

Test Report

Requisition Number	17023053	Patient Phone Number		Date of Collection	2017/07/10
Patient Name	RENU AGARWAL	Patient E-mail		Date of Report	2017/08/04
ID		Name of Lab	MolQ Laboratory	Sample type	Blood
Date of Birth	66 Years	Lab Phone Number			
Gender	Female	Name of Physician	Dr. Amit Verma		

Patient Test Result Details

Summary Result: Negative

No Clinically Significant Genetic Mutations Detected

Gene	Mutation	Interpretation
98 Genes Screened	None Detected	

Variants are classified according to the standards and guidelines for sequence variant interpretation of the American College of Medical Genetics and Genomics (ACMG). They are evaluated by a board certified pathologist. The five variant classification categories include pathogenic, likely pathogenic, variant of uncertain significance (VUS), likely benign, and benign. CellMax Life only reports pathogenic variants, which have strong lines of evidence associated with increased cancer risk. A summary of all variants found from each classification category can be provided for each patient upon request by their physician.

Comments

No known or potential disease-causing mutations were detected in all genes covered by CellMax Life's 98 gene panel. Variants with no evidence towards pathogenicity were not included in this report.

Electronic Signatures

Lab Supervisor Leon Chen.

Date 2017/08/04

Pathologist M. J. [Signature]

Date 2017/08/04

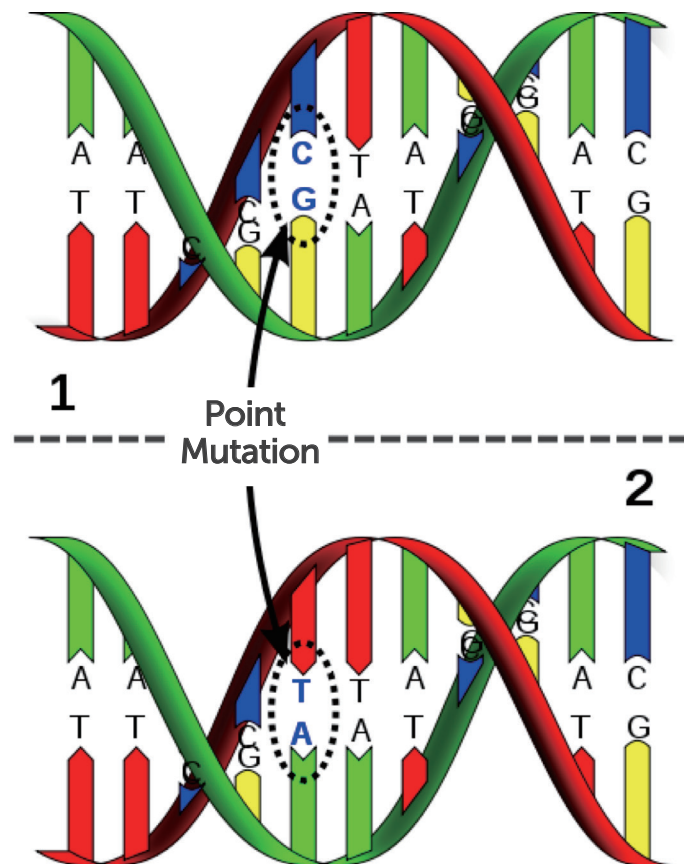
Patient Test Result Summary

This test consists of advanced genetic sequencing of 98 genes to check for multiple different cancers.

<i>AIP</i>	<i>ALK</i>	<i>APC</i>	<i>ATM</i>	<i>BAP1</i>	<i>BARD1</i>	<i>BLM</i>	<i>BMPR1A</i>	<i>BRCA1</i>	<i>BRCA2</i>
<i>BRIP1</i>	<i>BUB1B</i>	<i>CDC73</i>	<i>CDH1</i>	<i>CDK4</i>	<i>CDKN1C</i>	<i>CDKN2A</i>	<i>CEBPA</i>	<i>CEP57</i>	<i>CHEK2</i>
<i>CYLD</i>	<i>DDB2</i>	<i>DICER1</i>	<i>DIS3L2</i>	<i>EGFR</i>	<i>EPCAM</i>	<i>ERCC2</i>	<i>ERCC3</i>	<i>ERCC4</i>	<i>ERCC5</i>
<i>EXT1</i>	<i>EXT2</i>	<i>EZH2</i>	<i>FANCA</i>	<i>FANCB</i>	<i>FANCC</i>	<i>FANCD2</i>	<i>FANCE</i>	<i>FANCF</i>	<i>FANCG</i>
<i>FANCI</i>	<i>FANCL</i>	<i>FANCM</i>	<i>FH</i>	<i>FLCN</i>	<i>GATA2</i>	<i>GPC3</i>	<i>HNF1A</i>	<i>HOXB13</i>	<i>HRAS</i>
<i>KIT</i>	<i>MAX</i>	<i>MEN1</i>	<i>MET</i>	<i>MLH1</i>	<i>MSH2</i>	<i>MSH6</i>	<i>MUTYH</i>	<i>NBN</i>	<i>NF1</i>
<i>NF2</i>	<i>NSD1</i>	<i>PALB2</i>	<i>PHOX2B</i>	<i>PMS1</i>	<i>PMS2</i>	<i>PPM1D</i>	<i>PRF1</i>	<i>PRKAR1A</i>	<i>PTCH1</i>
<i>PTEN</i>	<i>RAD51C</i>	<i>RAD51D</i>	<i>RB1</i>	<i>RECQL4</i>	<i>RET</i>	<i>RHBDF2</i>	<i>RUNX1</i>	<i>SBDS</i>	<i>SDHAF2</i>
<i>SDHB</i>	<i>SDHC</i>	<i>SDHD</i>	<i>SLX4</i>	<i>SMAD4</i>	<i>SMARCA4</i>	<i>SMARCB1</i>	<i>STK11</i>	<i>SUFU</i>	<i>TMEM127</i>
<i>TP53</i>	<i>TSC1</i>	<i>TSC2</i>	<i>VHL</i>	<i>WT1</i>	<i>WRN</i>	<i>XPA</i>	<i>XPC</i>		

KEY

- Mutations were detected in this gene
- Mutations were not detected in this gene



Patient Test Result Summary

List of Genes Tested

Gene Symbol	Gene Name	Major Associated Tumor Types	Test Result
APC	<i>adenomatous polyposis coli</i>	Mutations in the <i>APC</i> gene lead to an increased risk of developing cancers/tumors of the exocrine pancreas, large bowel and rectum (colorectal), stomach, liver, thyroid glands, and the central nervous system.	No mutations of clinical significance detected
BRCA1	<i>breast cancer 1, early onset</i>	Mutations in the <i>BRCA1</i> gene lead to an increased risk to develop female or male breast cancer, ovarian, fallopian tube, or primary peritoneal cancer, pancreatic cancer, prostate cancer, and possibly other types of cancer.	No mutations of clinical significance detected
BRCA2	<i>breast cancer 2, early onset</i>	Mutations in the <i>BRCA2</i> gene lead to an increased risk to develop female or male breast cancer, ovarian, fallopian tube, or primary peritoneal cancer, pancreatic cancer, prostate cancer, and possibly other types of cancer.	No mutations of clinical significance detected
BRIP1	<i>BRCA1 interacting protein C-terminal helicase 1</i>	Mutations in the <i>BRIP1</i> gene lead to an increased risk to develop ovarian cancer (up to 9%, compared to 2% in the average woman), as well as an increased risk to develop female breast cancer.	No mutations of clinical significance detected
CDH1	<i>cadherin 1, type 1, E-cadherin (epithelial)</i>	Mutations in the <i>CDH1</i> gene lead to an increased risk to develop a particular type of gastric cancer (diffuse) and a particular type of female breast cancer (lobular).	No mutations of clinical significance detected
CHEK2	<i>checkpoint kinase 2</i>	Mutations in the <i>CHEK2</i> gene lead to an increased risk to develop female breast cancer (around twice as high as the average woman), colorectal cancer, and possibly other cancers such as male breast cancer, prostate, thyroid, ovarian, or kidney.	No mutations of clinical significance detected
PALB2	<i>partner and localizer of BRCA2</i>	Mutations in the <i>PALB2</i> gene lead to an increased risk to develop female breast cancer, pancreatic cancer, ovarian cancer, and possibly other types of cancer like male breast cancer. The risk to develop breast cancer can vary based on how many close relatives also have breast cancer.	No mutations of clinical significance detected
PTEN	<i>phosphatase and tensin homolog</i>	Mutations in the <i>PTEN</i> gene lead to an increased risk to develop female breast cancer, endometrial/uterine cancer, kidney cancer, colorectal cancer, and possibly other types of cancer.	No mutations of clinical significance detected

Disclaimer

DNA studies do not constitute a definitive test for any disease conditions in an individual. This test was developed and its performance characteristics determined by CellMax Life. Clinical decisions regarding care and treatment of patients should not be solely based on this test. How this information is used to guide patient care is the responsibility of the physician.

The CellMax Life test is designed to assist health care practitioners in providing additional clinical information. The information therein should not be relied upon as being complete or accurate, nor should it be considered as inclusive of all proper treatments or methods of care or as a statement of the standard of care. Medical knowledge develops rapidly and new evidence may emerge between the time information is developed to when it is published or read.

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