.964-1G>C (p.?)	N/A

INTERPRETATION:

Notes and Recommendations:

- Based on these results, this individual is positive for carrier mutations in 4 genes. Carrier screening for the reproductive partner is recommended to accurately assess the risk for any autosomal recessive conditions. A negative result reduces, but does not eliminate, the chance to be a carrier for any condition included in this screen. Please see the supplemental table for details.
- Testing for copy number changes in the SMN1 gene was performed to screen for the carrier status of Spinal Muscular Atrophy. The results for this individual are within the normal range for non-carriers. See Limitations section for more information.
- This carrier screening test does not screen for all possible genetic conditions, nor for all possible mutations in every gene tested. This report does not include variants of uncertain significance; only variants classified as pathogenic or likely pathogenic at the time of testing, and considered relevant for reproductive carrier screening, are reported. Please see the gene specific notes for details. Please note that the classification of variants can change over time.
- Patients may wish to discuss any carrier results with blood relatives, as there is an increased chance that they are also carriers. These results should be interpreted in the context of this individual's clinical findings, biochemical profile, and family history.
- X-linked genes are not routinely analyzed for male carrier screening tests. Gene specific notes and limitations may be present. See below.
- Genetic counseling is recommended. Available genetic counselors and additional resources can be found at the National Society of Genetic Counselors (NSGC; https://www.nsgc.org)

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SCHINDLER DISEASE TYPES 1 AND 3

Patient	13186, donor	Partner
Result	⊕ Carrier	N/A
Variant Details	***NAGA*** (NM_000262.3) c.973G>T (p.Glu325*)	N/A

What is Schindler disease types 1 and 3?

Schindler disease types 1 and 3 are similar disorders that affect the nervous system but with differing onset and severity. Individuals with type 1 experience symptoms beginning earlier in life and have a more severe expression of symptoms than those with type 3. People with this disorder experience developmental, neurologic, and psychiatric manifestations. Common manifestations include a loss of developmental milestones, low or weak muscle tone, seizures, and loss of or impaired vision. Individuals with this condition over time may lose awareness of their surroundings and become unresponsive.

What is my risk of having an affected child?

Schindler disease types 1 and 3 are inherited in an autosomal recessive manner. If the patient and the partner are both carriers, the risk for an affected child is 1 in 4 (25%).

What kind of medical management is available?

There are no approved treatments for Schindler disease types 1 and 3, although this is an area of active research and clinical trials. Lifelong monitoring by a medical geneticist or other specialist is recommended.

What mutation was detected?

The detected heterozygous variant was NM_000262.3:c.973G>T (p.Glu325*). This variant is predicted to introduce a premature stop codon at least 50 nucleotides upstream of the canonical donor splice site of the penultimate exon and to result in the loss of function of the protein product due to nonsense-mediated mRNA decay (PubMed: 25741868, 30192042, 27618451, 11532962, 18066079). There's sufficient evidence that loss of function in this gene is a known disease mechanism for Schindler disease (PubMed: 7707696, 8782044, 29100083). The laboratory classifies this variant as likely pathogenic.

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RETINITIS PIGMENTOSA 28

Patient	13186, donor	Partner
Result	⊕ Carrier	N/A
Variant Details	***FAM161A*** (NM_001201543.2) c.1837G>T (p.Glu613*)	N/A

What is Retinitis pigmentosa 28?

Retinitis pigmentosa, also known as RP, refers to a group of inherited diseases causing retinal degeneration. People with RP experience a gradual decline in their vision, because photoreceptor cells in the retina degenerate. Night blindness is one of the earliest and most frequent symptoms of RP, and most people with RP are legally blind by age 40. Symptoms of RP are most often recognized in children, adolescents and young adults, with progression of the disease continuing throughout the individual's life. The pattern and degree of visual loss are variable depending on the various types of genes that cause RP.

What is my risk of having an affected child?

Retinitis pigmentosa 28 is inherited in an autosomal recessive manner. If the patient and the partner are both carriers, the risk for an affected child is 1 in 4 (25%).

What kind of medical management is available?

There is no known cure for retinitis pigmentosa. However, there are few treatment options such as light avoidance and/or the use of low-vision aids to slow down the progression of RP. A number of services and devices are available to help people with vision loss carry out daily activities and maintain their independence.

What mutation was detected?

The detected heterozygous variant was NM_001201543.2:c.1837G>T (p.Glu613*). This variant is predicted to introduce a premature stop codon at least 50 nucleotides upstream of the canonical donor splice site of the penultimate exon and to result in the loss of function of the protein product due to nonsense-mediated mRNA decay (PubMed: 25741868, 30192042, 27618451, 11532962, 18066079). There's sufficient evidence that loss of function in this gene is a known disease mechanism for Retinitis pigmentosa (PubMed: 20705278, 27874104, 30902645). The laboratory classifies this variant as likely pathogenic.

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OSTEOGENESIS IMPERFECTA, TYPE XIII

Patient	13186, donor	Partner
Result	⊕ Carrier	N/A
Variant Details	***BMP1*** (NM_006129.5) c.584dup (p.Gln197Profs*10)	N/A

What is Osteogenesis imperfecta, type XIII?

Osteogenesis imperfecta (OI), type XIII is an inherited connective tissue disorder characterized by bone fragility and recurrent fractures. The term "osteogenesis imperfecta" means imperfect bone formation. Affected individuals can have up to 15 fractures a year, with their first fractures occurring before the age of 3. Additional features include developmental delays, light blue sclera, scoliosis, and osteoporosis. Hearing loss has not been associated with this subtype of OI.

What is my risk of having an affected child?

Osteogenesis imperfecta, type XIII is inherited in an autosomal recessive manner. If the patient and the partner are both carriers, the risk for an affected child is 1 in 4 (25%).

What kind of medical management is available?

Treatment for osteogenesis imperfecta, type XIII is aimed at preventing symptoms, maintaining individual mobility, and strengthening bone and muscle. Individuals with osteogenesis imperfecta, type XIII should consult with their physicians and physical therapists to determine a safe and appropriate exercise program. Treatment with bisphosphonate can increase bone mineral density and decrease fractures. Surgery may be necessary for some individuals with severe bone malformation.

What mutation was detected?

The detected heterozygous variant was NM_006129.5:c.584dup (p.Gln197Profs*10). This variant is predicted to introduce a premature stop codon at least 50 nucleotides upstream of the canonical donor splice site of the penultimate exon and to result in the loss of function of the protein product due to nonsense-mediated mRNA decay (PubMed: 25741868, 30192042, 27618451, 11532962, 18066079). There's sufficient evidence that loss of function in this gene is a known disease mechanism for Osteogenesis imperfecta (PubMed: 33452237, 33818922, 35703132).This variant was reported in the compound heterozygous state in one individual with osteogenesis imperfecta XIII (PubMed: 35703132) The laboratory classifies this variant as pathogenic.

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SMITH-LEMLI-OPITZ SYNDROME

Patient	13186, donor	Partner
Result	⊕ Carrier	N/A
Variant Details	***DHCR7*** (NM_001360.3) c.964-1G>C (p.?)	N/A

What is Smith-Lemli-Opitz syndrome?

Smith-Lemli-Opitz syndrome (SLOS) is an impairment of the body's ability to make cholesterol. As a result, affected infants typically have cleft palate, hypotonia (low muscle syndrome), and failure to thrive. Other characteristics can include ambiguous genitalia, cardiac defects, kidney problems, dysmorphic facial features, microcephaly (abnormally small head), syndactyly (webbed or conjoined fingers or toes), and intellectual disability. The clinical features and severity of symptoms vary from person to person.

What is my risk of having an affected child?

SLOS is inherited in an autosomal recessive manner. This means that when both parents are carriers for the condition, there is a 25% (1 in 4) risk of having an affected child.

What kind of medical management is available?

There is currently no cure for SLOS, but symptoms can be addressed. Dietary cholesterol supplementation may improve clinical symptoms. Additionally, early intervention with physical, speech, and occupational therapy may improve developmental issues.

What mutation was detected?

The detected heterozygous variant was NM_001360.3:c.964-1G>C (p.?). This intronic variant, c.964-1G>C, alters the highly conserved splice acceptor site for exon 9 of this transcript and is predicted by all four splice site prediction tools queried to abolish the canonical splice acceptor site. A functional study in patient-derived mRNA demonstrated that this variant disrupts the canonical acceptor site, resulting in the use of a cryptic splice acceptor site, the insertion of 134 bp, a frameshift, and a premature stop codon (PubMed: 9653161). There's sufficient evidence that loss of function in this gene is a known disease mechanism for Smith-Lemli-Opitz syndrome (PubMed: 11111101, 16618793, 22226660). This variant accounts for a third of all mutant alleles in the *DHCR7* gene in individuals with Smith-Lemli-Opitz syndrome (SLOS) (PubMed:9653161, 10995508, 12949967, 17965227, 29455191, 28805615, 22226660, 28369852, 12818773, 12914579, 22929031). This variant is classified as "Pathogenic" or "Likely Pathogenic" in ClinVar, with multiple submitters in agreement (ClinVar: 93725). The laboratory classifies this variant as pathogenic.

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GENES TESTED:

Beacon Preconception Carrier Screening - 515 Genes (without X-linked Disorders) - 515 Genes

This analysis was run using the Beacon Preconception Carrier Screening - 515 Genes (without X-linked Disorders) gene list. 515 genes were tested with 99.5% of targets sequenced at >20x coverage. For more gene-specific information and assistance with residual risk calculation, see the SUPPLEMENTAL TABLE.

AAAS, ABCA12, ABCA3, ABCA4, ABCB11, ABCB4, ABCC2, ABCC8, ACAD9, ACADM, ACADVL, ACAT1, ACOX1, ACSF3, ADA, ADAMTS2, ADAMTSL4, ADGRG1, ADGRV1, AGA, AGL, AGPS, AGXT, AHI1, AIPL1, AIRE, ALDH3A2, ALDH7A1, ALDOB, ALG1, ALG6, ALMS1, ALPL, AMN, AMT, ANO10, AP1S1, AQP2, ARG1, ARL6, ARSA, ARSB, ASL, ASNS, ASPA, ASS1, ATM, ATP6V1B1, ATP7B, ATP8B1, BBS1, BBS10, BBS12, BBS2, BBS4, BBS5, BBS7, BBS9, BCKDHA, BCKDHB, BCS1L, BLM, BLOC1S3, BLOC1S6, BMP1, BRIP1, BSND, CAD, CANT1, CAPN3, CASQ2, CBS, CC2D1A, CC2D2A, CCDC103, CCDC39, CCDC88C, CD3D, CD3E, CD40, CD59, CDH23, CEP152, CEP290, CERKL, CFTR, CHAT, CHRNE, CHRNG, CIITA, CLCN1, CLN3, CLN5, CLN6, CLN8, CLRN1, CNGB3, COL11A2, COL17A1, COL27A1, COL4A3, COL4A4, COL7A1, COX15, CPS1, CPT1A, CPT2, CRB1, CRTAP, CRYL1, CTNS, CTSA, CTSC, CTSD, CTSK, CYBA, CYP11A1, CYP11B1, CYP11B2, CYP17A1, CYP19A1, CYP1B1, CYP21A2, CYP27A1, CYP27B1, CYP7B1, DBT, DCAF17, DCLRE1C, DDX11, DGAT1, DGUOK, DHCR7, DHDDS, DLD, DLL3, DNAH11, DNAH5, DNAI1, DNAI2, DNMT3B, DOK7, DUOX2, DYNC2H1, DYSF, EIF2AK3, EIF2B1, EIF2B2, EIF2B3, EIF2B4, EIF2B5, ELP1, EPG5, ERCC2, ERCC6, ERCC8, ESCO2, ETFA, ETFB, ETFDH, ETHE1, EVC, EVC2, EXOSC3, EYS, FAH, FAM161A, FANCA, FANCC, FANCD2, FANCE, FANCG, FANCI, FANCL, FBP1, FBXO7, FH, FKBP10, FKRP, FKTN, FMO3, FOXN1, FOXRED1, FRAS1, FREM2, FUCA1, G6PC, G6PC3, GAA, GALC, GALE, GALK1, GALNS, GALNT3, GALT, GAMT, GATM, GBA, GBE1, GCDH, GCH1, GDF5, GFM1, GHR, GJB2, GJB6, GLB1, GLDC, GLE1, GNE, GNPAT, GNPTAB, GNPTG, GNS, GORAB, GRHPR, GRIP1, GSS, GUCY2D, GUSB, HADH, HADHA, HADHB, HAMP, HAX1, HBA1, HBA2, HBB, HEXA, HEXB, HGSNAT, HJV, HLCS, HMGCL, HMOX1, HOGA1, HPD, HPS1, HPS3, HPS4, HPS5, HPS6, HSD17B3, HSD17B4, HSD3B2, HYAL1, HYLS1, IDUA, IGHMBP2, IKBKB, IL7R, INVS, ITGA6, ITGB3, ITGB4, IVD, JAK3, KCNJ1, KCNJ11, LAMA2, LAMA3, LAMB3, LAMC2, LARGE1, LCA5, LDLR, LDLRAP1, LHX3, LIFR, LIG4, LIPA, LMBRD1, LOXHD1, LPL, LRAT, LRP2, LRPPRC, LYST, MAK, MAN2B1, MANBA, MCEE, MCOLN1, MCPH1, MECR, MED17, MESP2, MFSD8, MKKS, MKS1, MLC1, MLYCD, MMAA, MMAB, MMACHC, MMADHC, MOCS1, MOCS2, MPI, MPL, MPV17, MRE11, MTHFR, MTR, MTRR, MTTP, MUSK, MUT, MVK, MYO15A, MYO7A, NAGA, NAGLU, NAGS, NBN, NCF2, NDRG1, NDUFAF2, NDUFAF5, NDUFS4, NDUFS6, NDUFS7, NDUFV1, NEB, NEU1, NGLY1, NPC1, NPC2, NPHP1, NPHS1, NPHS2, NR2E3, NSMCE3, NTRK1, OAT, OCA2, OPA3, OSTM1, OTOA, OTOF, P3H1, PAH, PANK2, PC, PCBD1, PCCA, PCCB, PCDH15, PCNT, PDHB, PEPD, PET100, PEX1, PEX10, PEX12, PEX13, PEX16, PEX2, PEX26, PEX5, PEX6, PEX7, PFKM, PGM3, PHGDH, PHKB, PHKG2, PHYH, PIGN, PJVK, PKHD1, PLA2G6, PLEKHG5, PLOD1, PMM2, PNPO, POLG, POLH, POMGNT1, POMT1, POMT2, POR, POU1F1, PPT1, PRCD, PRDM5, PRF1, PROP1, PSAP, PTPRC, PTS, PUS1, PYGM, QDPR, RAB23, RAG1, RAG2, RAPSN, RARS2, RDH12, RLBP1, RMRP, RNASEH2A, RNASEH2B, RNASEH2C, RPE65, RPGRIP1L, RTEL1, RXYLT1, RYR1, SACS, SAMD9, SAMHD1, SCO2, SEC23B, SEPSECS, SGCA, SGCB, SGCD, SGCG, SGSH, SKIV2L, SLC12A1, SLC12A3, SLC12A6, SLC17A5, SLC19A2, SLC19A3, SLC1A4, SLC22A5, SLC25A13, SLC25A15, SLC25A20, SLC26A2, SLC26A3, SLC26A4, SLC27A4, SLC35A3, SLC37A4, SLC38A8, SLC39A4, SLC45A2, SLC4A11, SLC5A5, SLC7A7, SMARCAL1, SMN1, SMPD1, SNAP29, SPG11, SPR, SRD5A2, ST3GAL5, STAR, STX11, STXBP2, SUMF1, SUOX, SURF1, SYNE4, TANGO2, TAT, TBCD, TBCE, TCIRG1, TCN2, TECPR2, TERT, TF, TFR2, TG, TGM1, TH, TK2, TMC1, TMEM216, TMEM67, TMPRSS3, TPO, TPP1, TREX1, TRIM32, TRIM37, TRMU, TSEN54, TSFM, TSHB, TSHR, TTC37, TTPA, TULP1, TYMP, TYR, TYRP1, UBR1, UNC13D, USH1C, USH2A, VDR, VLDLR, VPS11, VPS13A, VPS13B, VPS45, VPS53, VRK1, VSX2, WISP3, WNT10A, WRN, XPA, XPC, ZBTB24, ZFYVE26, ZNF469

METHODS:

Genomic DNA was isolated from the submitted specimen indicated above (if cellular material was submitted). DNA was barcoded, and enriched for the coding exons of targeted genes using hybrid capture technology. Prepared DNA libraries were then sequenced using a Next Generation Sequencing technology. Following alignment to the human genome reference sequence (assembly GRCh37), variants were detected in regions of at least 10x coverage. For this specimen, 99.54% and 99.49% of coding regions and splicing junctions of genes listed had been sequenced with coverage of at least 10x and 20x, respectively, by NGS or by Sanger sequencing. The remaining regions did not have 10x coverage, and were not evaluated. Variants were interpreted manually using locus specific databases, literature searches, and other molecular biological principles. To minimize false positive results, any variants that do not meet internal quality standards are confirmed by Sanger sequencing. Variants classified as pathogenic, likely pathogenic, or risk allele which are located in the coding regions and nearby intronic regions (+/- 20bp) of the genes listed above are reported. Variants outside these intervals may be reported but are typically not guaranteed. When a single pathogenic or likely pathogenic variant is identified in a clinically relevant gene with autosomal recessive inheritance, the laboratory will attempt to ensure 100% coverage of coding sequences either through NGS or Sanger sequencing technologies ("fill-in"). All genes listed were evaluated for large deletions and/or duplications. However, single exon deletions or duplications will not be detected in this assay, nor will copy number alterations in regions of genes with significant pseudogenes. Putative deletions or duplications are analyzed using Fulgent Germline proprietary pipeline for this specimen. Bioinformatics: The Fulgent Germline v2019.2 pipeline was used to analyze this specimen.

LIMITATIONS:

General Limitations

These test results and variant interpretation are based on the proper identification of the submitted specimen, accuracy of any stated familial relationships, and use of the correct human reference sequences at the queried loci. In very rare instances, errors may result due to mix-up or co-mingling of specimens. Positive results do not imply that there are no other contributors, genetic or

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otherwise, to future pregnancies, and negative results do not rule out the genetic risk to a pregnancy. Official gene names change over time. Fulgent uses the most up to date gene names based on HUGO Gene Nomenclature Committee (https://www.genenames.org) recommendations. If the gene name on report does not match that of ordered gene, please contact the laboratory and details can be provided. Result interpretation is based on the available clinical and family history information for this individual, collected published information, and Alamut annotation available at the time of reporting. This assay is not designed or validated for the detection of low-level mosaicism or somatic mutations. This assay will not detect certain types of genomic aberrations such as translocations, inversions, or repeat expansions other than specified genes. DNA alterations in regulatory regions or deep intronic regions (greater than 20bp from an exon) may not be detected by this test. Unless otherwise indicated, no additional assays have been performed to evaluate genetic changes in this specimen. There are technical limitations on the ability of DNA sequencing to detect small insertions and deletions. Our laboratory uses a sensitive detection algorithm, however these types of alterations are not detected as reliably as single nucleotide variants. Rarely, due to systematic chemical, computational, or human error, DNA variants may be missed. Although next generation sequencing technologies and our bioinformatics analysis significantly reduce the confounding contribution of pseudogene sequences or other highly-homologous sequences, sometimes these may still interfere with the technical ability of the assay to identify pathogenic alterations in both sequencing and deletion/duplication analyses. Deletion/duplication analysis can identify alterations of genomic regions which include one whole gene (buccal swab specimens and whole blood specimens) and are two or more contiguous exons in size (whole blood specimens only); single exon deletions or duplications may occasionally be identified, but are not routinely detected by this test. When novel DNA duplications are identified, it is not possible to discern the genomic location or orientation of the duplicated segment, hence the effect of the duplication cannot be predicted. Where deletions are detected, it is not always possible to determine whether the predicted product will remain in-frame or not. Unless otherwise indicated, deletion/duplication analysis has not been performed in regions that have been sequenced by Sanger.

Gene Specific Notes and Limitations

ALG1: Due to the interference by highly homologous regions, our current testing method has less sensitivity to detect variants in exons 6-13 of the ALG1 gene (NM_019109.4). *CEP290:* Copy number analysis for exons 8-13 and exons 39-42 may have reduced sensitivity in the CEP290 gene. Confirmation of these exons are limited to individuals with a positive personal history of CEP290-related conditions and/or individuals carrying a pathogenic/likely pathogenic sequence variant. *CFTR:* Analysis of the intron 8 polymorphic region (e.g. IVS8-5T allele) is only performed if the p.Arg117His (R117H) mutation is detected. Single exon deletion/duplication analysis is limited to deletions of previously reported exons: 1, 2, 3, 11, 19, 20, 21. Analysis of the intron 8 polymorphic region (e.g. IVS8-5T allele) is only performed if the p.Arg117His (R117H) mutation is detected. Single exon deletion/duplication analysis is limited to deletions of previously reported exons: 1, 2, 3, 11, 19, 20, 21. CFTR variants primarily associated with CFTR-related isolated congenital bilateral absence of the vas deferens and CFTR-related pancreatitis are not included in this analysis. CFTR variants with insufficient evidence of being cystic fibrosis mutations will not be reported either. *CRYL1:* As mutations in the CRYL1 gene are not known to be associated with any clinical condition, sequence variants in this gene are not analyzed. However, to increase copy number detection sensitivity for large deletions including this gene and a neighboring on gene on the panel (GJB6, also known as connexin 30), this gene was evaluated for copy number variation. *CYP11B1:* The current testing method is not able to reliably detect certain pathogenic variants in this gene due to the interference by highly homologous regions. This analysis is not designed to detect or rule-out copy-neutral chimeric CYP11B1/CYP11B2 gene. *CYP11B2:* The current testing method is not able to reliably detect certain pathogenic variants in this gene due to the interference by highly homologous regions. This analysis is not designed to detect or rule-out copy-neutral chimeric CYP11B1/CYP11B2 gene. *CYP21A2:* Significant pseudogene interference and/or reciprocal exchanges between the CYP21A2 gene and its pseudogene, CYP21A1P, have been known to occur and may impact results. As such, the relevance of variants reported in this gene must be interpreted clinically in the context of the clinical findings, biochemical profile, and family history of each patient. LR-PCR is not routinely ordered for NM_000500.9:c.955C>T (p.Gln319Ter). Individuals with c.955C>T (p.Gln319Ter) will be reported as a Possible Carrier indicating that the precise nature of the variant has not been determined by LR-PCR and that the variant may occur in the CYP21A2 wild-type gene or in the CYP21A1P pseudogene. The confirmation test is recommended if the second reproductive partner is tested positive for variants associated with classic CAH. *DDX11:* Due to the interference by highly homologous regions, our current testing method has less sensitivity to detect variants in the DDX11 gene. *DUOX2:* The current testing method is not able to reliably detect variants in exons 6-8 of the DUOX2 gene (NM_014080.5) due to significant interference by the highly homologous gene, DUOX1. *FANCD2:* Due to pseudogene interference, copy-number-variants within exon 14-17 of the FANCD2 gene (NM _033084.4) are not evaluated and detection of single-nucleotide variants and small insertions/deletions in this region is not guaranteed. *GALT:* In general, the D2 "Duarte" allele is not reported if detected, but can be reported upon request. While this allele can cause positive newborn screening results, it is not known to cause clinical symptoms in any state. See GeneReviews for more information: https://www.ncbi.nlm.nih.gov/books/NBK1518/ *GBA:* Significant pseudogene interference and/or reciprocal exchanges between the GBA gene and its pseudogene, GBAP1, have been known to occur and may impact results. As such, the relevance of variants reported in this gene must be interpreted clinically in the context of this individual's clinical findings, biochemical profile, and family history. The current testing method cannot detect copy-neutral rearrangements between the pseudogene and the functional gene, which have been reported in very rare cases of Gaucher disease (PubMed: 21704274). *HBA1:* Significant interference

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Lab Director: Arash Radfar M.D.
CLIA: 22D0957540

from highly homologous regions in exons 1-2 of the HBA1 gene has been recognized to occur, potentially impeding the assay's technical capability to detect pathogenic alterations during sequencing analyses. *HBA2:* Significant interference from highly homologous regions in exons 1-2 of the HBA2 gene has been recognized to occur, potentially impeding the assay's technical capability to detect pathogenic alterations during sequencing analyses. *HSD17B4:* Copy number analysis for exons 4-6 may have reduced sensitivity in the HSD17B4 gene. Confirmation of these exons are limited to individuals with a positive personal history of D-bifunctional protein deficiency and Perrault syndrome and/or individuals carrying a pathogenic/likely pathogenic sequence variant. *LMBRD1:* Copy number analysis for exons 9-12 may have reduced sensitivity in the LMBRD1 gene. Confirmation of these exons are limited to individuals with a positive personal history of combined methylmalonic aciduria and homocystinuria and/or individuals carrying a pathogenic/likely pathogenic sequence variant. *MTHFR:* As recommended by ACMG, the two common polymorphisms in the MTHFR gene - c.1286A>C (p.Glu429Ala, also known as c.1298A>C) and c.665C>T (p.Ala222Val, also known as c.677C>T) - are not reported in this test due to lack of sufficient clinical utility to merit testing (PubMed: 23288205). *NEB:* This gene contains a 32-kb triplicate region (exons 82-105) which is not amenable to sequencing and deletion/duplication analysis. *NPHS2:* If detected, the variant NM_014625.3:c.686G>A (p.Arg229Gln) will not be reported as this variant is not significantly associated with disease when homozygous or in the compound heterozygous state with variants in exons 1-6 of NPHS2. *OTOA:* Due to pseudogene interference, our current testing method is not able to reliably detect variants in exons 20-28 (NM_144672.3) in the OTOA gene. *SMN1:* The current testing method detects sequencing variants in exon 7 and copy number variations in exons 7-8 of the SMN1 gene (NM_022874.2). Sequencing and deletion/duplication analysis are not performed on any other region in this gene. About 5%-8% of the population have two copies of SMN1 on a single chromosome and a deletion on the other chromosome, known as a [2+0] configuration (PubMed: 20301526). The current testing method cannot directly detect carriers with a [2+0] SMN1 configuration but can detect linkage between the silent carrier allele and certain population-specific single nucleotide changes. As a result, a negative result for carrier testing greatly reduces but does not eliminate the chance that a person is a carrier. Only abnormal results will be reported. *TERT:* The TERT promoter region is analyzed for both sequencing and copy number variants. *TYR:* Due to the interference by highly homologous regions, our current testing method has less sensitivity to detect variants in exons 4-5 of the TYR gene (NM_000372.5). *VPS45:* LoF is not a known disease mechanism *WRN:* Due to the interference by highly homologous regions within the WRN gene, our current testing method has less sensitivity to detect variants in exons 10-11 of WRN (NM_000553.6).

SIGNATURE:

Shuxi Liu, Ph.D., FACMG
Laboratory Director, Fulgent

Zhenbin Chen, Ph.D., CGMB, FACMG on **11/8/2024**
Laboratory Director, Fulgent

DISCLAIMER:

This test was developed and its performance characteristics determined by Fulgent Therapeutics LLC CAP #8042697 CLIA #05D2043189; 4399 Santa Anita Ave., El Monte, CA, 91731. It has not been cleared or approved by the FDA. The laboratory is regulated under CLIA as qualified to perform high-complexity testing. This test is used for clinical purposes. It should not be regarded as investigational or for research. Since genetic variation, as well as systematic and technical factors, can affect the accuracy of testing, the results of testing should always be interpreted in the context of clinical and familial data. For assistance with interpretation of these results, healthcare professionals may contact us directly at **626-350-0537** or by email at **info@fulgentgenetics.com**. It is recommended that patients receive appropriate genetic counseling to explain the implications of the test result, including its residual risks, uncertainties and reproductive or medical options.

To view the supplemental table describing the carrier frequencies, detection rates, and residual risks associated with the genes on this test please visit the following link:
Beacon Expanded Carrier Screening Supplemental Table

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El Monte, CA, 91731
(p) 626-350-0537 (f) 626-454-1667
info@fulgentgenetics.com
www.fulgentgenetics.com

Patient Information:
13186, donor
DOB: [redacted]
Sex: M
MR#:
Patient#: FT-PT8878938

Partner Information:
Not Tested

Physician:
Kuan, James
ATTN:
GeneticsDepartment,SeattleSpermBank
Seattle Sperm Bank
4915 25th Avenue NE, Ste 204W
Seattle, WA 98105
Phone: (206) 588-1484

Laboratory:
Fulgent Therapeutics LLC
CAP#: 8042697
CLIA#: 05D2043189
Laboratory Director:
Dr. Amar Jariwala
Report Date: **Jul 27,2026**

Accession:
FT-6945987
Test#: FT-TS15644957
Specimen Type: Blood (EDTA)
Collected: Oct 21,2024

Accession:
N/A

FINAL RESULTS

No carrier mutations identified

TEST PERFORMED

Custom Beacon Preconception Carrier Screening Panel
(2 Gene Panel: *BTD and CTC1*; gene sequencing with deletion and duplication analysis)

INTERPRETATION:

Notes and Recommendations:

- No carrier mutations were identified in the submitted specimen. A negative result does not rule out the possibility of a genetic predisposition nor does it rule out any pathogenic mutations in areas not assessed by this test or in regions that were covered at a level too low to reliably assess. Also, it does not rule out mutations that are of the sort not queried by this test; see Methods and Limitations for more information. A negative result reduces, but does not eliminate, the chance to be a carrier for any condition included in this screen. Please see the supplemental table for details.
- This carrier screening test does not screen for all possible genetic conditions, nor for all possible mutations in every gene tested. This report does not include variants of uncertain significance; only variants classified as pathogenic or likely pathogenic at the time of testing, and considered relevant for reproductive carrier screening, are reported. Please see the gene specific notes for details. Please note that the classification of variants can change over time.
- Patients may wish to discuss any carrier results with blood relatives, as there is an increased chance that they are also carriers. These results should be interpreted in the context of this individual's clinical findings, biochemical profile, and family history.
- Gene specific notes and limitations may be present. See below.
- Genetic counseling is recommended. Available genetic counselors and additional resources can be found at the National Society of Genetic Counselors (NSGC; https://www.nsgc.org).

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info@fulgentgenetics.com
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GENES TESTED:

Custom Beacon Preconception Carrier Screening Panel - 2 Genes

This analysis was run using the Custom Beacon Preconception Carrier Screening Panel gene list. 2 genes were tested with 100.0% of targets sequenced at >20x coverage. For more gene-specific information and assistance with residual risk calculation, see the SUPPLEMENTAL TABLE.

BTD, CTC1

METHODS:

Genomic DNA was isolated from the submitted specimen indicated above (if cellular material was submitted). DNA was barcoded, and enriched for the coding exons of targeted genes using hybrid capture technology. Prepared DNA libraries were then sequenced using a Next Generation Sequencing technology. Following alignment to the human genome reference sequence (assembly GRCh37), variants were detected in regions of at least 10x coverage. For this specimen, 100.00% and 100.00% of coding regions and splicing junctions of genes listed had been sequenced with coverage of at least 10x and 20x, respectively, by NGS or by Sanger sequencing. The remaining regions did not have 10x coverage, and were not evaluated. Variants were interpreted manually using locus specific databases, literature searches, and other molecular biological principles. To minimize false positive results, any variants that do not meet internal quality standards are confirmed by Sanger sequencing. Variants classified as pathogenic, likely pathogenic, or risk allele which are located in the coding regions and nearby intronic regions (+/- 20bp) of the genes listed above are reported. Variants outside these intervals may be reported but are typically not guaranteed. When a single pathogenic or likely pathogenic variant is identified in a clinically relevant gene with autosomal recessive inheritance, the laboratory will attempt to ensure 100% coverage of coding sequences either through NGS or Sanger sequencing technologies ("fill-in"). All genes listed were evaluated for large deletions and/or duplications. However, single exon deletions or duplications will not be detected in this assay, nor will copy number alterations in regions of genes with significant pseudogenes. Putative deletions or duplications are analyzed using Fulgent Germline proprietary pipeline for this specimen. Bioinformatics: The FPLMv2.0 pipeline was used to analyze this specimen.

LIMITATIONS:

General Limitations

These test results and variant interpretation are based on the proper identification of the submitted specimen, accuracy of any stated familial relationships, and use of the correct human reference sequences at the queried loci. In very rare instances, errors may result due to mix-up or co-mingling of specimens. Positive results do not imply that there are no other contributors, genetic or otherwise, to future pregnancies, and negative results do not rule out the genetic risk to a pregnancy. Official gene names change over time. Fulgent uses the most up to date gene names based on HUGO Gene Nomenclature Committee (https://www.genenames.org) recommendations. If the gene name on report does not match that of ordered gene, please contact the laboratory and details can be provided. Result interpretation is based on the available clinical and family history information for this individual, collected published information, and Alamut annotation available at the time of reporting. This assay is not designed or validated for the detection of low-level mosaicism or somatic mutations. This assay will not detect certain types of genomic aberrations such as translocations, inversions, or repeat expansions other than specified genes. DNA alterations in regulatory regions or deep intronic regions (greater than 20bp from an exon) may not be detected by this test. Unless otherwise indicated, no additional assays have been performed to evaluate genetic changes in this specimen. There are technical limitations on the ability of DNA sequencing to detect small insertions and deletions. Our laboratory uses a sensitive detection algorithm, however these types of alterations are not detected as reliably as single nucleotide variants. Rarely, due to systematic chemical, computational, or human error, DNA variants may be missed. Although next generation sequencing technologies and our bioinformatics analysis significantly reduce the confounding contribution of pseudogene sequences or other highly-homologous sequences, sometimes these may still interfere with the technical ability of the assay to identify pathogenic alterations in both sequencing and deletion/duplication analyses. Deletion/duplication analysis can identify alterations of genomic regions which include two or more contiguous exons in size (whole blood specimens only); single exon deletions or duplications may occasionally be identified, but are not routinely detected by this test. Saliva swab samples may have reduced sensitivity for CNVs and a higher CNV failure rate. When novel DNA duplications are identified, it is not possible to discern the genomic location or orientation of the duplicated segment, hence the effect of the duplication cannot be predicted. Where deletions are detected, it is not always possible to determine whether the predicted product will remain in-frame or not. Unless otherwise indicated, deletion/duplication analysis has not been performed in regions that have been sequenced by Sanger.

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info@fulgentgenetics.com
www.fulgentgenetics.com

Gene Specific Notes and Limitations

No gene specific limitations apply to the genes on the tested panel.

SIGNATURE:

Dr. Harry Gao, DABMG, FACMG on **07/27/2026**
Laboratory Director, Fulgent

DISCLAIMER:

This test was developed, performed, and its performance characteristics determined by**Fulgent Therapeutics LLC** (CAP# 8042697, CLIA# 05D2043189), 4399 Santa Anita Ave., El Monte, CA 91731. It has not been cleared or approved by the FDA. The laboratory is regulated under CLIA as qualified to perform high-complexity testing. This test is used for clinical purposes. It should not be regarded as investigational or for research. Since genetic variation, as well as systematic and technical factors, can affect the accuracy of testing, the results of testing should always be interpreted in the context of clinical and familial data. For assistance with interpretation of these results, healthcare professionals may contact us directly at **(626) 350-0537** or **info@fulgentgenetics.com**. It is recommended that patients receive appropriate genetic counseling to explain the implications of the test result, including its residual risks, uncertainties and reproductive or medical options.

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El Monte, CA, 91731
(p) 626-350-0537 (f) 626-454-1667
info@fulgentgenetics.com
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To view the supplemental table describing the carrier frequencies, detection rates, and residual risks associated with the genes tested on any Beacon panel, please visit the following link:
Beacon Expanded Carrier Screening Supplemental Table

Patient Information	Specimen Information	Client Information
13186, DONOR **DOB:** [redacted] **AGE:** [redacted] Gender: M Fasting: U Phone: 206.588.1484 Patient ID: 13186 Health ID: 8573035562003034	Specimen: OW879499B Requisition: 0000725 Collected: 10/21/2024 / 13:30 PDT Received: 10/22/2024 / 07:12 PDT Reported: 11/02/2024 / 11:17 PDT	Client #: 98105026 VNLZR00 KUAN, JAMES K SEATTLE SPERM BANK 4915 25TH AVE NE STE 204W SEATTLE, WA 98105-5668

COMMENTS: FASTING:UNKNOWN

Cytogenetic Report

CHROMOSOME ANALYSIS, BLOOD - 14596 Lab:EZ

CHROMOSOME ANALYSIS, BLOOD

Order ID: 24-506485
Specimen Type: Blood
Clinical Indication: GAMETE DONOR

RESULT:
NORMAL MALE KARYOTYPE

INTERPRETATION:
Chromosome analysis revealed normal G-band patterns within the limits of standard cytogenetic analysis.

Please expect the results of any other concurrent study in a separate report.

NOMENCLATURE:
46,XY

ASSAY INFORMATION:

Method: G-Band (Digital Analysis: MetaSyst
Cells Counted: 20
Band Level: 450
Cells Analyzed: 5
Cells Karyotyped: 5

This test does not address genetic disorders that cannot be detected by standard cytogenetic methods or rare events such as low level mosaicism or subtle rearrangements.

Morteza Hemmat, PhD, FACMG (800) NICHOLS-4307

Electronic Signature: 11/2/2024 1:36 PM

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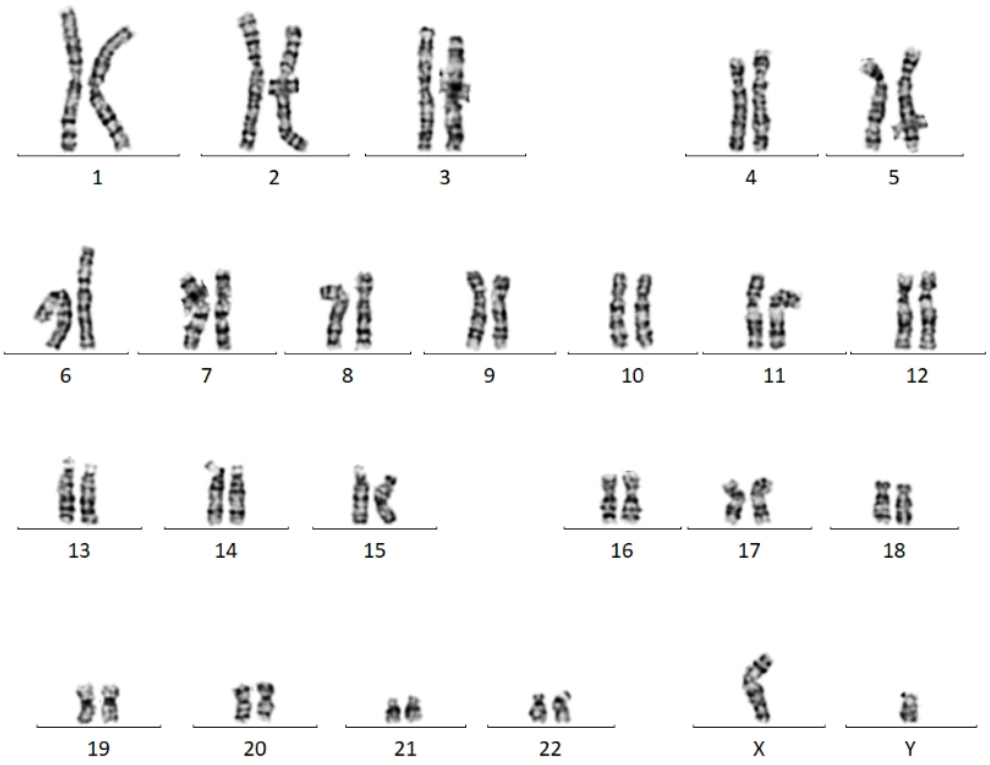
Patient Information	Specimen Information	Client Information
13186, DONOR **DOB:** [redacted] **AGE:** [redacted] Gender: M Fasting: U Patient ID: 13186 Health ID: 8573035562003034	Specimen: OW879499B Collected: 10/21/2024 / 13:30 PDT Received: 10/22/2024 / 07:12 PDT Reported: 11/02/2024 / 11:17 PDT	Client #: 98105026 KUAN, JAMES K



PERFORMING SITE:
EZ QUEST DIAGNOSTICS/NICHOLS SJC, 33608 ORTEGA HWY, SAN JUAN CAPISTRANO, CA 92675-2042 Laboratory Director: IRINA MARAMICA,MD,PHD,MBA, CLIA: 05D0643352

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13186, Donor
Patient ID:
Specimen ID: **295-129-1540-0**

DOB: [redacted]
Age: [redacted]
Sex: **Male**

Patient Report

Account Number: **46857540**
Ordering Physician: **J KUAN**

labcorp

Date Collected: **10/21/2024** Date Received: **10/22/2024** Date Reported: **10/24/2024** Fasting: **Not Given**

Ordered Items: **LP+12AC+CBC/D/Plt+UA+Rh+ABO...**

Date Collected: **10/21/2024**

LP+12AC+CBC/D/Plt+UA+Rh+ABO...

	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
▲	**Glucose** [01]	**102**	**High**		mg/dL	70-99
	Uric Acid [01]	5.5			mg/dL	3.8-8.4
				Therapeutic target for gout patients: <6.0		
	BUN [01]	9			mg/dL	6-20
	Creatinine [01]	0.91			mg/dL	0.76-1.27
	eGFR	121			mL/min/1.73	>59
	Calcium [01]	9.7			mg/dL	8.7-10.2
	Protein, Total [01]	7.3			g/dL	6.0-8.5
	Albumin [01]	4.8			g/dL	4.3-5.2
▲	**Bilirubin, Total** [01]	**1.4**	**High**		mg/dL	0.0-1.2
	Alkaline Phosphatase [01]	68			IU/L	44-121
	LDH [01]	140			IU/L	121-224
	AST (SGOT) [01]	18			IU/L	0-40
	ALT (SGPT) [01]	27			IU/L	0-44
	Cholesterol, Total [01]	122			mg/dL	100-199
	Triglycerides [01]	123			mg/dL	0-149
▼	**HDL Cholesterol** [01]	**34**	**Low**		mg/dL	>39
	LDL Chol Calc (NIH)	66			mg/dL	0-99
	LDL/HDL Ratio	1.9			ratio	0.0-3.6
	Please Note: [01]					

LDL/HDL Ratio

	Men	Women
1/2 Avg.Risk	1.0	1.5
Avg.Risk	3.6	3.2
2X Avg.Risk	6.2	5.0
3X Avg.Risk	8.0	6.1

	Hgb Fractionation by CE: [02]					
	Hgb F [02]	0.0			%	0.0-2.0
	Hgb A [02]	97.4			%	96.4-98.8
	Hgb A2 [02]	2.6			%	1.8-3.2
	Hgb S [02]	0.0			%	0.0
	Interpretation: [02]					

Normal hemoglobin present; no hemoglobin variant or beta thalassemia identified.
Note: Alpha thalassemia may not be detected by the Hgb Fractionation Cascade panel. If alpha thalassemia is suspected, Labcorp offers Alpha-Thalassemia DNA Analysis (#511172).

	ABO Grouping [01]	O				
	Rh Factor [01]	Positive				

Please note: Prior records for this patient's ABO / Rh type are not available for additional verification.

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Date Collected: **10/21/2024**

LP+12AC+CBC/D/Plt+UA+Rh+ABO... (Cont.)

.[01]			
CBC, Platelet Ct, and Diff[01]			
WBC[01]	4.9	x10E3/uL	3.4-10.8
RBC[01]	5.09	x10E6/uL	4.14-5.80
Hemoglobin[01]	15.7	g/dL	13.0-17.7
Hematocrit[01]	46.4	%	37.5-51.0
MCV[01]	91	fL	79-97
MCH[01]	30.8	pg	26.6-33.0
MCHC[01]	33.8	g/dL	31.5-35.7
RDW[01]	12.5	%	11.6-15.4
Platelets[01]	298	x10E3/uL	150-450
Neutrophils[01]	65	%	Not Estab.
Lymphs[01]	23	%	Not Estab.
Monocytes[01]	10	%	Not Estab.
Eos[01]	1	%	Not Estab.
Basos[01]	1	%	Not Estab.
Neutrophils (Absolute)[01]	3.2	x10E3/uL	1.4-7.0
Lymphs (Absolute)[01]	1.2	x10E3/uL	0.7-3.1
Monocytes(Absolute)[01]	0.5	x10E3/uL	0.1-0.9
Eos (Absolute)[01]	0.0	x10E3/uL	0.0-0.4
Baso (Absolute)[01]	0.0	x10E3/uL	0.0-0.2
Immature Granulocytes[01]	0	%	Not Estab.
Immature Grans (Abs)[01]	0.0	x10E3/uL	0.0-0.1
.[01]			
Urinalysis Gross Exam[01]			
Specific Gravity[01]	1.012		1.005-1.030
pH[01]	7.0		5.0-7.5
Urine-Color[01]	Yellow		Yellow
Appearance[01]	Clear		Clear
WBC Esterase[01]	Negative		Negative
Protein[01]	Negative		Negative/Trace
Glucose[01]	Negative		Negative
Ketones[01]	Negative		Negative
Occult Blood[01]	Negative		Negative
Bilirubin[01]	Negative		Negative
Urobilinogen,Semi-Qn[01]	0.2	mg/dL	0.2-1.0
Nitrite, Urine[01]	Negative		Negative
Microscopic Examination[01]	Microscopic not indicated and not performed.		

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The Previous Result is listed for the most recent test performed by Labcorp in the past 5 years where there is sufficient patient demographic data to match the result to the patient. Results from certain tests are excluded from the Previous Result display.

Icon Legend
▲ Out of Reference Range ■ Critical or Alert

Performing Labs
01: SE - Labcorp Seattle, 550 17th Avenue Ste 300, Seattle, WA 98122-5789 Dir: Daniel Toweill, MD
02: BN - Labcorp Burlington, 1447 York Court, Burlington, NC 27215-3361 Dir: Sanjai Nagendra, MD
For Inquiries, the physician may contact Branch: 206-861-7000 Lab: 206-861-7000

Patient Details
13186, Donor

Phone:
Date of Birth: [redacted]
Age: [redacted]
Sex: **Male**
Patient ID:
Alternate Patient ID:

Physician Details
J KUAN
Seattle Sperm Bank
4915 25th Ave Ne Ste 204, SEATTLE, WA, 98105

Phone: **206-588-1484**
Account Number: **46857540**
Physician ID:
NPI: **1891885745**

Specimen Details
Specimen ID: **295-129-1540-0**
Control ID: **L2407953480**
Alternate Control Number: **L2407953480**
Date Collected: **10/21/2024 1350 Local**
Date Received: **10/22/2024 0000 ET**
Date Entered: **10/22/2024 0204 ET**
Date Reported: **10/24/2024 0909 ET**

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