Clinic Note

XIE, JIANHUA DRACO - 1275567

Weird. This note was modified 4 months later.

Result Type:	Clinic Note
Result Date:	23 December 2013 0:00
Result Status:	Modified
Result Title:	Transplant Clinic Outpt Report
Performed By:	Horslen, Simon P, MB ChB on 24 April 2014 11:24
Verified By:	Horslen, Simon P, MB ChB on 24 April 2014 11:24
Encounter info:	94953662, CHMC, Outpatient, 12/23/2013 - 12/28/2013
Contributor system:	SOFTMED

Transplant Clinic Outpt Report

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OUTPATIENT NOTE

XIE, JIANHUA DRACO
DOB: 09/16/2012 M -MR #: 01-27-55-67

CLINIC: TRANSPLANT

DATE OF SERVICE: 12/23/2013

I met with Jianhua's father on 12/23/2013 and was accompanied by Vicky Kuiper, Transplant Nurse Coordinator. This was a scheduled appointment to review the results of Jianhua's full exome screening. At the request of the parents and an outside consultation, whole exome sequencing was undertaken by the Baylor College of Medicine and the results returned to us. Father has always expressed the opinion that he believes that Jianhua has various metabolic diseases and yet, we have had no evidence for this. I therefore spent time with him explaining the findings, although Jianhua and Jianhua's mother were not in attendance on that occasion.

We went through all 22 pages of the report, which showed no significant abnormalities in genes related to the clinical phenotype. There were unrelated heterozygous mutations and polymorphisms of doubtful significance and each of the genes was carefully discussed with the father. I know he was particularly concerned when we got to the section on pharmacogenetics when it mentioned polymorphisms in genes known to metabolize antidepressant medications. He then shared with me medical notes from

his own physician that say that he suffers from significant depressive illness and he also mentioned that this worries him greatly as his own father apparently committed suicide.

Having spent an hour with Mr. Xie, we discussed that there really was no evidence in any of this information that furthered the diagnosis of Jianhua's liver disease. We did, however, make a plan for us to discuss it with our Genetics Team and planned follow up with Biochemical Genetics for more discussion. We planned to see Jianhua in Transplant Clinic for routine follow-up, which took place by my colleague, Dr. Hsu on 1/7/2014.

Electronically Authenticated by
Simon P Horslen, MB ChB 04/24/2014 11:24 A

Simon P Horslen, MB ChB , Attending Physician, Gastroenterology

SPH/cyh Doc #3421424 d: 04/24/2014 09:22 A t: 04/24/2014 10:15 A (1885512-)
Location: TXP
cc: Evelyn K Hsu, MD
Peggy J Kulpa, MD

Completed Action List:
* Perform by Horslen, Simon P, MB ChB on 23 December 2013 0:00
* Transcribe by on 23 December 2013 0:00
* Perform by Horslen, Simon P, MB ChB on 24 April 2014 11:24
* VERIFY by Horslen, Simon P, MB ChB on 24 April 2014 11:24

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Patient Name:	JIANHUA XIE			Sendouts,	
Date of Birth:	September 16, 2012	Lab Number:	563848	Seattle Children's Hospital Laboratory	
Gender:	M	Family #:	353253	Tel No.:	206-987-2563
Hospital/MR #:	1275567	Date Collected:	August 27, 2013	Fax No:	206-987-3840
Accession #:	13-239-0681	Date Received:	August 30, 2013		
Sample Type:	EXT DNA	Date Ordered:	September 4, 2013		
Test Code:	1500	Date Reported:	December 16, 2013		
Indication:	Symptomatic				

Whole Exome Sequencing: Focused Report

563848-1170285

HETEROZYGOUS MUTATION IN THE TNFRSF13B GENE AND OTHER VUS DETECTED, POSSIBLY RELATED TO PATIENT'S CLINICAL PHENOTYPE

According to clinical information submitted by the referring center, this individual has features include micronodular liver cirrhosis, which is associated with coagulopathy, mild jaundice, pruritus, anemia, hyperammonemia, splenomegaly, and failure to thrive. Family history indicates a significant maternal family history of gall bladder issues. In addition, the patient has one sister with GI issues including possible dairy intolerance and a low platelet count, and his mother has a history of heart murmur as a child and hypothyroidism. Previous testing at a different laboratory have identified a heterozygous missense variant of unknown significance in the ATP8B1 gene. We were requested to perform whole exome sequencing on extracted DNA sample from this individual. We also received blood samples of the mother (Sample# 563849) and father (Sample# 563850).

1. Deleterious mutations in disease genes related to clinical phenotype (Table 1)

A heterozygous c.777G>A (p.W259X) mutation in the TNFRSF13B gene was detected in this individual. This mutation is categorized as deleterious according to guidelines (ACMG Recommendations for Standards for Interpretation and Reporting of Sequence Variations: Revision 3567. Genet Med 3568:10:294). Defects in TNFRSF13B are the cause of Immunodeficiency, common variable, 2 (CVID2) [MIM:240500], a primary immunodeficiency characterized by antibody deficiency, hypogammaglobulinemia, recurrent bacterial infections, hepatomegaly, splenomegaly, diarrhea, and an inability to mount an antibody response to antigen. Defects in TNFRSF13B are also the cause of Immunoglobulin A deficiency 2 (IGAD2) [MIM:609529]. The inheritance for both disorders related to TNFRSF13B defects can be autosomal dominant or recessive. This individual is heterozygous for this mutation, which was confirmed by Sanger sequencing. Sanger sequencing also showed that the mother is heterozygous for this change, the father is negative. a second mutation or variant was not detected in the gene, however, the presence of a large or small deletion or duplication cannot be ruled out. It is recommended that correlation of these findings with the clinical phenotype be performed.

2. Variants of unknown clinical significance in disease genes related to clinical phenotype (selected variants are discussed below, see Table 2 for the complete list)

Heterozygous variant(s) of unknown clinical significance (VUS) in other gene(s) were detected in this individual (see Table 2). For dominant disorder(s), the variant(s) in the gene(s) were inherited from a parent. For recessive disorder(s), a second mutation or variant was not detected in the gene(s), however, the presence of a large or small deletion or duplication cannot be ruled out. It is recommended that correlation of these findings with the clinical phenotype be performed.

3. Medically actionable mutations in disease genes unrelated to clinical phenotype (Table 3):

None detected

4. Carrier Status for Recessive Mendelian Disorders (Table 4):

None detected

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Patient Name:	JIANHUA XIE			Sendouts,	
Date of Birth:	September 16, 2012	Lab Number:	563848	Seattle Children's Hospital Laboratory	
Gender:	M	Family #:	353253	Tel No. :	206-987-2563
Hospital/MR #:	1275567	Date Collected:	August 27, 2013	Fax No:	206-987-3840
Accession #:	13-239-0681	Date Received:	August 30, 2013		
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Indication:	Symptomatic				

5. Pharmacogenetic Results (Table 5)

See Table 5.

6. Whole Mitochondrial Genome Sequencing

No large deletion or deleterious mutations were detected.

RECOMMENDATION SUMMARY:

It is recommended that correlation of these findings with the clinical phenotype be performed. Please note that the focused report is limited to genes predicted to be related to the clinical phenotype, medically actionable mutations, as well as carrier status for autosomal recessive conditions recommended for carrier screening by professional organizations such as ACMG or ACOG. An expanded report of the Whole Exome Sequencing Test is available. The Expanded Report will give additional information on mutations and variants in genes which cause disease unrelated to the indication for testing and predicted deleterious mutations in genes with no known current association with disease. It is available at no additional cost if requested within 6 months of the date of the Focused report. The turn-around time for the Expanded Report is 4 weeks. To order, please visit our website at www.bcmgeneticlabs.org and complete the Whole Exome Expanded Report Requisition (Test Code 1510) and FAX to 713-798-2787

RESULT TABLES

Table 1: Deleterious Mutations in Disease Genes Related to Clinical Phenotype

Disease	Inheritance Pattern	Gene	Position	Isoform	Location	Nucleotide	Amino Acid	Zygosity	References/Comments	ESP5400 AA/EA	SIFT / PolyPhen-2
Immunodeficiency, common variable, 2 [MIM:240500]; Immunoglobulin A deficiency 2 [MIM:609529]	AD/AR	TNFRSF13B	Chr17: 16842966	NM_012452	exon5	c.777G>A	p.W259X	Het	Novel mutation; Proband confirmed by Sanger, mother is heterozygous, father is negative.	N/R N/R	N/A/ N/A

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Table 2: Variants of Unknown Clinical Significance in Disease Genes Related to Clinical Phenotype

Disease	Inheritance Pattern	Gene	Position	Isoform	Location	Nucleotide	Amino Acid	Zygosity	References/Comments	ESP5400 AA/EA	SIFT / PolyPhen-2
HEM skeletal dysplasia [MIM:215140]; Pelger-Huet anomaly [MIM:169400]; Reynolds syndrome [MIM:613471]	AD/AR	LBR	Chr1: 225600276	NM_002296	exon8	c.964G>A	p.V322M	Het	rs145104817; Proband confirmed by Sanger sequencing, mother is negative, father is heterozygous.	N/R N/R	Damaging/ Benign
ACAD9 deficiency [MIM:611126]	AR	ACAD9	Chr3: 128622934	NM_014049	exon10	c.988A>C	p.K330Q	Het	rs79530903	2/3736 3/7017	Tolerated/ Probably damaging
Cholestasis, benign recurrent intrahepatic [MIM:243300]; Cholestasis, intrahepatic, of pregnancy, 1 [MIM:147480]; Cholestasis, progressive familial intrahepatic 1 [MIM:211600]	AR	ATP8B1	Chr18: 55329769	NM_005603	exon21	c.2364A>C	p.E788D	Het	rs77317429	N/R N/R	Tolerated/ Possibly damaging
Crigler-Najjar syndrome, type I [MIM:218800], Crigler-Najjar syndrome, type II [MIM:606785], Hyperbilirubinemia, familial transcient neonatal [MIM237900]	AR	UGT1A1	Chr2: 234681073	NM_000463	exon5	c.C1470T	p.D490D	Het	PMID 22710376; rs114123636	0/3738 2/7018	N/A/ N/A

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Developmental delay, multifocal epilepsy & abnormal liver function	AR	NGLY1	Chr3: 25824791	NM_018297	exon1	c.91G>A	p.A31T	Het	Novel variant	N/R N/R	Tolerated/ Probably damaging
Hypercholanemia, familial [MIM:607748]	AR	BAAT	Chr9: 104133522	NM_001701	exon2	c.165C>G	p.F55L	Het	Novel variant	N/R N/R	Tolerated/ Benign
Meckel syndrome 7 [MIM:267010]; Nephronophthisis 3 [MIM:604387]; Renal-hepatic-pancreatic dysplasia 1 [MIM:208540]	AR	NPHP3	Chr3: 132432006	NM_153240	exon6	c.1082C>G	p.S361C	Het	rs146250226	N/R N/R	Damaging/ Probably damaging

Table 3: Medically Actionable Deleterious Mutations in Disease Genes Unrelated to Clinical Phenotype
No mutation/variant detected

Table 4: Carrier Status for Recessive Mendelian Disorders
No mutation/variant detected.

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Patient Name:	JIANHUA XIE			Sendouts,	
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Gender:	M	Family #:	353253	Tel No. :	206-987-2563
Hospital/MR #:	1275567	Date Collected:	August 27, 2013	Fax No:	206-987-3840
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Table 5: Pharmacogenetic Profile Variant Alleles

Drug	Allele	Zygosity	References/Comments
Anti-depressants, anti-epileptics, Plavix	CYP2C19*3	Het	Non-functional allele, PMID 8195181, www.pharmgkb.org/gene/PA124; rs4986893
Anti-depressants, anti-epileptics, Plavix	CYP2C19*2	Het	Non-functional allele, PMID 8195181, www.pharmgkb.org/gene/PA124, CYP2C19*2; rs4244285
Warfarin	VKORC1-1639G>A	Hom	Decreased expression, PMID 21900891, www.warfarindosing.org, www.pharmgkb.org/gene/PA133787052; rs9923231

METHODOLOGY:

1. Whole exome sequencing (WES)*: for the paired-end pre-capture library procedure, genome DNA is fragmented by sonicating genomic DNA and ligating to the Illumina multiplexing PE adapters. The adapter-ligated DNA is then PCR amplified using primers with sequencing barcodes (indexes). For target enrichment/exome capture procedure, the pre-capture library is enriched by hybridizing to biotin labeled VCRome 2.1 (reference 3) in-solution Exome Probes at 47°C for 64 - 72 hours. For massively parallel sequencing, the post-capture library DNA is subjected to sequence analysis on Illumina HiSeq platform for 100 bp paired-end reads. The following quality control metrics of the sequencing data are generally achieved: >70% of reads aligned to target, >95% target base covered at >20X, >85% target base covered at >40X, mean coverage of target bases >100X. SNP concordance to genotype array: >99%. This test may not provide detection of certain genes or portions of certain genes due to local sequence characteristics or the presence of closely related pseudogenes. Gross deletions or duplications, mutations from repetitive sequences may not be accurately identified by this methodology.
2. As a quality control measure**, the individual's DNA is also analyzed by a SNP-array (Illumina HumanExome-12v1 array). The SNP data are compared with the WES data to ensure correct sample identification and to assess sequencing quality.
3. Data analysis and interpretation by Mercury 1.0**: The output data from Illumina HiSeq are converted from bcl file to FastQ file by Illumina CASAVA 1.8 software, and mapped by BWA program. The variant calls are performed using Atlas-SNP and Atlas-indel developed in-house by BCM HGSC. The variant annotations are performed using in-house developed software: HGSC-SNP-anno and HGSC-indel-anno. Synonymous variants, intronic variants not affecting splicing site, and common benign variants are excluded from interpretation unless they were previously reported as deleterious mutations. The variants were interpreted according to ACMG guidelines (reference 5) and patient phenotypes. Mutations/variants related to patient phenotypes are usually confirmed by Sanger sequencing for patients and if available, parents. Sanger confirmation is noted in the "References/Comments" section of the tables if performed. It should be noted that the data interpretation are based on our current understanding of genes and variants at the time of reporting.
4. The focused report contains results of genes related to the patient's clinical phenotype. This interpretation is based on the clinical information provided by the referring center. Results reported in the focused report should be carefully correlated to the individual's clinical findings by the referring physician. Results of carrier status for autosomal recessive conditions are limited to disorders which are recommended for carrier screening by professional societies such as ACMG or ACOG. Pharmacogenetic variants are limited to CYP2C9*2, CYP2C9*3, CYP2C9*5, CYP2C9*6, VKORC1-1639G>A, CYP2C19*2, CYP2C19*3, CYP2C19*4, CYP2C19*5, CYP2C19*8, CYP2C19*10, and CYP2C19*17.
5. The WES test also includes a mitochondrial genome screening*. The mitochondrial genome is amplified by a long-range PCR reaction and subjected to paired-end library construction and Illumina HiSeq 2000 sequencing analyses as described above. The mean depth of coverage for targeted bases is greater than 25,000X by this assay. Only deleterious point mutations and large structural rearrangements with heteroplasmy greater than 20%, i.e. clinically relevant mutations, will be reported. The Revised Cambridge Reference Sequence (RCRS) and the reported polymorphisms and mutations listed in http://mitomap.org database are used as references.
6. The WES report includes minor allele frequency (MAF) data for variants reported in the NHLBI GO Exome Sequencing Project (ESP) ESP5400 database. For example, "1/3738 3/7017" in "ESP5400 AA/EA" column means for the variant listed, the minor allele was observed one time, the major allele was observed 3738 times in African Americans (AA), while the minor allele was observed three times, the major allele was observed

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Gender:	M	Family #:	353253	Tel No. :	206-987-2563
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7017 times in Europeans (EA). N/R in the "ESP5400 AA/EA" column denotes "Not Reported".

7. The WES report also includes in silico predictions for nonsynonymous (missense) changes by SIFT (reference 6) and PolyPhen-2 (reference 7). The predictions are listed in the "SIFT/PolyPhen-2" column with predictions from SIFT (Damaging, Tolerated or N/A) listed first followed by predictions from PolyPhen-2 (Probably damaging, Possibly damaging, Benign or N/A). N/A denotes predictions that cannot be obtained. The N/A situation applies to nonsynonymous changes for which the algorithm used failed to output a prediction or changes not classified as nonsynonymous, for example, nonsense or frameshift mutations. It should be noted that in silico prediction results may vary depending on the algorithm and databases used at the time and should only be used as a guide but not solely relied upon for variant classifications.

References:

1. Illumina, Inc. (2011) Multiplexing Sample Preparation Guide (Part # 1005361 Rev. D). 2011.
2. Roche NimbleGen, Inc. (2010) NimbleGen SeqCap EZ Exome Library SR User's Guide (Version 2.2).
3. Bainbridge MN, Wang M, Wu Y, Newsham I, Muzny DM, Jefferies JL, Albert TJ, Burgess DL, Gibbs RA (2011). Targeted Enrichment beyond the Consensus Coding DNA Sequence Exome Reveals Exons with Higher Variant Densities. Genome Biology 12(7):R68. PMID:21787409.
4. Li H. and Durbin R. (2009) Fast and accurate short read alignment with Burrows-Wheeler Transform. Bioinformatics, 25:1754-60. PMID:19451168.
5. Richards CS, Bale S, Bellissimo DB, Das S, Grody WW, Hegde MR, Lyon E, Ward BE; Molecular Subcommittee of the ACMG Laboratory Quality Assurance Committee (2008). ACMG recommendations for standards for interpretation and reporting of sequence variations: Revisions 2007. Genet Med, 10, 294-300. PMID:18414213.
6. Kumar P, Henikoff S, Ng PC (2009). Predicting the effects of coding non-synonymous variants on protein function using the SIFT algorithm. Nat Protoc. 2009;4:1073-1081. PMID:19561590.
7. Adzhubei IA, Schmidt S, Peshkin L, Ramensky VE, Gerasimova A, Bork P, Kondrashov AS, Sunyaev SR (2010). PolyPhen-2. Nat Methods 7:248-249. PMID:20354512.

Christine M. Eng, M.D.
Medical Director

Yaping Yang, Ph.D.
Laboratory Co-Director

This test was developed and its performance determined by this laboratory. It has not been cleared or approved by U.S. Food and Drug Administration. Since FDA is not required for clinical use of this test, this laboratory has established and validated the test's accuracy and precision, pursuant to the requirement of CLIA '88. This laboratory is licensed and/or accredited under CLIA and CAP. (CAP# 2109314 / CLIA# 45D0660090 WGL CAP# 8004250 / CLIA# 45D2027450). * performed at the Whole Genome Laboratory; ** performed at both the Medical Genetics Laboratories and the Whole Genome Laboratory

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Patient Name:	**CHEN, LIYING**			Sendouts	
Date of Birth:	12/07/1973	Lab Number:	563849	Seattle Children's Hospital Laboratory	
Gender:	F	Family #:	353253	Tel No. :	206-987-2563
Hospital/MR #:	1299135	Date Collected:	08/29/2013	Fax No. :	206-987-3840
Accession #:	13-241-0568	Date Received:	08/30/2013		
Sample Type:	BLOOD	Date Reported:	12/16/2013		
Test Code:	1505				

Whole Exome Sequencing: Focused Report - Parental

563849-1170289

MEMORANDUM

DNA from this individual was received and prepared as control, at no charge, in order to facilitate the interpretation of the Whole Exome Sequencing results for this individual's child. Any parental test results obtained appear in the accompanying child's report, along with information as to how the results relate to the child's analysis. All results for this family are reported under the same family number indicated in all reports.

This report was prepared by Alicia Braxton and electronically signed by Christine M. Eng, M.D.

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Patient Name:	**XIE, YONG**			Sendouts	
Date of Birth:	01/07/1973	Lab Number:	563850	Seattle Children's Hospital Laboratory	
Gender:	M	Family #:	353253	Tel No. :	206-987-2563
Hospital/MR #:	1299131	Date Collected:	08/29/2013	Fax No. :	206-987-3840
Accession #:	13-241-0569	Date Received:	08/30/2013		
Sample Type:	BLOOD	Date Reported:	12/16/2013		
Test Code:	1505				

Whole Exome Sequencing: Focused Report - Parental

563850-1170290

MEMORANDUM

DNA from this individual was received and prepared as control, at no charge, in order to facilitate the interpretation of the Whole Exome Sequencing results for this individual's child. Any parental test results obtained appear in the accompanying child's report, along with information as to how the results relate to the child's analysis. All results for this family are reported under the same family number indicated in all reports.

This report was prepared by Alicia Braxton and electronically signed by Christine M. Eng, M.D.

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Department of Laboratories

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Patient Name: Xie, Jianhua D

Date/Time of Collection: 08/27/2013 1203

MRN: 1275567

Financial Number: 94633493

Tech: 026