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PATIENT INFORMATION	SAMPLE	REFERRING PHYSICIAN
Name: **JOHN DOE**	Date Collected: **09/20/2018**	Name: **Physician, M.D.**
Gender: **Male**	Date Received: **09/25/2018**	Institution:
Birthday: **09/04/1969**	Date of Report: **01/25/2019**	Address: **21 Any Street Any City, Philippines**
Age: **49**	Lab ID:	Contact: **+632 123-4567**
Address: **14 Any Street, Any Village Any City, Philippines**	Testing Method: **Plasma Biopsy (NGS)**	

Tumor Profile for Doe, John

ICD-10: C18.9: Malignant neoplasm of colon, unspecified

LiquidGx™ NGS
17 Genes

Result: **POSITIVE**
Mutations Detected: BRAF-V600E, PIK3CA-E545K, MSI-High
MSI status: **High**

Medically Actionable Alterations

THERAPIES LINKED TO VARIANTS OF KNOWN CLINICAL SIGNIFICANCE

Gene	Molecular Abnormality	Therapies	Tumor Type	Reference	Allele Freq	Clinical Trial
MSI	MSI-High	Pembrolizumab, Nivolumab	Cancer	FDA	UND	N

THERAPIES LINKED TO VARIANTS OF POTENTIAL CLINICAL SIGNIFICANCE

Gene	Molecular Abnormality	Therapies	Tumor Type	Reference	Allele Freq	Clinical Trial
BRAF	V600E	Trametinib	Melanoma	FDA	0.002	Y
BRAF	V600E	Cobimetinib	Melanoma	FDA	0.002	Y

THERAPIES LINKED TO RESISTANCE VARIANTS OF KNOWN CLINICAL SIGNIFICANCE

Gene	Molecular Abnormality	Therapies	Tumor Type	Reference	Allele Freq	Clinical Trial
No medically actionable mutations were detected in this category.						

CNA: Copy Number Amplification; UND: Undetermined

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CLINICAL TRIALS TO CONSIDER

1. BRAF Associated Clinical Trials

Therapies	NCT ID	Title	Phase	Locations#
Adavosertib, Trametinib, Afatinib, Sapanisertib, Dasatinib, Palbociclib, Taselisib, Osimertinib, Defactinib, Sunitinib Malate, Larotrectinib, Crizotinib, Capivasertib, AZD4547, Trastuzumab Emtansine, Dabrafenib, Pertuzumab, GSK2636771, Nivolumab, Trastuzumab, Binimetinib, Vismodegib	NCT02465060	NCI-MATCH: Targeted Therapy Directed by Genetic Testing in Treating Patients With Advanced Refractory Solid Tumors, Lymphomas, or Multiple Myeloma	2	Washington, Delaware
Cetuximab, Folinic Acid, 5-Fluorouracil, Irinotecan, Encorafenib, Binimetinib	NCT02928224	Study of Encorafenib + Cetuximab Plus or Minus Binimetinib vs. Irinotecan/Cetuximab or Infusional 5-Fluorouracil (5-FU)/Folinic Acid (FA)/Irinotecan (FOLFIRI)/Cetuximab With a Safety Lead-in of Encorafenib + Binimetinib + Cetuximab in Patients With BRAF V600E-mutant Metastatic Colorectal Cancer	3	Washington, District Of Columbia
Dabrafenib, Navitoclax, Trametinib	NCT01989585	Dabrafenib, Trametinib, and Navitoclax in Treating Patients With BRAF Mutant Melanoma or Solid Tumors That Are Metastatic or Cannot Be Removed by Surgery	1,2	New Jersey, Massachusetts

2. PIK3CA Associated Clinical Trials

Therapies	NCT ID	Title	Phase	Locations#
AZD1775, AZD2281, AZD6738, AZD5363	NCT02576444	OLAParib COmbinations	2	Tennessee, Connecticut

 # The locations closest to the patient's address based on zip code are shown (for US locations, otherwise show all locations).
Note: Select clinical trials are shown. For a full list of clinical trials, please search the ClinicalTrials.gov website.

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MEDICALLY ACTIONABLE MUTATIONS INFORMATION

Gene	Position	Molecular Abnormality	Nucleotide	Read Depth	Allele Freq	RG	AD
BRAF	chr7:140453136	V600E	c.1799T>A	96305	0.002	5489	11
PIK3CA	chr3:178936091	E545K	c.1633G>A	116085	0.003	5660	17

RG: Quality Read Depth of bases with Phred score >= 20
AD: Depth of variant-supporting bases

MSI Result:MSI-High

Microsatellite instability (MSI) is determined by detecting the length of mononucleotide repeats at five genomic sites (BAT-25, BAT-26, NR-21, NR-24, and NR-27) indicating a defect in DNA repair. Anti-PD-1 therapy has been FDA approved for patients classified as MSI-High. A positive call at ≥2 sites is required for a patient to be classified as MSI-High.

Repeat Site	Position	Result
BAT-25	chr4:55598212-55598236	Positive
BAT-26	chr2:47641560-47641586	Positive
NR-21	chr14:23652347-23652367	Positive
NR-24	chr2:95849362-95849384	Negative
NR-27	chr11:102193509-102193534	Negative

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ABOUT GENES

BRAF *BRAF*

BRAF encodes the signaling protein Braf, which is downstream of Ras and activates the MAPK pathway. Braf signaling is critically involved in the processes of cell division and differentiation. BRAF activating mutations occur predominantly at a single location (V600E) and result in uncontrolled cell growth and tumorigenesis.

Mutation location in gene and/or protein

BRAF V600E is a missense alteration located in the activation domain of the Braf protein (Cantwell-Dorris et al., 2011, Köhler et al., 2016). This alteration has been reported as the most frequently occurring BRAF mutation in cancer, and shown to lead to constitutive activation of the Braf protein and subsequent activation of the MAPK pathway; BRAF V600E has also been shown to be oncogenic and lead to increased survival, proliferation, tumor formation, and invasion, as compared with wild-type BRAF (Pritchard et al., 2007, Cantwell-Dorris et al., 2011).

Mutation prevalence

BRAF mutations have been reported in 11% (7446/67386) of Colorectal adenocarcinoma samples analyzed in COSMIC (Apr 2017). BRAF mutations have been reported in 4.3-8.3% of Colorectal adenocarcinoma samples (cBioPortal for Cancer Genomics, Apr 2017). In the scientific literature, BRAF mutations have been reported in 5-15% of CRC cases (Safaee et al., 2012, Phipps et al., 2015, Ogura et al., 2014, Kadowaki et al., 2015, Carter et al., 2015, Mima et al., 2016, Zhang et al., 2015, El-Deiry et al., 2015). Scientific studies have reported that BRAF mutation incidence in colorectal carcinoma can vary depending on where a given tumor arises in the bowel (Yamauchi et al., 2012, Russo et al., 2014).

Effect of mutation

BRAF-V600E is an activating mutation. BRAF encodes the signaling protein Braf, which is downstream of Ras and activates the MAPK pathway (Vultur et al., 2011). Activating mutations in BRAF may predict sensitivity to Raf or MEK inhibitors. The V600-specific inhibitors vemurafenib and dabrafenib, as well as the MEK inhibitors trametinib and cobimetinib, have been FDA-approved for use in melanoma, and strategies to treat BRAF-mutant tumors are in clinical trials in other tumor types (Solit et al., 2006, Flaherty et al., 2010, Dienstmann et al., 2012, Flaherty et al., 2012, Hauschild et al., 2012, Aprile et al., 2013, Flaherty et al., 2012).

PIK3CA *PIK3CA*

PIK3CA encodes the protein p110-alpha, which is the catalytic subunit of phosphatidylinositol 3-kinase (PI3K). The PI3K pathway is involved in cell signaling that regulates a number of critical cellular functions, including cell growth, proliferation, differentiation, motility, and survival.

Mutation location in gene and/or protein

PIK3CA E545K is a missense alteration located in the helical domain of the p110-alpha protein, at a mutational hotspot (Kang et al., 2005, Bader et al., 2006). This alteration has been shown to lead to activation of p110-alpha and be oncogenic, with transforming capacity in cell lines and induction of tumor formation in mice (Meyer et al., 2013, Kang et al., 2005, Bader et al., 2006).

Mutation prevalence

PIK3CA mutations have been reported in 14% (1811/13024) of Colorectal adenocarcinoma samples analyzed in COSMIC (Apr 2017). PIK3CA mutations have been reported in 20-31% of Colorectal adenocarcinoma samples (cBioPortal for Cancer Genomics, Apr 2017). PIK3CA mutations have been reported in 6.4-16.7% of colorectal cancer cases analyzed in studies in the literature (Karapetis et al., 2014, Shen et al., 2013, Zhu et al., 2014, Kawazoe et al., 2015, Chang et al., 2016, Tabernero et al., 2015, Li et al., 2011).

Effect of mutation

PIK3CA-E545K is an activating mutation. PIK3CA encodes the protein p110-alpha, which is the catalytic subunit of phosphatidylinositol 3-kinase (PI3K). The PI3K pathway is involved in cell signaling that regulates a number of critical

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cellular functions, including cell growth, proliferation, differentiation, motility, and survival (Samuels et al., 2005, Engelman, 2009). Activating PIK3CA alterations may predict sensitivity to PI3K/Akt/mTOR pathway inhibitors, several of which are currently being tested in clinical trials (Janku et al., 2011, Massacesi et al., 2013).

CANCER DRUG INFORMATION

MEKINIST® (Trametinib)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2014/204114s001lbl.pdf

COTELLIC® (Cobimetinib)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2015/206192s000lbl.pdf

OPDIVO® (Nivolumab)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2014/125554lbl.pdf

KEYTRUDA® (Pembrolizumab)
http://www.accessdata.fda.gov/drugsatfda_docs/label/2015/125514s004s006lbl.pdf

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References:

- NCCN Biomarkers Compendium at: http://www.nccn.org/professionals/biomarkers/content/
- 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/
- Knowledge Base of Precision Oncology at: https://pct.mdanderson.org/
- Catalogue Of Somatic Mutations In Cancer (COSMIC) at: cancer.sanger.ac.uk
- Cantwell-Dorris ER, et al. BRAFV600E: implications for carcinogenesis and molecular therapy. Mol Cancer Ther. 2011 Mar;10(3):385-94. (PMID: 21388974)
- Köhler M, et al. Activation loop phosphorylation regulates B-Raf in vivo and transformation by B-Raf mutants. EMBO J. 2016 Jan 18;35(2):143-61. (PMID: 26657898)
- Pritchard C, et al. Mouse models for BRAF-induced cancers. Biochem Soc Trans. 2007 Nov;35(Pt 5):1329-33. (PMID: 17956344)
- Safaee Ardekani G, et al. The prognostic value of BRAF mutation in colorectal cancer and melanoma: a systematic review and meta-analysis. PLoS One. 2012;7(10):e47054. (PMID: 23056577)
- Phipps AI, et al. Association between molecular subtypes of colorectal cancer and patient survival. Gastroenterology. 2015 Jan;148(1):77-87.e2. (PMID: 25280443)
- Ogura T, et al. Clinicopathological characteristics and prognostic impact of colorectal cancers with NRAS mutations. Oncol Rep. 2014 Jul;32(1):50-6. (PMID: 24806883)
- Kadowaki S, et al. Prognostic value of KRAS and BRAF mutations in curatively resected colorectal cancer. World J Gastroenterol. 2015 Jan 28;21(4):1275-83. (PMID: 25632202)
- Carter J, et al. Non-p.V600E BRAF Mutations Are Common Using a More Sensitive and Broad Detection Tool. Am J Clin Pathol. 2015 Oct;144(4):620-8. (PMID: 26386083)
- Mima K, et al. Tumor LINE-1 methylation level and colorectal cancer location in relation to patient survival. Oncotarget. 2016 Jul 4;. (PMID: 27391152)
- Zhang J, et al. Molecular spectrum of KRAS, NRAS, BRAF and PIK3CA mutations in Chinese colorectal cancer patients: analysis of 1,110 cases. Sci Rep. 2015 Dec 22;5:18678. (PMID: 26691448)
- El-Deiry WS, et al. Molecular profiling of 6,892 colorectal cancer samples suggests different possible treatment options specific to metastatic sites. Cancer Biol Ther. 2015;16(12):1726-37. (PMID: 26553611)
- Yamauchi M, et al. Assessment of colorectal cancer molecular features along bowel subsites challenges the conception of distinct dichotomy of proximal versus distal colorectum. Gut. 2012 Jun;61(6):847-54. (PMID: 22427238)
- Russo AL, et al. Mutational analysis and clinical correlation of metastatic colorectal cancer. Cancer. 2014 May 15;120(10):1482-90. (PMID: 24500602)
- Vultur A, et al. Targeting BRAF in advanced melanoma: a first step toward manageable disease. Clin Cancer Res. 2011 Apr 1;17(7):1658-63. (PMID: 21447722)
- Solit DB, et al. BRAF mutation predicts sensitivity to MEK inhibition. Nature. 2006 Jan 19;439(7074):358-62. (PMID: 16273091)
- Flaherty KT, et al. Inhibition of mutated, activated BRAF in metastatic melanoma. N Engl J Med. 2010 Aug 26;363(9):809-19. (PMID: 20818844)
- Dienstmann R, et al. Molecular profiling of patients with colorectal cancer and matched targeted therapy in phase I clinical trials. Mol Cancer Ther. 2012 Sep;11(9):2062-71. (PMID: 22723336)
- Flaherty KT, et al. Combined BRAF and MEK inhibition in melanoma with BRAF V600 mutations. N Engl J Med. 2012 Nov;367(18):1694-703. (PMID: 23020132)
- Hauschild A, et al. Dabrafenib in BRAF-mutated metastatic melanoma: a multicentre, open-label, phase 3 randomised controlled trial. Lancet. 2012 Jul 28;380(9839):358-65. (PMID: 22735384)
- Aprile G, et al. Regorafenib for gastrointestinal malignancies : from preclinical data to clinical results of a novel multi-target inhibitor. BioDrugs. 2013 Jun;27(3):213-24. (PMID: 23435872)
- Flaherty KT, et al. Improved survival with MEK inhibition in BRAF-mutated melanoma. N Engl J Med. 2012 Jul 12;367(2):107-14. (PMID: 22663011)
- Davies H, et al. Mutations of the BRAF gene in human cancer. Nature. 2002 Jun 27;417(6892):949-54. (PMID: 12068308)
- De Roock W, et al. Effects of KRAS, BRAF, NRAS, and PIK3CA mutations on the efficacy of cetuximab plus chemotherapy in chemotherapy-refractory metastatic colorectal cancer: a retrospective consortium analysis. Lancet Oncol. 2010 Aug;11(8):753-62. (PMID: 20619739)
- Deng G, et al. BRAF mutation is frequently present in sporadic colorectal cancer with methylated hMLH1, but not in hereditary nonpolyposis colorectal cancer. Clin Cancer Res. 2004 Jan 1;10(1 Pt 1):191-5. (PMID: 14734469)
- Kang S, et al. Phosphatidylinositol 3-kinase mutations identified in human cancer are oncogenic. Proc Natl Acad Sci U S A. 2005 Jan 18;102(3):802-7. (PMID: 15647370)
- Bader AG, et al. Cancer-specific mutations in PIK3CA are oncogenic in vivo. Proc Natl Acad Sci U S A. 2006 Jan 31;103(5):1475-9. (PMID: 16432179)
- Meyer DS, et al. Expression of PIK3CA mutant E545K in the mammary gland induces heterogeneous tumors but is less potent than mutant H1047R. Oncogenesis. 2013 Sep 30;2:e74. (PMID: 24080956)
- Karapetis CS, et al. PIK3CA, BRAF, and PTEN status and benefit from cetuximab in the treatment of advanced colorectal cancer--results from NCIC CTG/AGITG CO.17. Clin Cancer Res. 2014 Feb 1;20(3):744-53. (PMID: 24218517)

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- Shen Y, et al. Effectors of epidermal growth factor receptor pathway: the genetic profiling ofKRAS, BRAF, PIK3CA, NRAS mutations in colorectal cancer characteristics and personalized medicine. PLoS One. 2013;8(12):e81628. (PMID: 24339949)
- Zhu K, et al. Mutations of KRAS and PIK3CA as independent predictors of distant metastases in colorectal cancer. Med Oncol. 2014 Jul;31(7):16. (PMID: 24861917)
- Kawazoe A, et al. A retrospective observational study of clinicopathological features of KRAS, NRAS, BRAF and PIK3CA mutations in Japanese patients with metastatic colorectal cancer. BMC Cancer. 2015 Apr 11;15:258. (PMID: 25886136)
- Chang SC, et al. Mutation Spectra of Common Cancer-Associated Genes in Different Phenotypes of Colorectal Carcinoma Without Distant Metastasis. Ann Surg Oncol. 2016 Mar;23(3):849-55. (PMID: 26471487)
- Tabernero J, et al. Analysis of circulating DNA and protein biomarkers to predict the clinical activity of regorafenib and assess prognosis in patients with metastatic colorectal cancer: a retrospective, exploratory analysis of the CORRECT trial. Lancet Oncol. 2015 Aug;16(8):937-48. (PMID: 26184520)
- Li HT, et al. KRAS, BRAF and PIK3CA mutations in human colorectal cancer: relationship with metastatic colorectal cancer. Oncol Rep. 2011 Jun;25(6):1691-7. (PMID: 21424126)
- Samuels Y, et al. Mutant PIK3CA promotes cell growth and invasion of human cancer cells. Cancer Cell. 2005 Jun;7(6):561-73. (PMID: 15950905)
- Engelman JA, et al. Targeting PI3K signalling in cancer: opportunities, challenges and limitations. Nat Rev Cancer. 2009 Aug;9(8):550-62. (PMID: 19629070)
- Janku F, et al. PIK3CA mutations in patients with advanced cancers treated with PI3K/AKT/mTOR axis inhibitors. Mol Cancer Ther. 2011 Mar;10(3):558-65. (PMID: 21216929)
- Massacesi C, et al. Challenges in the clinical development of PI3K inhibitors. Ann N Y Acad Sci. 2013 Mar;1280:19-23. (PMID: 23551097)
- Yamamoto H, et al. PIK3CA mutations and copy number gains in human lung cancers. Cancer Res. 2008 Sep 1;68(17):6913-21. (PMID: 18757405)
- Janku F, et al. PIK3CA mutations frequently coexist with RAS and BRAF mutations in patients with advanced cancers. PLoS One. 2011;6(7):e22769. (PMID: 21829508)
- Cui B, et al. Studies on the expression patterns of class I PI3K catalytic subunits and its prognostic significance in colorectal cancer. Cell Biochem Biophys. 2012 Jan;62(1):47-54. (PMID: 21910030)
- Wu S, et al. PIK3CA and PIK3CB silencing by RNAi reverse MDR and inhibit tumorigenic properties in human colorectal carcinoma. Tumour Biol. 2016 Jul;37(7):8799-809. (PMID: 26747178)

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Test Methodology and Limitations for LiquidGx™:

Target regions of interest were sequenced using tagging of individual molecules followed by amplicon library generation and massive parallel sequencing (Illumina platform). The detected mutations are annotated based on hg19 reference genome assembly. The LiquidGx™ test was developed by Admera Health, including determination and validation of performance characteristics. The limit of detection is 0.1% for single nucleotide variants, insertion/deletions, and fusions. Greater than 2% fraction of microsatellite instability can be detected. For copy number variation, LiquidGxTM can detect as low as an extra 0.5 copies of a gene (equivalent to 2.5 total copies). This test has not been approved by the U.S. Food and Drug Administration (FDA) and is for research purposes only. 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.

The panel includes 17 genes, 5 repeat regions, and approximately 170 variants in alignment with National Comprehensive Cancer Network (NCCN) guidelines. The following genetic variants may be detected in the assay: BRAF D594G/V, L597R/Q/S/V, V600D/E/G/K/M/R, K601E; EGFR G719A/C/S, K745_A750del, E746_A750del, E746_A750delELREA, E746_S752delinsA, L747_S752del, L747_P753delinsS, A763_Y764insFQEA, D770_N771insSVD, T790M, C797S, L858R, L861Q/R, Exon19_del, Exon19_ins, Exon20_ins, Amplification; ERBB2 G776L, A775_G776insYVMA, G776_777insVC, Amplification; KRAS G12A/C/D/R/S/V, G13A/C/D/R/S/V, Q61H/K/L/P/R, K117N, A146P/T/V; AKT1 E17K; ALK T1151_L1152insT, F1174L, L1196M, G1202R, S1206Y, G1269A, EML4-ALK, KIF5B-ALK, TFG-ALK, STRN_ALK; MET Exon14_skipping, Amplification; PIK3CA E542K, E545K/G, H1047R; RET M918T, C634R/Y/W, CCDC6-RET, NCOA4-RET, KIF5B-RET; LRIG3-ROS1, TPM3-ROS1, EZR-ROS1, SDC4-ROS1, GOPC-ROS1, SLC34A2-ROS1, CD74-ROS1; NRAS G12C/S/A/D/V/R, G13R/C/A/D/V, Q61K/R/L/H/E/P; KIT W557R, L559A/D, L576P, K642E, W557-L576_indel; MAP2K1 I111S, C121S, P124S/L; PDGFRA D842V; HRAS G12R/V, G13C/R, Q61R; TP53 R175H, G245S, R248Q/W, R249S, R273H/C, R282W; PTEN R130G//Q, R159S, R233*, P248fs, K267fs, T319fs, N323fs. A normal (wild type) genotype signifies the absence of the targeted alleles and does not indicate the absence of other mutations not covered by the assay. The possibility cannot be ruled out that the indicated variants may be present but below the limits of detection for this assay.*

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 and knowledge of gene variants 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.

I certify that these lab results are accurate.

Signatures:

James J. Dermody, Ph.D.
Laboratory Director
Admera Health LLC

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

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