# PREVENTEST™ MOLECULAR REPORT
# PHYSICIAN GUIDE

**CONFIDENTIAL**

| PATIENT | | SPECIMEN | | HEALTHCARE PROVIDER |
|---|---|---|---|---|
| Name: | **Sample Patient** | Specimen Type: | **Saliva** | Dr. Sample Doctor, MD. |
| Date of Birth: | **xx/xx/xxxx** | Completion of Testing: | **xx/xx/xxxx** | Test Medical Center |
| Gender: | **Female** | | | 123 Main St |
| Accession #: | **PVT-19-xxxxx** | | | Testville, TX 55555 |
| Test Type: | **Cancer Risk Analysis** | | | Phone: |
| | | | | Fax: |

**Ordering Physician: Dr. Sample Doctor, MD**

## Result: Positive – Pathogenic Variant Detected at BRCA2 Gene

Note: This mutation is associated with Hereditary Breast and Ovarian Cancer Syndrome (HBOC). The patient may also be considered a carrier for Fanconi anemia. Please see section "Reproductive Recommendations."

**Additional Findings: No other variants of clinical significance identified**

| FINDING | CODON | PROTEIN | INTERPRETATION |
|---|---|---|---|
| **BRCA2. Nonsense Mutation. Exon 11** | **c.3922G>T** | **p.E1308*** | **Pathogenic** |
| | | | |

## PERSONAL/FAMILY HISTORY SUMMARY AND MANAGEMENT INFORMATION*

| FAMILY MEMBER | DIAGNOSIS/CANCER | AGE |
|---|---|---|
| Mother | Breast | 45 |
| Maternal Aunt | Breast | 42 |
| Maternal Aunt | Ovarian | 50 |
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| | | |

| FAMILY MEMBER | DIAGNOSIS/CANCER | AGE |
|---|---|---|
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Genes tested: APC, ATM, BARD1, BMPR1A, BRCA1, BRCA2, BRIP1, CDH1, CDK4, CDKN2A, CHEK2, ELAC2, EPCAM, HOXB13, MLH1, MRE11A, MSH2, MSH6, MUTYH, NBN, NF1, PALB2, PMS2, POLD1, POLE, PTCH1, PTEN, RAD50, RAD51C, RAD51D, RECQL4, RET, RINT1, SMAD4, STK11, TP53.

*Patient personal/family history was provided by the healthcare provider on the requisition form and may not have been verified by GeneID.

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## OVERVIEW OF BRCA2 MUTATIONS

- Mutations on BRCA2 are associated with Hereditary Breast and Ovarian Cancer Syndrome (HBOC).[1,2]
- Females with HBOC have a much higher risk of breast and ovarian cancer than the general population.[1] They are also at an increased risk for fallopian tube cancer and primary peritoneal cancer.[1]
- Males have a higher risk of male breast cancer than the general population, although the absolute risk is still low. Males are also at an increased risk for prostate cancer.[4]
- Males and females with BRCA2 mutations have an elevated risk of pancreatic cancer and melanoma.[3]

## GENE INFORMATION

The BRCA2 gene codes for the BRCA2 protein which acts as a tumor suppressor. Tumor suppressor proteins help prevent cells from growing and dividing too rapidly or in an uncontrolled way, as they do in cancer.[4] The mutation identified prevents or alters the functioning of this gene, leading to an increased cancer risk.

## CANCER RISK

Statistics below refer to patients with mutations compared to the general population. Having a genetic mutation does not guarantee cancer, and there are many factors that can contribute to cancer risk.

| GENDER | CANCER | AGE | RISK |
|---|---|---|---|
| **Females** | Breast Cancer | Up to 70 years | 49% |
| | Contralateral Breast Cancer | Up to 70 years | Increased |
| | Ovarian Cancer | Up to 70 years | 11-18% |
| | Pancreatic Cancer | Up to 80 years | 2.8% |
| | Melanoma | Lifetime | Increased |
| **Males** | Breast Cancer | Lifetime | 7-8% |
| | Prostate Cancer | Lifetime | 2-6 fold increase |
| | Pancreatic Cancer | Up to 80 years | 6.9% |
| | Melanoma | Lifetime | Increased |

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## RECOMMENDATIONS OVERVIEW

The overview on medical management is based on the genetic test results. The recommendations included below are only those issued by the National Comprehensive Cancer Network® (NCCN®) unless otherwise indicated. Recommendations may focus only on the most common cancers associated with the mutation identified. References are provided and should be relied upon for additional information. Some genetic mutations may have implications for other conditions. Only cancer-related recommendations are included.

Recommendations within this document are for informational purposes only. Specific recommendations and treatment plans should be developed by doctors and genetic counselors with expertise in the relevant syndromes and cancers. Strategies may warrant adjustment due to additional information such as patient medical history, family history, other treatments or surgeries. Patient should be educated regarding signs and symptoms of cancers(s), especially those associated with BRCA gene mutations.

## NATIONAL COMPREHENSIVE CANCER NETWORK (NCCN) RECOMMENDATIONS

| PROTOCOL | SUMMARY | WHO | BEGIN | FREQUENCY |
|---|---|---|---|---|
| **Breast and Ovarian Recommendations for Women** | | | | |
| **Breast Awareness**[5] | Women should be familiar with their breasts through self-examinations and promptly report changes to their health care provider. | All | 18 years | Periodically and consistently |
| **Clinical Breast Exam**[5] | Examination by a healthcare provider trained to recognize abnormalities and warning signs. | All | 25 years | Every 6-12 months |
| **Breast Screenings**[5] | Breast MRI with contrast. * If MRI is unavailable, mammogram with consideration of tomosynthesis. | All | 25-29 years** | Annually |
| | Mammogram with consideration of tomosynthesis and Breast MRI screening with contrast. | All | 30-75 years | Annually |
| | Individualized management. | All | >75 years | Individually-based |
| | For women with a BRCA mutation who are treated for breast cancer and have not had a bilateral mastectomy, screening with annual mammogram and breast MRI should continue as described above. | Women treated for breast cancer | - | Annually |
| **Chemoprevention**[5] | Use of estrogen receptor modulators has been shown to reduce the risk of invasive breast cancer in postmenopausal women considered at high-risk for developing breast cancer. However, limited data are available on the specific use of these agents in patients with BRCA1/2 mutations. | | | Individually-based |
| **Risk-Reducing Mastectomy**[5] | Discuss the option of a risk-reducing mastectomy. Counseling should include a discussion regarding the degree of protection, reconstruction options, and risks. | | | Individually-based |

*Breast MRI is preferably performed on days 7-15 of a menstrual cycle in premenopausal women
**Age to begin the MRIs can be individualized based on family history if a breast cancer diagnosis before age 30 is present.

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| PROTOCOL | SUMMARY | WHO | BEGIN |
|---|---|---|---|
| **Ovarian Cancer Recommendations[5]** | | | |
| **Bilateral Salpingo-Oophorectomy[5]** | Recommend a risk-reducing Salpingo-oophorectomy (RRSO) for ovarian cancer. Oophorectomy likely reduces the risk of developing breast cancer in premenopausal women, but the magnitude is uncertain and may be gene specific. | All | Typically, 35-40 and upon completion of child bearing, but it is reasonable to delay RRSO for management of ovarian cancer risk until age 40-45 unless age at diagnosis in the family warrants earlier age for consideration of prophylactic surgery. |
| **Salpingectomy[5]** | Salpingectomy alone is not the standard of care for risk reduction, although clinical trials are ongoing; the concern for risk-reducing salpingectomy alone is that women are still at risk for developing ovarian cancer. In addition, in premenopausal women, oophorectomy likely reduces the risk of developing breast cancer, but the magnitude is uncertain and may be gene-specific. | | |
| **Transvaginal ultrasound and/or CA-125[5]** | For those patients who have not elected for a risk-reducing salpingo-oophorectomy, transvaginal ultrasound combined with serum CA-125 for ovarian cancer screening, although of uncertain benefit, may be considered at the clinician's discretion. | | |
| **Other Recommendations Related to Breast and Ovarian Cancer[5]** | | | |
| Consider risk-reducing agents as options for breast and ovarian cancer, including a discussion of the risks and benefits. | | | |
| Consider investigational imaging and screening studies, when available (e.g. novel imaging technology and/or more frequent screening intervals) in the context of a clinical trial. | | | |
| Address psychosocial, social, and quality-of-life aspects of undergoing risk reducing mastectomy and/or Salpingo-oophorectomy. | | | |
| **Pancreatic Cancer and Melanoma Recommendations[5]** | | | |
| No specific screening guidelines exist for pancreatic cancer and melanoma, but screening may be individualized based on cancers observed in the family. Additionally, for both men and women testing positive for a BRCA1/2 mutation, a full body skin and eye exam for melanoma screening and investigational protocols for pancreatic cancer screening should be considered. | | | |

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## REPRODUCTIVE RECOMMENDATIONS

There is a concern for patients with mutations in passing their risk onto their children. Patients of reproductive age should be advised about options for prenatal diagnosis and assisted reproduction including pre-implantation genetic diagnosis. Discussion should include known risks, limitations, and benefits of these technologies. Referral to a reproductive specialist and a genetic counselor is recommended.

Beyond the risk of passing the single mutation to offspring, many BRCA2 mutations are linked to a rare autosomal recessive condition called Fanconi anemia. For this reason, it is recommended that individuals with BRCA2 mutations have their partners tested to determine if they too have a mutation. If both partners have BRCA2 mutations, any pregnancy would carry a 25% risk of the child having Fanconi anemia.

## RISK TO FAMILY MEMBER

Blood relatives of mutation carriers are at an increased risk of having a BRCA2 mutation.

- 1st degree relatives (Mother, Father, Sister, Brother, Daughter, Son) of an individual with a mutation have a 50% chance of having the mutation.
- 2nd degree relatives (Grandmother, Grandfather, Aunt, Uncle, Niece, Nephew, Granddaughter, Grandson, as well as Half-Brothers and Half-Sisters) of an individual with a mutation have a 25% chance of having the mutation.
- 3rd degree relatives (Great Grandmother, Great Grandfather, Great Aunt, Great Uncle, Cousins, etc.) of an individual with a mutation have a 12.5% chance of having the mutation.

HIPAA restrictions prevent a physician from sharing this information with family members without permission from the patient. The patient is recommended to share information about possible inherited cancer risk, options for risk assessment and management with family or friends. Recommend genetic counseling and consideration of genetic testing for at-risk relatives.

## ADDITIONAL RESOURCES AND SUPPORT

| | |
|---|---|
| National Society of Genetic Counselors | http://www.nsgc.org |
| National Cancer Institute | http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq |
| Center for Disease Control and Prevention | http://www.cdc.gov/cancer/knowledge/provider-education/genetics/hboc-syndrome.htm |
| FORCE- Facing Our Risk of Cancer Empowered | http://www.facingourrisk.org/get-support/ |
| National Comprehensive Cancer Network | https://www.nccn.org/ |
| Genetic Information Nondiscrimination Act | http://ginahelp.org |

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## TECHNICAL OVERVIEW OF MUTATION

| Finding | Codon | Protein | Interpretation |
|---|---|---|---|
| **BRCA2. Nonsense Mutation. Exon 11** | **c.3922G>T** | **p.E1308*** | **Pathogenic** |

The outcomes of this analysis are consistent with a germline heterozygous BRCA2 truncating mutation at exon 11, noted as c.3922G>T (rs80358638; Chr13: 32338277 (on Assembly GRCh38) and Chr13: 32912414 (on Assembly GRCh37), which results in a premature truncation of the BRCA2 protein, designated as p.E1308* or p.Glu1308Ter. The substitution is predicted to result in a non-functional BRCA2 protein, either through protein truncation or nonsense-mediated mRNA decay.

This mutation is considered a non-tolerated amino acid change based on "in silico" prediction algorithms (disease causing), and it has been reported as Pathogenic in the Clinical Variant Database (NCBI National Library of Medicine, NIH, Bethesda MD; 2002, 2012, 2014, 2015, 2016), including the 2016 report from the Evidence-based Network for the Interpretation of Germline Mutant Alleles (ENIGMA Consortium).

Although the exact risk of breast and ovarian cancer conferred by this specific variant has not been determined, studies of this type of mutations in high-risk families indicate that deleterious BRCA2 variants may confer as much as a 45% risk of breast cancer and an 11-18% risk of ovarian cancer by age 70 in women (American Journal of Human Genetics 2003; 72(5):1117–1130, Journal of Clinical Oncology 2007; 25(11):1329–1333).

Women with BRCA2 mutations have a 3.4-fold increased risk of developing contralateral breast cancer (Journal of Clinical Oncology 2010; 28(14):2404-10). Additionally, the pancreatic cancer risk by the age 80 is approximately 2.8% for female and 6.9% for male patients (J Med Genet. 2005 Sep;42(9):711-9).

Male breast cancer is very rare, with a life time risk of 1,000 for non-carriers. Nonetheless, the estimate cumulative breast cancer risks for BRCA2 male mutations carriers at age 70 is as high as 7% (J Natl Cancer Inst. 2007; 99(23): 1811–1814, J Med Genet. 2010;47(10):710–1). The risk of prostate cancer is also increased in BRCA2 mutation carriers by 2- to 6-fold by age 70 (Clin Cancer Res. 2009 Feb 1;15(3):1112-20|Clin Cancer Res. 2010 Apr 1;16(7):2115-21|Fam Cancer. 2012 June;11(2):235-42).

Biallelic mutations in BRCA2 are associated with Fanconi anemia subtype D1 (FANCD1), a syndrome that is related to acute myeloid leukemia and childhood solid tumors (Blood. 2004 Apr 15;103(8):3226-9, Pediatr Blood Cancer. 2012 Mar;58(3):462-5).

Cancer risks can be further modified by family history, reproductive choices, lifestyle and environmental factors and other genetic factors.

## ADDITIONAL FINDINGS

Other Variants: Variants of clinical significance are reported. Variants identified as benign or likely benign polymorphisms are not reported. Evidence indicates that these variants do not impact cancer risk.

Current medical opinion recommends against using findings of variants that are not clinically significant to modify patient medical management. Medical management decisions should be made based on personal and family history and any other clinically significant findings.

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## SUMMARY & METHODOLOGY

PREVENTEST® MOLECULAR PANEL is a full risk sequencing of germline mutations involved in familial cancer predisposition. The panel interrogates 35 germline key-cancer predisposition genes, targeting mutational hotspots associated with both common and rare familial cancer syndromes. All translated exons and immediately adjacent intronic regions are sequenced. Single nucleotide polymorphisms, duplications, insertions, deletions, and variants of uncertain significance can be detected. Sequencing analysis on the other genes included in the panel described above are consistent with intron variant and synonymous variant polymorphisms and are considered benign.[6-10]

Genomic DNA from **Sample Patient's** submitted specimen was enriched for the complete coding regions and splice site junctions of the genes described in the panel. The products were sequenced on two different massive parallel sequencing platforms: Miniseq Illumina platform (Clonal Bridge Amplification/Reversible Dye Terminator) and Ion Torrent Platform (Ion Sphere Particles - Chef System/S5XL). The sequences were aligned to reference sequences based on Human Genome build GRCh37/UCSChg19. BRCA-1/BRCA2 concurrent deletion/duplication testing was performed by Multiple Ligation Probe Amplification (MLPA). Fragment analysis and comparative analysis were performed by Coffalyser DB software, v.140701 (MRC-Holland). Sequencing bio-informatics pipelines were analyzed by Illumina VariantStudio v.3.0 and Torrent Suite Software v.4.0.2., respectively. Discrepancies between platforms, if any, were resolved by selective incorporation of chain-terminating dideoxynucleotides (Sanger Sequencing) targeting with specific FWD/REV primer 5' M13 tailed and HPLC purified. All sequence alterations are described according to the Human Genome Variation Society (HGVS) nomenclature guidelines. Genetic data are stored under Variant call format (VCF).[11,12] AMD follows internal policies and ACMG recommendations for variants reporting.[13] Benign and likely benign variants, if present, are not included in this report, but are available upon request.

## COMMENTS & CONCLUSION

Most human cancers are "sporadic" because there is no identifiable inherited gene mutation involved. Those cancers develop as a result of environmental factors, such as carcinogenic cigarette smoke, that randomly induce mutations in cells, leading to uncontrolled growth. Such factors are encountered throughout life and act over a long period of time.

Familial cancers, on the other hand, tend to occur because it is a specific gene with a defined inheritance pattern. Thus, one is born with a preexisting risk factor for cancer, acting as "one strike". Years later another event triggers the cancer growth. Most of the classic familial cancer syndrome involve a tumor suppressor gene with the "two hit" hypothesis. A person inherits one copy of the mutated gene (first hit), but still has another functional copy of this gene on the other chromosome. Sometime later, a mutation wipes out the remaining normal copy (second hit), and the ability to regulate cellular growth is lost. This allows a clone of neoplastic cells to arise and multiple organs can be affected. Thus, familial cancers often involve more than one organ, and affected individuals can have more than one cancer.

PREVENTEST® is a multiple gene panel that includes the most frequent tumor suppressor genes involved in the inheritance of genetic factors increasing cancer risk.

**Conclusion**: Up to date, the BRCA2 mutation detected in this patient meets the requirements to be considered pathogenic.

***Dr. Daniel Cohen, M.D., Laboratory Director***

*This report was electronically signed*

Disclaimer: The accompanying Technical Specifications summary describes the analysis, method, performance characteristics, nomenclature, and interpretive criteria of this test. This test result does not exclude the possibility of other predisposing mutations that have been reported in individuals with increased risk. There are infrequent genetic abnormalities in long homopolymers or highly homologous regions that this test may not detect. This result, however, rules out the majority of abnormalities believed to be responsible for hereditary cancer susceptibility due to mutations on the gene panel described. The classification and interpretation of all variants identified in this assay reflects the current state of scientific understanding at the time this report was issued, and may change as new scientific information becomes available. The interpretation of this test may be impacted if the patient has a hematologic malignancy or an allogeneic bone marrow transplant. This test may be considered investigational by some states. This test and its performance characteristics were determined by Advanced Molecular Diagnostics Laboratory. It has not been reviewed by the U.S. Food and Drug Administration. The FDA has determined that such clearance or approval is not necessary.

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## ENDNOTES AND FURTHER RESEARCH INFORMATION

(1) Antoniou A, Pharoah PD, Narod S, et al. Average risks of breast and ovarian cancer associated with BRCA1 or BRCA2 mutations detected in case series unselected for family history: A combined analysis of 22 studies. American Journal of Human Genetics 2003; 72(5):1117–1130. (2) Chen S, Parmigiani G. Meta-analysis of BRCA1 and BRCA2 penetrance. Journal of Clinical Oncology 2007; 25(11):1329–1333. (3) https://www.ncbi.nlm.nih.gov/books/NBK1247/ (4) https://ghr.nlm.nih.gov/gene/BRCA2. (5) Referenced with permission from the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Genetic/Familial High-Risk Assessment: Breast and Ovarian V.2.2019. © National Comprehensive Cancer Network, Inc 2018. All rights reserved. Accessed [July 03, 2018]. To view the most recent and complete version of the guideline, go online to NCCN.org. NATIONAL COMPREHENSIVE CANCER NETWORK®, NCCN®, NCCN GUIDELINES®, and all other NCCN Content are trademarks owned by the National Comprehensive Cancer Network, Inc. (6) Flicek et al. Nucleic Acids Research 2013 41 Database issue: D48-D55. doi: 10.1093/nar/gks1236. (7) Helga Thorvaldsdóttir, James T. Robinson, Jill P. Mesirov. Integrative Genomics Viewer (IGV): high-performance genomics data visualization and exploration. Briefings in Bioinformatics 2012. (8) Genome Res. 2009 Jul; 19(7):1316-23. doi: 10.1101/gr.080531.108. Epub 2009 Jun 4. (9) Sherry ST, Ward MH, Kholodov M, Baker J, Phan L, Smigielski EM, Sirotkin K. dbSNP: the NCBI database of genetic variation. Nucleic Acids Res. 2001 Jan 1; 29(1):308-11. (10) Fokkema IF, Taschner PE, Schaafsma GC, Celli J, Laros JF, den Dunnen JT (2011). LOVD v.2.0: the next generation in gene variant databases. Hum Mutat. 2011 May; 32(5):557-63. (11) Bio-IT World, Davies, K. Powering Preventative Medicine. Bio-IT World 2011. (13) GenomeWeb DNA Electronics Licenses IP to Ion Torrent. August 2010. (13) 2013 Annual Clinical Genetics Meeting. American College of Medical Genetics and Genomics. Green R, Berg JS, Grody WW et al.

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## CONFIDENTIAL

A **PREVENTEST™ cancer-risk predisposition test** was ordered on your behalf in consult with your health care provider due to factors such as personal and/or family history of cancer which indicated that you may have a change in your DNA (called a "mutation") that increases your risk of one or more kinds of cancer.

**Result: Positive – Risk-increasing mutation found on the BRCA2 gene**
This mutation is associated with Hereditary Breast and Ovarian Cancer Syndrome (HBOC)

**Additional Findings: All other findings not listed are considered benign, and do not increase your risk of cancer.**

Your DNA test revealed a mutation on the BRCA2 gene. This mutation is associated with a condition called Hereditary Breast and Ovarian Cancer Syndrome (HBOC). HBOC increases your risk for more than one form of cancer. There are medical interventions that your doctor and genetic counselor can recommend which may help to reduce your risk significantly. A genetic counselor is a genetics expert who is trained in counseling and providing guidance regarding genetics and genetic conditions. A positive result for a mutation does not mean that you will get cancer, but reflects an increased risk compared to the general population without the mutation.

## RISK STATISTICS

Hereditary Breast and Ovarian Cancer Syndrome (HBOC) has been shown to increase the risk for certain types of cancers. Below are the statistics for the cancers that are most commonly found in patients who have the same mutated gene as you.

| GENDER | CANCER | AGE | RISK |
|---|---|---|---|
| **Females** | Breast Cancer | Up to 70 years | 49% |
| | Contralateral Breast Cancer | Up to 70 years | Increased |
| | Ovarian Cancer | Up to 70 years | 11-18% |
| | Pancreatic Cancer | Up to 80 years | 2.8% |
| | Melanoma | Lifetime | Increased |
| **Males** | Breast Cancer | Lifetime | 7-8% |
| | Prostate Cancer | Lifetime | 2-6 fold increase |
| | Pancreatic Cancer | Up to 80 years | 6.9% |
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## CAN I REDUCE MY RISK OF CANCER?

Individuals with BRCA2 mutations are at a higher risk of being diagnosed with cancer than the general population. Whether this will occur is based on many factors such as lifestyle, diet, and other influences. Ask your healthcare provider and genetic counselor about steps you can take to help reduce your risk of a future cancer diagnosis.

## HOW DO MY RESULTS AFFECT MY FAMILY?

While this test may only include your specific risk for cancer, your family members are likely to have similar risks. If you have a positive result, your first-degree blood relatives (Mother, Father, Sister, Brother, Daughter, Son) have a 50% risk for the same genetic mutation. Your second-degree blood relatives (Grandmother, Grandfather, Aunt, Uncle, Niece, Nephew, Granddaughter, Grandson, as well as Half-Brothers and Half-Sisters) will have a 25% risk for the same mutation, while your third-degree blood relatives (Great Grandmother, Great Grandfather, Great Aunt, Great Uncle, Cousins, etc.) are at a 12.5% risk for the same positive mutation, indicating a higher risk of cancer.

## NEXT STEPS

- Communicate with your doctor to help determine your next steps. If the cancer(s) are in a specialty that your doctor is not an expert, speak to your doctor about specialists in that area who can be of assistance.
- Speaking to a genetic counselor can help you better understand your risks and options. A genetic counselor can help you understand the impacts of a mutation, what your options are, and how to communicate with family members you may want to speak to about genetic cancer risks. They can also help you process the information and deal with any emotional ramifications. Your doctor can help you find a genetic counselor or you can search at www.NSGC.org to find a counselor in your area, or one who provides counseling over the phone.
- If you are considering having children, discuss reproductive options with your doctor, and if needed, a reproductive specialist. When having children, there is a risk of passing the genetic change to them, but options exist to reduce or eliminate this risk.
- Learn about clinical studies that are being done on patients with your genetic conditions. Clinical studies can often provide higher levels of care and cutting-edge options while helping to further the scientific understanding of your condition.
- Connect to others who also have genetic mutations - you are not alone! There are hundreds of thousands of others who also have genetic mutations. Support groups and other resources (see below) can be a great source of support and information.

Though recommendations in this report are suggested, and many are recommended by authoritative organizations, the best course of action is to speak to your physician and relevant specialists to determine your next steps. It is beneficial to find out as much as you can about your family history of cancer and bring that information with you for any discussions, as that information will play a role in identifying the best course of action.

## ADDITIONAL RESOURCES AND SUPPORT

| | |
|---|---|
| National Society of Genetic Counselors | http://www.nsgc.org |
| National Cancer Institute | http://www.cancer.gov/types/breast/hp/breast-ovarian-genetics-pdq |
| Center for Disease Control and Prevention | http://www.cdc.gov/cancer/knowledge/provider-education/genetics/hboc-syndrome.htm |
| FORCE- Facing Our Risk of Cancer Empowered | http://www.facingourrisk.org/get-support/ |
| Genetic Information Nondiscrimination Act | http://www.ginahelp.org |

This document provides an overview of the results for your genetic test for cancer risk. Your doctor has received a more comprehensive report which he/she can share with you. You can also request a full report by sending a request in writing to reporting@geneidlab.com.