SURETOX

SureGx

Hereditary Cancer Test

Sample Variant Report

495 Boulevard Suite 1A
Elmwood Park, NJ 07407
Phone: (201)791-7293
Fax: (201)866-425-4630
Director: Robert Rush, Ph.D.
Reviewed By: Vepkhia Pilauri, Ph.D.,
Sophia Simonishvili Ph.D.

CLIA #: 31D2063148

Client: DR. ANONIMOUS **Phys.:** ANN ANONIMOUS M.D.	**Patient Name:** JOHN JOHNSON **DOB:** 05/15/1942 **Gender:** M **Specimen Type:** Oral Swab	**Lab Acc#:** 1805237001 10:10AM **Collected:** 05/22/18 11:25AM **Accessioned:**05/23/18 10:10AM **Reported:** 06/15/18 09:28AM

Discover™ Hereditary Cancer Risk Assessment Report

Cancer Panel
BreastDiscover, OvarianDiscover, UterineDiscover, ColorectalDiscover, MelanomaDiscover, PancreaticDiscover, GastricDiscover, ProstaticDiscover, LungDiscover, CNSDiscover, KidneyDiscover,BladderDiscover.
Test Indication Information provided indicates that this individual has a personal and/or family history of cancer.

TEST RESULT SUMMARY:

Cancer Panel: Complete Cancer Panel			
Gene	**Variant**	**Classification**	**Zygosity**
BRCA1	NM_007294.3:c.507	PATHOGENIC	HOMOZYGOUS
HNF1A	NM_000545.5:c.1504C>A	LIKELY PATHOGENIC	HETEROZYGOUS
TP53	NM_000546.5:c.1163A>C	UNCERTAIN SIGNIFICANCE	HETEROZYGOUS
ALK	NM_004304.4(ALK):c.4836G>A	LIKELY BENIGN	HETEROZYGOUS
RET	NM_020975.5:c.73+9277T>C	BENIGN	HOMOZYGOUS

Recommendations

- Genetic counseling is recommended to discuss the implications of these results.
- In the absence of definitive pathogenic variant, this patient's risk for future cancers and medical management recommendations must be based on personal and family history of cancer.
- The clinical implications of the variant(s) of uncertain significance remain unclear. For that reason,

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predictive testing for variants of uncertain significance is not recommended for at-risk family members. However, targeted testing of certain family members may help to clarify the effect of such variants. Detailed review of the patient's clinical and family history information by our clinical genetics team is necessary for enrollment in our variant testing program.

- If you would like to discuss these results in further detail, please call one of our genetic counselors.
- GenomeConnect is an NIH initiative created to enable individuals and families with the same genetic variant or medical history to connect and share de-identified information. If you are interested in participating, please visit www.genomeconnect.org.

COMMENTS ON RESULTS

Gene / Coordinate	Variant	Classification	Zygosity
HNF1A / 121437073	NM_000545.5:c.1504C>A	LIKELY PATHOGENIC	HETEROZYGOUS

Interpretation:

MODY is a form of familial noninsulin-dependent diabetes mellitus (NIDDM; 125853) and is characterized by an early age of onset (childhood, adolescence, or young adulthood under 25 years) and autosomal dominant inheritance. For general information on MODY and on genetic heterogeneity in this disorder, see 606391. In their review of MODY, Fajans et al. (2001) stated that, not unexpectedly, the pathophysiologic mechanisms of MODY due to mutations in the HNF4A gene (MODY1) and MODY due to mutations in the HNF1A (MODY3) are very similar since HNF4-alpha regulates the expression of HNF1-alpha. Patients with mutations in these genes may present with a mild form of diabetes. Despite similarly mild elevations in fasting plasma glucose concentrations, patients with mutations in HNF4A or HNF1A have significantly higher plasma glucose concentrations 2 hours after glucose administration than do persons with glucokinase mutations. The hyperglycemia in patients with MODY1 and MODY3 tends to increase over time, resulting in the need for treatment with oral hypoglycemic drugs or insulin in may of these patients (30 to 40% require insulin). These forms of MODY are associated with a progressive decrease in insulin secretion. In most populations, mutations in the HNF1A gene are the most common cause of MODY. Patients with MODY1 or MODY3 may have the full spectrum of complications of diabetes. Microvascular complications, particularly those involving the retina or kidneys, are as common in these patients as in patients with type I or type II diabetes (matched according to the duration of diabetes and the degree of glycemic control) and are probably determined by the degree of glycemic control. Patients with MODY1 lose the glucose priming effect of mild hyperglycemia on insulin secretion. Both prediabetic and diabetic persons with mutations in the HNF4A gene secrete decreased amounts of insulin in response to glucose and in response to arginine and also have an impairment of glucagon secretion in response to arginine.

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Furthermore, a defect in the hypoglycemia-induced secretion of pancreatic polypeptide has been found in prediabetic and diabetic persons who have mutations in the gene for HNF4A. These findings suggested that a deficiency of HNF4A resulting from mutations in this gene may affect the function of the beta, alpha, and pancreatic polypeptide cells within pancreatic islets. Patients with mutations in HNF1A have decreased renal absorption of glucose (i.e., a low renal threshold for glucose) and glycosuria. A deficiency of HNF4A affects triglyceride and apolipoprotein biosynthesis and is associated with a 50% reduction in serum triglyceride concentrations and a 25% reduction in serum concentrations of apolipoproteins AII and CIII and Lp(a). Fajans et al. (2001) reported that mutations in the HNF1A gene have been identified in all racial and ethnic backgrounds, including European, Chinese, Japanese, African, and American Indian. Mutations in the HNF1A gene appear to be the most common cause of MODY among adults seen in diabetic clinics. Ellard (2000) stated that 65 different mutations in the TCF1 gene had been found to cause MODY3 in a total of 116 families worldwide. They noted that diagnostic and predictive genetic testing is possible for the majority of patients with MODY, opening new avenues for the classification, prediction, and perhaps eventually the prevention of diabetes in these families. Vaxillaire et al. (1995) studied linkage in 12 French MODY families in which diabetes was not genetically linked to previously identified MODY loci. By a genomewide segregation analysis of highly informative microsatellite markers, they localized the gene for a MODY susceptibility locus (MODY3) to 12q in 6 families. The locus in question was thought to lie within a 7-cM interval bracketed by D12S86 and D12S342 (in 12q22-qter). The patients exhibited major hyperglycemia with a severe insulin (176730) secretory defect, suggesting that the causal gene is implicated in pancreatic beta-cell function. Lesage et al. (1995) studied the possible implication of the MODY3 locus in late-onset NIDDM. In 600 affected sib pairs from 172 French families, linkage was rejected by all methods of analysis, implying that the MODY gene on 12q is not a major gene in late-onset NIDDM in this population. Menzel et al. (1995) found evidence of linkage to chromosome 12 in 3 families with MODY from Denmark, Germany, and the U.S. (Michigan) and suggestive evidence of linkage in a family from Japan. They placed the locus in a 5-cM interval between markers D12S86 and D12S807/D12S820. The age of onset of NIDDM was less than 25 years of age in the youngest generation in each pedigree and the segregation was consistent with autosomal dominant inheritance. In 1 pedigree, the body weight of 18 of 22 diabetic subjects was known and only 1 was obese. Diabetes was diagnosed in all but 1 of the subjects before 20 years of age. From the location of the linked markers the MODY3 locus was thought to be in the region 12q24.1-q24.32.

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Test Methods and Limitations

Developed by Illumina, this panel focuses on the coding exonic regions of genes annotated in HG19 reference genome. Genomic targets were identified based on information in the Human Gene Mutation Database (HGMD), the Online Mendelian Inheritance in Man (OMIM) catalog, GeneTests.org, Illumina TruSight sequencing panels and other commercially available sequencing panels. Combining data from these sources ensured that genes currently identified in clinical research settings as pathogenic were included in the panel.
Targeted regions for “Inherited Cancer Gene Panel” includes the whole regions of the genes indicated below:

BreastDiscover:
ATM, BARD1, BRCA1, BRCA2, BRIP1, BUB1B, CDH1, CDKN1B, CHEK2, CYLD, ERCC4, FANCA, FANCE, NBN, NF1, NF2, PALB2, PTEN, SLX4, TP53, WRN

OvarianDiscover:
BARD1, BRCA1, BRCA2, BRIP1, CDC73, DDB2, DICER1, EPCAM, FANCM, MLH1, MSH2, MSH6, NF2, PALB2, PMS2, PTEN, RAD51C, RAD51D, STK11, TP53, WRN

UterineDiscover:
DDB2, EPCAM, ERCC3, ERCC4, EZH2, FH, HRAS, MLH1, MSH2, MSH6, NF2, PALB2, PMS2, PRF1, PTEN, RECQL4, STK11, TP53

ColorectalDiscover:
APC, BMPR1A, CDH1, CHEK2, CYLD, DDB2, DIS3L2, EPCAM, FANCA, FANCL, GNAS, KIT, MLH1, MSH6, MUTYH, PMS1, PMS2, POLD1, POLE, PTEN, SMAD4, STK11, TP53, WRN

MelanomaDiscover:
BAP1, BLM, BRCA2, CDK4, CDKN2A, DDB2, DICER1, ERCC3, ERCC5, EZH2, FANCB, FANCE, FANCL, GATA2, KIT, NF2, PTEN, TP53, XPA,XPC

PancreaticDiscover:
BRCA1, BRCA2, CDC73, CDKN1B, CHEK2, DICER1, FANCD2, FH, HNF1A, NBN, NF2, RAD50, TP53

GastricDiscover:
AIP, APC, BMPR1A, CDH1, EPCAM, FANCA, FANCB, KIT, MLH1, MSH2, MSH6, NF2, PRKAR1A, RAD50, RHBDF2, SMAD4, STK11, TP53

ProstaticDiscover:
BRCA1, BRCA2, CDC73, CDKN1B, CHEK2, DICER1, FANCD2, FH, HNF1A, NBN, NF2, RAD50, TP53

LungDiscover:
ALK, CYLD, EGFR, ERCC2, EXT1, EXT2, FANCA, FANCM, GATA2, GPC3, HRAS, NF2, PHOX2B, RHBDF2, WRN, XPA

CNSDiscover:
ALK, BLM, BUB1B, CDKN1B, CDKN1C, DIS3L2, EGFR, EXT1, EXT2, FANCC, FANCG, GPC3, MAX, NF1, NF2, PALB2, PDE4D, SDHA, SDHAF2, SDHB, SDHC, SDHD, SUFU, TMEM127, TSC1, TSC2, VHL, WT1

KidneyDiscover:
CASR, DIS3L2, EGFR, EPCAM, EXT2, FANCC, FANCD2, FANCM, FLCN, GPC3, MET, MLH1, MSH2, NF2, PMS2, PTEN, SDHB, SDHC, SDHD, SMARCB1, TP53, TSC1, TSC2, VHL

BladderDiscover:
DDB2, ERCC2, ERCC5, EZH2, FH, GNAS, HRAS, PRF1, RB1, RET, RUNX1

Other Clinical Comments:

A negative result may not correlate with lower risk due to the following reasons:
1. This report is based on the selected genes included in the test panel, and does not predict risks associated with other genes or unknown genes not included in the test.
2. Only the protein coding regions and splicing sites of these genes are included in the analysis and reported herein. Sequences outside the sequenced regions of these genes are not sequenced for genetic risk analysis.

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3. Certain types of mutations other than single nucleotide changes or smaller deletions/ duplications may not be identified based on the current Next Generation Sequencing analysis technology used in this test.
4. A more complete limitation of the test can be found in Appendix A.

Sequencing and Variant Detection

Genomic DNA was extracted from clinical sample (oral swab), library preparation via Illumina protocols, capture-based enrichment of a targeted region was performed by solution-based hybridization which enriches for coding regions of targeted genes with specific probes. Multiple quality control steps were performed for sample and derivative quality evaluation. Sequencing was performed using the Illumina Next Generation Sequencing, with 100-151 bp reads, sequence QC metrics were required, and a minimum average coverage depth of 200X was required, Sequencing reads were aligned to the reference genome (UCSC hg19) by Local Run Manager enrichment module with GATK settings and validated by proprietary algorithm. The minimum sequence depth for all targeted regions was evaluated, further validation is recommended for exons with depth of coverage <50X. We recommend that variants of interest which do not meet the coverage minimum be confirmed clinically before treatment is undertaken.

Variant Analysis and Report Generation

Reported variants were filtered to include those present in the targeted coding exonic regions and adjacent splice sites. Resulting variants were analyzed and reported using the GCTK platform. To maintain the most up-to-date annotations, the database is updated regularly. As a result, variant classification and/or interpretation may change over time as more information becomes available.
The following databases and tools are included in the Illumina software platform:
1. Disease association: ClinVar
2. Population Frequencies: dbSNP (http://ncbi.nih.gov/SNP/), ensembl (www.wnsembl.org) , 1000 Genomes Project (www.1000genomes.org/), ExAC (http://exac.broadinstitute.org/).
3. Severity prediction: SIFT, MutationTaster, POLYPhen2
4. Conservation prediction: GERP++, PhyloP
5. Gene tolerance: RVIS score, according to published work 10.1371/journal.pgen.1003709
Secondary/incidental Sequence Variant(s) based on ACMG guidelines are not in this report.
Not all mutations compared to the reference sequence have been listed on this report. Mutations were identified using the filters described below. These mutations were further reviewed by a medical geneticist, and only variations of clinical significance (primary findings) are included in this report.

DISCLAIMER

This Report was generated using the materials and methods described above, which required the use of various reagents, protocols, instruments, software, databases, and other items, some of which were provided or made accessible by third parties. A defect or malfunction in any such reagents, protocols, instruments, software, databases, and/or other items may compromise the quality or accuracy of the Report.
The Report has been generated based on, and incorporates references to various scientific manuscripts, references, and other sources of information that were prepared by third parties that describe correlations between certain genetic mutations and particular diseases (and/or certain therapeutics that may be useful in ameliorating the effects of such diseases). Such information and correlations are subject to change over time in response to future scientific and medical findings. Suretox makes no representation or warranty of any kind, expressed or implied, regarding the accuracy of the information provided by or contained in such manuscripts, references, and other sources of information. If any of the information provided by or contained in such manuscripts, references, and other sources is later determined to be inaccurate, the accuracy and quality of the Report may be adversely impacted. Suretox is not obligated to notify you of any impact that future scientific or medical research findings may have on the Report. The Report must always be interpreted and considered within the clinical context, and a physician should always consider the Report along with all other pertinent information and data that a physician would prudently consider prior to providing a diagnosis to a patient or developing and implementing a plan of care for a patient. The Report should never be considered or replied upon alone in making any diagnosis or prognosis. The manifestation of many diseases are caused by more than one gene variant, a single gene variant may be relevant to more than one disease, and certain relevant gene variants may not have been considered in the Report. In addition, many diseases are caused or influenced by modifier genes, epigenetic factors, environment factors, and other variables that are not addressed by the Report (or that are otherwise unknown). As such, the relevance of the Report should be interpreted in the context of a patient's clinical manifestations. Medical knowledge annotation is constantly updated and reflects the current knowledge at the time.
The test performance characteristics were determined by Suretox. The Report was generated by Suretox as required by the CLIA 1988 regulations. The Report, and the tests used to generate the Report, have not been cleared or approved by the U.S. Food and Drug Administration

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(FDA), since FDA has determined that such clearance or approval is not necessary. This laboratory is CLIA certified to perform high complexity testing.