Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

PATIENT INFORMATION	SAMPLE	REFERRING PHYSICIAN
Name: **JOHN SMITH**	Date Collected: **12/22/2015**	Name: **Oncologist, M.D.**
Gender: **Male**	Date Received: **12/27/2015**	Institution: **Any Hospital**
Birthday: **04/22/1973**	Source: **FFPE Slides**	Address: **21 Any Street Any City, Philippines**
Age: **43**		Contact: **+632 123-4567**
Address: **14 Any Street, Any Village Any City, Philippines**		

Tumor Profile for John Smith

ICD-10: C34.90 Malignant neoplasm of unsp part of unsp bronchus or lung

Result: **POSITIVE**

Mutations Detected: EGFR-T790M, EGFR-L858R

OncoGxOne™
64 Genes

POTENTIAL CLINICAL BENEFIT SAME TUMOR TYPE

Targeted Therapies	Gene Alteration
Osimertinib	*EGFR*-T790M
Afatinib, Gefitinib, Erlotinib	*EGFR*-L858R

POTENTIAL CLINICAL BENEFIT DIFFERENT TUMOR TYPE

Targeted Therapies	Gene Alteration
Afatinib	*EGFR*-T790M

POTENTIAL DRUG RESISTANCE SAME TUMOR TYPE

Targeted Therapies	Gene Alteration
Erlotinib, Gefitinib	*EGFR*-T790M

CLINICAL TRIALS TO CONSIDER

Drug Target	# Clinical Trials
EGFR-L858R	4
EGFR-T790M	5

HELICE
GENOMIC SCIENCES, INC.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

KEY FINDINGS

1. **Potential Clinical Benefit** in EGFR-mutant NSCLC with **Osimertinib** due to *EGFR* T790M.
2. **Potential Clinical Benefit** in EGFR-mutant NSCLC with **Afatinib, Gefitinib, Erlotinib** due to *EGFR* L858R.
3. **Potential Clinical Benefit** in EGFR-mutant NSCLC with **Afatinib** due to *EGFR* T790M.
4. **Potential Drug Resistance** in Lung adenocarcinoma with **Erlotinib, Gefitinib** due to *EGFR* T790M.
5. Potential clinical side effects with **Carboplatin, Cisplatin, Cyclophosphamide, Oxaliplatin** due to *XRCC1* genotype.

**The Key Findings section is an overview of potential therapeutic benefit or lack thereof. Please refer to the information below for details.*

Medically Actionable Alterations

POTENTIAL CLINICAL BENEFIT – SAME TUMOR TYPE

Gene	Alteration Detected	Therapies	Tumor Type	Reference
EGFR	T790M	Osimertinib	EGFR-mutant NSCLC	FDA
EGFR	L858R	Afatinib, Gefitinib, Erlotinib	EGFR-mutant NSCLC	FDA

POTENTIAL CLINICAL BENEFIT – DIFFERENT TUMOR TYPE

Gene	Alteration Detected	Therapies	Tumor Type	Reference
EGFR	T790M	Afatinib	EGFR-mutant NSCLC	FDA

POTENTIAL DRUG RESISTANCE– SAME TUMOR TYPE

Gene	Alteration Detected	Therapies	Reference
EGFR	T790M	Erlotinib, Gefitinib	FDA

- **EGFR-T790M:** The NCCN guidelines note that EGFR T790M mutations may be associated with resistance to Egfr tyrosine kinase inhibitors, and that osimertinib has been approved for metastatic NSCLC patients with EGFR T790M mutations (v.1.2016).
- **EGFR:** For NSCLC patients with metastatic disease and tumors harboring a sensitizing EGFR mutation, the NCCN guidelines (v.2.2016) suggest treating with erlotinib, afatinib, or gefitinib if the alteration is discovered prior to first-line chemotherapy or interrupting/completing current therapy and treating with erlotinib, afatinib, or gefitinib if the alteration is discovered during first-line chemotherapy.

HELICE GENOMIC SCIENCES, INC.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

CLINICAL TRIALS TO CONSIDER

EGFR-T790M Associated Clinical Trials

Therapies	NCT ID	Title	Phase	Locations #
EGF816	NCT02108964	A Phase I/II, Multicenter, Open-label Study of EGFRmut-TKI EGF816, Administered Orally in Adult Patients With EGFRmut Solid Malignancies	1,2	New York, Massachusetts
Afatinib	NCT01746251	Adjuvant Afatinib in Stage I-III NSCLC With EGFR Mutation	2	New York, Massachusetts
Osimertinib, Necitumumab	NCT02496663	EGFR Inhibitor AZD9291 and Necitumumab in Treating Patients With EGFR-Positive Stage IV or Recurrent Non-small Cell Lung Cancer Who Have Progressed on a Previous EGFR Tyrosine Kinase Inhibitor	1	California
Pemetrexed, Cisplatin, Afatinib	NCT01553942	Afatinib With CT and RT for EGFR-Mutant NSCLC	2	Massachusetts
AZD9291	NCT02511106	AZD9291 Versus Placebo in Patients With Stage IB-IIIA Non-small Cell Lung Carcinoma, Following Complete Tumour Resection With or Without Adjuvant Chemotherapy	3	New Jersey, Maryland

EGFR-L858R Associated Clinical Trials

Therapies	NCT ID	Title	Phase	Locations #
AZD3759, AZD9291	NCT02228369	Oral Epidermal Growth Factor Receptor Tyrosine Kinase Inhibitors, AZD3759 or AZD9291, in Patients Who Have Advanced Non-Small Cell Lung Cancer	1	New York, California
Dasatinib, Afatinib	NCT01999985	Phase I Trial of Afatinib (BIBW 2992) and Dasatinib in Non-small Cell Lung Cancer (NSCLC)	1	Florida
BIBW 2992	NCT02271906	The ATTAIN Study: A Therapeutic Trial of Afatinib In the Neoadjuvant Setting	2	Texas
Capecitabine, Afatinib Dimaleate	NCT02451553	Afatinib Dimaleate and Capecitabine in Treating Patients With Advanced Refractory Solid Tumors, Pancreatic Cancer or Biliary Cancer	1	Indiana, Washington

The two locations closest to the patient's address based on zip code are shown.

Please note: Select clinical trials are shown. For a full list of clinical trials, please search the ClinicalTrials.gov website.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

ALTERATION DETAILS WITH THERAPEUTIC IMPLICATIONS BY TUMOR TYPE

EGFR		
Gene: *EGFR*	Nucleotide: c.2369C>T	Pathways: ErbB family
Alteration Detected: T790M		Variation Type: Missense
Response to Afatinib:		➢ Potential Clinical Benefit in EGFR-mutant NSCLC
Response to Osimertinib:		➢ Potential Clinical Benefit in EGFR-mutant NSCLC
Response to Erlotinib, Gefitinib:		➢ Potential Drug Resistance in Lung adenocarcinoma

EGFR		
Gene: *EGFR*	Nucleotide: c.2573T>G	Pathways: ErbB family
Alteration Detected: L858R		Variation Type: Missense
Response to Afatinib, Gefitinib, Erlotinib:		➢ Potential Clinical Benefit in EGFR-mutant NSCLC

Genes with medically actionable alterations are shown above. No alterations of known clinical significance were detected in the remainder of the OncoGxOne™ Panel Genes shown in Table 1.

THERAPIES WITH POTENTIAL CLINICAL SIDE EFFECTS

Gene	Genotype	Tumor Type	Therapies and Clinical Side Effects	Reference
XRCC1	Q399R/Q399R	Colorectal Cancer, Cervical Cancer, Lung Cancer, Ovarian Cancer, Pancreatic Cancer	Carboplatin, Cisplatin, Cyclophosphamide, Oxaliplatin: Increased risk of severe neutropenia	PharmGKB
XRCC1	Q399R/Q399R	Colorectal Cancer, Cervical Cancer, Lung Cancer, Pancreatic Cancer, Ovarian Cancer	Carboplatin, Cisplatin, Cyclophosphamide, Oxaliplatin: Increased survival and response	PharmGKB

HELICE
GENOMIC SCIENCES, INC.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

ABOUT GENES

EGFR

EGFR encodes the Epidermal growth factor receptor (Egfr), a receptor tyrosine kinase that passes biochemical messages to the cell that stimulate it to grow and divide. Amplification, mutation, and overexpression of EGFR may cause excessive proliferation and tumor formation.

Mutation location in gene and/or protein

EGFR T790M is located within the kinase domain of the Egfr protein and has been shown to result in activation of Egfr (Sutto and Gervasio, 2013, Dixit and Verkhivker, 2009, Yun et al., 2008). This mutation has been described as a gatekeeper mutation that confers resistance to the tyrosine kinase inhibitors erlotinib and gefitinib (Suda et al., 2009, Yun et al., 2008).

EGFR L858R is located in the activation loop of Egfr and has been shown to lead to the activation of Egfr; this mutation has also been reported to confer sensitivity to Egfr tyrosine kinase inhibitors such as erlotinib and gefitinib (Lynch et al., 2004, Paez et al., 2004, Pao et al., 2004).

Mutation prevalence

EGFR mutations have been reported in 37% (14460/39313) of Lung adenocarcinoma samples analyzed in COSMIC (Sep 2016). EGFR mutations have been reported in 5.9-18% of Lung adenocarcinoma samples (cBioPortal for Cancer Genomics, Sep 2016). EGFR mutations have been reported in 14-49% of NSCLC cases, and found to be more common in East Asian patients as compared with other ethnicities (Vallee et al., 2013, Rizzo et al., 2016, Arrieta et al., 2015, Zhou et al., 2016, Lee et al., 2016).

Effect of mutation

EGFR-T790M is an activating mutation. EGFR activating mutations or amplification may predict sensitivity to Egfr-targeted therapies, including inhibitors of multiple ErbB family members, and several have received FDA approval in some tumor types (Mok et al., 2009, Rosell et al., 2009, Tsao et al., 2005). Egfr activation or overexpression may also lead to activation of the PI3K and MAPK pathway and may confer sensitivity to PI3K and MAPK pathway inhibitors (Bancroft et al., 2002). EGFR T790M has been reported to confer resistance to the first generation Egfr TKIs erlotinib and gefitinib (Li et al., 2007). Third generation irreversible Egfr TKIs that target the EGFR T790M mutation, such as rociletinib and osimertinib, have shown efficacy in NSCLC cases that harbor EGFR T790M and are resistant to first generation Egfr TKIs (Yang et al., 2015; IASLC 2015, Abstract 943, Sequist et al., 2015, Mitsudomi et al., 2015; IASLC 2015, Abstract 1406, Jänne et al., 2015). Osimertinib has been approved by the FDA for the treatment of EGFR T790M-mutant metastatic NSCLC (Yang et al., 2015; IASLC 2015, Abstract 943, Mitsudomi et al., 2015; IASLC 2015, Abstract 1406).

EGFR-L858R is an activating mutation. EGFR activating mutations or amplification may predict sensitivity to Egfr-targeted therapies, including inhibitors of multiple ErbB family members, and several have received FDA approval in some tumor types (Mok et al., 2009, Rosell et al., 2009, Tsao et al., 2005). Egfr activation or overexpression may also lead to activation of the PI3K and MAPK pathway and may confer sensitivity to PI3K and MAPK pathway inhibitors (Bancroft et al., 2002).

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

CANCER DRUG INFORMATION

TAGRISSO® (Osimertinib)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2015/208065s000lbl.pdf

GILOTRIF® (Afatinib)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2013/201292s002lbl.pdf

TARCEVA® (Erlotinib)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2013/021743s018lbl.pdf

IRESSA® (Gefitinib)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2015/206995s000lbl.pdf

PARAPLATIN® (Carboplatin)
http://www.accessdata.fda.gov/drugsatfda_docs/nda/2003/20-452.pdf_Paraplatin_Prntlbl.pdf

PLATINOL® (Cisplatin)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2011/018057s080lbl.pdf

ENDOXAN® (Cyclophosphamide)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2012/012141s089lbl.pdf

ELOXATIN® (Oxaliplatin)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2009/021492s011,021759s009lbl.pdf

HELICE
GENOMIC SCIENCES, INC.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

Table 1: OncoGxOne™ Panel Genes

OncoGxOne™ is a single-panel cancer test panel designed to provide comprehensive genomic analysis for cancer therapy. This test detects all types of genetic alterations (point mutations, small insertions/deletions, gene fusions, copy number variations) in the 64 genes listed in the table below. Gene coverage includes all coding exons and untranslated regions (UTRs), as well as select intronic regions known to be involved in gene fusion events.

HGNC Gene Name	RefSeq Accession
ABL1	NP_009297
AKT1	NP_001014432
ALK	NP_004295
ATM	NP_000042
AURKA	NP_940839
BCL2	NP_000624
BCL6	NP_001128210
BCR	NP_004318
BRAF	NP_004324
BRCA1	NP_009225
BRCA2	NP_000050
CCND1	NP_444284
CCNE1	NP_001229
CDK4	NP_000066
CEBPA	NP_004355
CRLF2	NP_071431
CTNNB1	NP_001895
CYP2C8	NP_000761
CYP2D6	NP_000097
DDR2	NP_001014796
DNMT3A	NP_783328
DPYD	NP_000101

HGNC Gene Name	RefSeq Accession
EGFR	NP_005219
ERBB2	NP_004439
ESR1	NP_001116214
ETV6	NP_001978
FGFR1	NP_075598
FGFR2	NP_000132
FGFR3	NP_000133
FLT3	NP_004110
GNA11	NP_002058
GNAQ	NP_002063
HRAS	NP_005334
IDH1	NP_005887
IDH2	NP_002159
JAK1	NP_002218
JAK2	NP_004963
KIT	NP_000213
KRAS	NP_004976
MAP2K1	NP_002746
MET	NP_000236
MLL	NP_005924
MPL	NP_005364
MTHFR	NP_005948

HGNC Gene Name	RefSeq Accession
MYC	NP_002458
NF1	NP_001035957
NPM1	NP_002511
NRAS	NP_002515
PDGFRA	NP_006197
PDGFRB	NP_002600
PIK3CA	NP_006209
PTCH1	NP_001077072
PTEN	NP_000305
RARA	NP_000955
RET	NP_066124
ROS1	NP_002935
RUNX1	NP_001745
SMO	NP_005622
TP53	NP_000537
TPMT	NP_000358
TSC1	NP_001155899
TYMS	NP_001062
UGT1A1	NP_061949
XRCC1	NP_006288

HELICE
GENOMIC SCIENCES, INC.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

References:

- NCCN Biomarkers Compendium.
- U.S. Food and Drug Administration, Table of Pharmacogenomic Biomarkers in Drug Labeling. Available online at: http://www.fda.gov/Drugs/ScienceResearch/ResearchAreas/Pharmacogenetics/ucm083378.htm
- My Cancer Genome at: http://www.mycancergenome.org/
- PharmGKB: The Pharmacogenomics Knowledgebase. Available online at: http://www.pharmgkb.org/index.jsp
- The Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline. Available online at: https://www.pharmgkb.org/page/cpic
- European Medicines Agency, Multidisciplinary: Pharmacogenomics. Available online at: http://www.ema.europa.eu/ema/index.jsp?curl=pages/regulation/general/general_content_000411.jsp&mid=WC0b01ac058002958e
- Swen JJ et al. Pharmacogenetics: from bench to byte - an update of guidelines. Clin Pharmacol Ther. 2011 May; 89(5):662-73.
- Catalogue Of Somatic Mutations In Cancer (COSMIC) at: http://cancer.sanger.ac.uk
- Lynch TJ, et al. Activating mutations in the epidermal growth factor receptor underlying responsiveness of non-small-cell lung cancer to gefitinib. N Engl J Med. 2004 May 20;350(21):2129-39. (PMID: 15118073)
- Paez JG, et al. EGFR mutations in lung cancer: correlation with clinical response to gefitinib therapy. Science. 2004 Jun 4;304 (5676):1497-500. (PMID: 15118125)
- Pao W, et al. EGF receptor gene mutations are common in lung cancers from never smokers and are associated with sensitivity of tumors to gefitinib and erlotinib. Proc Natl Acad Sci U S A. 2004 Sep 7;101(36):13306-11. (PMID: 15329413)
- Vallee A, et al. Detection of EGFR gene mutations in non-small cell lung cancer: lessons from a single-institution routine analysis of 1,403 tumor samples. Int J Oncol. 2013 Oct;43(4):1045-51. (PMID: 23934203)
- Rizzo S, et al. CT Radiogenomic Characterization of EGFR, K-RAS, and ALK Mutations in Non-Small Cell Lung Cancer. Eur Radiol. 2016 Jan;26(1):32-42. (PMID: 25956936)
- Arrieta O, et al. Different mutation profiles and clinical characteristics among Hispanic patients with non-small cell lung cancer could explain the Hispanic paradox. Lung Cancer. 2015 Nov;90(2):161-6. (PMID: 26358312)
- Zhou J, et al. Prevalence and Clinical Profile of EGFR Mutation In Non- Small-Cell Lung Carcinoma Patients in Southwest China. Asian Pac J Cancer Prev. 2016;17(3):965-71. (PMID: 27039821)
- Lee B, et al. Clinicopathologic characteristics of EGFR, KRAS, and ALK alterations in 6,595 lung cancers. Oncotarget. 2016 Mar 14;. (PMID: 26992209)
- Mok TS, et al. Gefitinib or carboplatin-paclitaxel in pulmonary adenocarcinoma. N Engl J Med. 2009 Sep 3;361(10):947-57. (PMID: 19692680)
- Rosell R, et al. Screening for epidermal growth factor receptor mutations in lung cancer. N Engl J Med. 2009 Sep 3;361(10):958-67. (PMID: 19692684)
- Tsao MS, et al. Erlotinib in lung cancer - molecular and clinical predictors of outcome. N Engl J Med. 2005 Jul 14;353(2):133-44. (PMID: 16014883)
- Bancroft CC, et al. Effects of pharmacologic antagonists of epidermal growth factor receptor, PI3K and MEK signal kinases on NF-kappaB and AP-1 activation and IL-8 and VEGF expression in human head and neck squamous cell carcinoma lines. Int J Cancer. 2002 Jun 1;99(4):538-48. (PMID: 11992543)
- Ciardiello F, et al. EGFR antagonists in cancer treatment. N Engl J Med. 2008 Mar 13;358(11):1160-74. (PMID: 18337605)
- Lee SM, et al. FDG uptake in non-small cell lung cancer is not an independent predictor of EGFR or KRAS mutation status: a retrospective analysis of 206 patients. Clin Nucl Med. 2015 Dec;40(12):950-8. (PMID: 26359571)
- Naderi S, et al. EGFR mutation status in Middle Eastern patients with non-squamous non-small cell lung carcinoma: A single institution experience. Cancer Epidemiol. 2015 Dec;39(6):1099-102. (PMID: 26362141)
- Sutto L, et al. Effects of oncogenic mutations on the conformational free-energy landscape of EGFR kinase. Proc Natl Acad Sci U S A. 2013 Jun 25;110(26):10616-21. (PMID: 23754386)
- Dixit A, et al. Hierarchical modeling of activation mechanisms in the ABL and EGFR kinase domains: thermodynamic and mechanistic catalysts of kinase activation by cancer mutations. PLoS Comput Biol. 2009 Aug;5(8):e1000487. (PMID: 19714203)
- Yun CH, et al. The T790M mutation in EGFR kinase causes drug resistance by increasing the affinity for ATP. Proc Natl Acad Sci U S A. 2008 Feb 12;105(6):2070-5. (PMID: 18227510)
- Suda K, et al. EGFR T790M mutation: a double role in lung cancer cell survival® J Thorac Oncol. 2009 Jan;4(1):1-4. (PMID: 19096299)
- Li D, et al. Bronchial and peripheral murine lung carcinomas induced by T790M-L858R mutant EGFR respond to HKI-272 and rapamycin combination therapy. Cancer Cell. 2007 Jul;12(1):81-93. (PMID: 17613438)

HELICE
GENOMIC SCIENCES, INC.

Third Floor CVMC Building
3434 Ramon Magsaysay Boulevard
Sta. Mesa, Manila, Philippines
+632 624 9684 · +63917 865 8743
wecare@helicegenomics.com

- Sequist LV, et al. Rociletinib in EGFR-mutated non-small-cell lung cancer. N Engl J Med. 2015 Apr 30;372(18):1700-9. (PMID: 25923550)
- Jänne PA, et al. AZD9291 in EGFR inhibitor-resistant non-small-cell lung cancer. N Engl J Med. 2015 Apr 30;372(18):1689-99. (PMID: 25923549)
- Wu JY, et al. Effectiveness of tyrosine kinase inhibitors on uncommon epidermal growth factor receptor mutations of unknown clinical significance in non-small cell lung cancer. Clin Cancer Res. 2011 Jun 1;17(11):3812-21. (PMID: 21531810)
- Gazdar A, et al. Hereditary lung cancer syndrome targets never smokers with germline EGFR gene T790M mutations. J Thorac Oncol. 2014 Apr;9(4):456-63. (PMID: 24736066)
- Girard N, et al. Analysis of genetic variants in never-smokers with lung cancer facilitated by an Internet-based blood collection protocol: a preliminary report. Clin Cancer Res. 2010 Jan 15;16(2):755-63. (PMID: 20068085)
- Vikis H, et al. EGFR-T790M is a rare lung cancer susceptibility allele with enhanced kinase activity. Cancer Res. 2007 May 15;67 (10):4665-70. (PMID: 17510392)
- Rosell R, et al. Pretreatment EGFR T790M mutation and BRCA1 mRNA expression in erlotinib-treated advanced non-small-cell lung cancer patients with EGFR mutations. Clin Cancer Res. 2011 Mar 1;17(5):1160-8. (PMID: 21233402)

Test Methodology and Limitations for OncoGxOne™:
Target regions of interest were captured using a custom probe library and sequenced by massive parallel sequencing method (Illumina platform). The detected mutations are annotated based on HG19 reference genome assembly. The OncoGxOne™ test was developed by Admera Health, including determination and validation of performance characteristics. The sensitivity and specificity of this test is greater than 98% and 97%, respectively, when a minimum of 10% tumor tissue is present in the sample. This test has not been approved by the U.S. Food and Drug Administration (FDA) but the FDA has determined that such clearance or approval is not necessary. The OncoGxOne™ test is used for clinical purposes. It should not be regarded as investigational or for research. The Admera Health clinical laboratory is certified under the Clinical Laboratory Improvement Amendments of 1988 (CLIA), accredited by the College of American Pathologists, and is qualified to perform high complexity clinical laboratory testing.

N-of-One, Inc. has provided to Admera Health research, analysis and interpretation, on a patient specific basis, of peer- reviewed studies and publicly available databases. This information may include the association between a specific molecular alteration and clinical benefit, or lack thereof, from FDA-approved therapies and therapies under clinical investigation. Additional information from N-of-One is available on its website at www.n-of-one.com.

Disclaimer of Liability:
The information contained in this report is provided as a service and does not constitute medical advice. At the time of report generation this information is believed to be current and is based upon published research; however, research data evolves and amendments to the prescribing information of the drugs listed will change over time. While this report is believed to be accurate and complete as of the date issued, THE DATA IS PROVIDED "AS IS", WITHOUT WARRANTIES OF ANY KIND, EXPRESS OR IMPLIED, INCLUDING WITHOUT LIMITATION, THE IMPLIED WARRANTIES OF MERCHANTABILITY AND FITNESS FOR A PARTICULAR PURPOSE. As medical advice must be tailored to the specific circumstances of each case, the treating health care professional has ultimate responsibility for all treatment decisions made with regard to a patient including any made on the basis of a patient's genotype.

Signature:

James Dermody, PhD
Laboratory Director
Admera Health
ABMG Certified, Clinical Molecular Genetics

Testing and interpretation performed by Admera Health LLC, 126 Corporate Blvd, South Plainfield, NJ 07080 James Dermody Ph.D. Laboratory Director