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SAMPLE REPORT

Ordered By Contact ID:1815168 Org ID:1 Last, First, MD, PhD, FACMG Ambry **Additional Authorized Recipient:** Last, Doctor MD	**Normal Specimen** Accession #: **00-108875** Type: Blood EDTA (Purple top) Specimen ID: Collected: 04/27/2020 Received: 04/28/2020 **Tumor Specimen** Accession #: **00-108876** Specimen Type: Tissue block Specimen Site: Right ovary Primary Tumor Site: Right ovary Tumor Type: High grade serous carcinoma Tumor Block ID: 1234-2A Collected: 04/27/2020 Received: 04/28/2020	Patient Name: **Last, First** AP2 Order #: 834411 DOB: 01/01/1987 Gender: F MRN #: Indication: Internal Testing

TumorNext®-HRD+CancerNext®: Paired Germline and Tumor Analyses of 11 Genes Associated with Homologous Recombination Repair plus Germline Analyses of 26 Additional Genes Associated with Hereditary Cancer

OVERALL SUMMARY

This individual is heterozygous for a pathogenic mutation in the *ATM* gene. See below for additional information.

SEQUENCING AND DELETION/DUPLICATION RESULTS

GERMLINE ORIGIN

Gene	Variant	Classification/Effect	FDA Approved Therapies
ATM	**p.K2756***	**Pathogenic Mutation**	**None**

Germline Genes Analyzed: *ATM, BARD1, BRIP1, CHEK2, MRE11A, NBN, PALB2, RAD51C, RAD51D, BRCA1, BRCA2, APC, BMPR1A, CDH1, CDKN2A, DICER1, MLH1, MSH2, MSH6, MUTYH, PMS2, PTEN, SMAD4, STK11, TP53, CDK4, NF1, MSH3, NTHL1, RECQL, SMARCA4, AXIN2* (sequencing and deletion/duplication); *POLD1, POLE, HOXB13* (sequencing only); *EPCAM, GREM1* (deletion/duplication only) .

SOMATIC ORIGIN

NO VARIANTS DETECTED

Somatic Genes Analyzed: *ATM, BARD1, BRIP1, CHEK2, MRE11A, NBN, PALB2, RAD51C, RAD51D, BRCA1, BRCA2* (sequencing only) .

INTERPRETATION

Germline Interpretation

- This individual is heterozygous for the **p.K2756*** pathogenic mutation in the *ATM* gene.
- **Risk Estimate:** up to a 4 fold increased risk of female breast cancer and increased lifetime pancreatic and prostate cancer risk.
- The expression and severity of disease for this individual cannot be predicted.
- Genetic testing for pathogenic mutations in family members can be helpful in identifying at-risk individuals.
- Genetic counseling is a recommended option for all individuals undergoing genetic testing.

Somatic Interpretation

- No somatic sequencing mutations or variants of unknown significance were detected in this individual's tumor.

GENE AND ALTERATION INFORMATION

ATM Additional Information

Germline Alteration Information

The **p.K2756*** pathogenic mutation (also known as c.8266A>T), located in coding exon 55 of the *ATM* gene, results from an A to T substitution at nucleotide position 8266. This changes the amino acid from a lysine to a stop codon within coding exon 55. This mutation has been reported in multiple individuals diagnosed with ataxia-telangiectasia (A-T) (Telatar M et al. *Am. J. Hum. Genet.* 1996 Jul;59:40-4; Teraoka SN et al. *Am. J. Hum. Genet.* 1999 Jun;64:1617-31; Buzin CH et al. *Hum. Mutat.* 2003 Feb;21:123-31). This alteration has also been detected in individuals with personal and/or family histories of breast cancer (Maxwell KN et al. *Genet. Med.* 2015 Aug;17:630-8; Aloraifi F et al. *FEBS J.* 2015 Sep;282:3424-37; Desmond A et al. *JAMA Oncol.* 2015 Oct;1:943-51). In addition to the clinical data presented in the literature, this alteration is expected to result in loss of function by premature protein truncation or nonsense-mediated mRNA decay. As such, this alteration is interpreted as a disease-causing mutation.

Gene Information

The *ATM* gene (NM_000051.3), which is classically associated with the autosomal recessive condition ataxia-telangiectasia, is involved in the cellular response to DNA damage and cell-cycle control. Monoallelic pathogenic mutations in this gene have been estimated to confer a 2-4 fold increased risk for female breast cancer compared to the general population; however this risk may be higher for female carriers under the age of 50 (Easton DF. *Int J Radiat Biol.*1994 Dec;66(6 Suppl):S177-82; Thompson D et al. *J Natl Cancer Inst.* 2005 Jun 1;97(11):813-22). In addition, there is evidence that rare missense alterations in *ATM*, especially those affecting highly-conserved residues in the C-terminal functional domains, act as dominant-negative mutations and are associated with higher breast cancer risks than protein truncating mutations (Gatti R et al. *Mol Genet Metab*. 1999 Dec;68(4):419-23; Tavtigian S et al. *Am J Hum Genet.* 2009 Oct;85(4):427-46; Goldgar D et al. *Breast Cancer Res.* 2011 Jul 25;13(4):R73). Monoallelic mutations in this gene have also been reported in patients with prostate cancer and hereditary pancreatic cancer (Roberts NJ et al. *Cancer Discov.* 2012 Jan;2(1):41-6. Epub 2011 Dec 29; Pritchard CC et al. *N. Engl. J. Med.* 2016 Aug;375(5):443-53). Cancer risk estimates for male *ATM* mutation carriers are not currently available. Biallelic pathogenic mutations in the *ATM* gene are known to cause ataxia-telangiectasia (A-T), an autosomal recessive neurodegenerative disorder affecting multiple body systems. Parents who each carry an *ATM* mutation have a 25% chance for a child with A-T in every pregnancy. These risks should be discussed with *ATM* pathogenic mutation carriers of reproductive age. Additionally, pathogenic mutations in genes involved in the homologous recombination repair pathway have been associated with sensitivity to poly(ADP-ribose) polymerase (PARP) inhibitors in preclinical models (Javie M & Curtin NJ. *Ther Adv Med Oncol.* 2011 Nov;3(6):257-67; McCabe N et al. *Cancer Res.* 2006 Aug 15;66(16):8109-15).

ASSAY INFORMATION

General Information: Ovarian cancer is the fifth most common female cancer, with up to 25% being attributed to inherited/germline mutations in cancer predisposition genes. Genes associated with increased ovarian cancer risk include (but are not limited to): *ATM*, *BARD1*, *BRCA1, BRCA2, BRIP1*, *CHEK2*, *NBN*, *PALB2*, *RAD51C*, *RAD51D*. Additionally, germline and somatic mutations in *BRCA1* and *BRCA2* lead to homologous recombination repair deficiency (HRD) and can predict a patient's increased sensitivity and response to treatments, including platinum-based chemotherapy and PARP inhibitors (Kim G et al. *Clin Cancer Res.* 2015 Oct 1;21(19):4257-61; Rose S. *Cancer Discov.* 2017 Feb;7(2):120-121; Tan DS & Kaye SB. *Am Soc Clin Oncol Educ Book*.2015:114-21).

Methodology: Genomic deoxyribonucleic acid (gDNA) is isolated from the patient's specimen(s) using standardized methodology and quantified. For FFPE section, one thin (5 micron) tissue section is first cut and stained with hematoxylin and eosin (H&E). The H&E slide is examined by a pathologist to determine tissue quantity/quality and neoplastic cellularity (20% minimum). Sequence enrichment of the germline and tumor sample for the targeted coding exons and adjacent intronic nucleotides is carried out by a bait-capture methodology using long biotinylated oligonucleotide probes followed by polymerase chain reaction (PCR) and Next-Generation sequencing (NGS). The bioinformatics pipeline performs paired analysis of sequence data from both tumor and germline specimens to differentiate variants of somatic origin from germline origin. Optimized variant calling filters require a read coverage depth of >100X for tumor and > 20X for matched control blood DNA. For molecular analysis of variants of germline origin only, additional Sanger sequencing is performed for any regions missing or with insufficient read depth coverage for reliable heterozygous variant detection. Suspect variant calls of germline origin other than those classified as "likely benign" or "benign" detected on the paired analysis are verified by Sanger sequencing. Germline variants in regions complicated by pseudogene interference, variant calls not satisfying depth of coverage and variant allele frequency quality thresholds, and potentially homozygous variants identified on the hereditary cancer panel are verified by Sanger sequencing. The germline *BRCA2* Portuguese founder mutation, c.156_157insAlu (also known as 384insAlu) and the *MSH2* coding exons 1-7 inversion are detected by next generation sequencing and confirmed by multiplex ligation-dependent probe amplification (MLPA) or PCR and agarose gel electrophoresis. Germline gross deletion/duplication analysis for the sequenced genes (excluding *HOXB13*, *POLD1, POLE,* and *PMS2*) is performed using a custom pipeline based on NGS data and/or targeted chromosomal microarray with confirmatory MLPA when applicable. Sequence analysis is based on the following NCBI reference sequences: *APC*- NM_000038.5 & NM_001127511.2, *ATM*- NM_000051.3, *AXIN2*- NM_004655.3, *BARD1*- NM_000465.2, *BMPR1A*- NM_004329.2, *BRCA1*- NM_007294.3, *BRCA2*- NM_000059.3, *BRIP1*- NM_032043.2, *CDH1*- NM_004360.3, *CDK4*- NM_000075.3, *CDKN2A*- NM_000077.4 and NM_058195.3 (p14ARF), *CHEK2*- NM_007194.3, *DICER1*-NM_177438.2, *HOXB13*- NM_006361.5, *MLH1*- NM_000249.3, *MRE11A*-NM_005591.3, *MSH2*- NM_000251.1, *MSH3*- NM_002439.3, *MSH6*- NM_000179.2, *MUTYH*- NM_001128425.1, *NBN*- NM_002485.4, *NF1*-NM_000267.3, *NTHL1*- NM_002528.5, *PALB2*- NM_024675.3, *PMS2*- NM_000535.5, *POLD1*-NM_002691.2, *POLE*-NM_006231.2, *PTEN*-NM_000314.4, *RAD51C*- NM_058216.1, *RAD51D*- NM_002878.3, *RECQL*- NM_002907.3, *SMAD4*- NM_005359.5, *SMARCA4*-NM_001128849.1, *STK11*- NM_000455.4, *TP53*- NM_000546.4.

Analytical Range: The **TumorNext®-HRD+CancerNext®** Test targets detection of germline and somatic variants in genes in the homologous recombination repair pathway and other hereditary cancer syndromes. Germline sequencing analysis is performed for 35 genes (*APC, ATM, AXIN2 BARD1, BMPR1A, BRCA1, BRCA2, BRIP1, CDH1, CDK4, CDKN2A, CHEK2, DICER1, HOXB13, MLH1, MSH2, MSH3, MSH6, MUTYH, MRE11A, NBN, NF1, NTHL1, PALB2, POLD1, POLE, PMS2, PTEN, , RAD51C, RAD51D, RECQL, SMAD4, SMARCA4, STK11,* and *TP53*) by either Next-Generation or Sanger sequencing. All coding domains and well into the flanking 5' and 3' ends of all the introns and untranslated regions are analyzed. For *HOXB13,* only alterations affecting p.G84 are analyzed and reported. For *POLD1* and *POLE*, only missense variants and in-frame indel variants in the exonuclease domains (codons 311-541 and 269-485, respectively) are routinely reported. For *RECQL*, only missense variants in the helicase and RCQ domains (codons 63-592) and exonic truncating variants and are routinely reported. The *MSH3* exon 1 repeat region is excluded from analysis. For the germline specimen, gross deletion/duplication analysis determines gene copy number for the covered exons and untranslated regions of all sequenced genes (excluding *HOXB13, POLD1, and POLE*), *GREM1*, and *EPCAM*. For *GREM1,* only the status of the 40kb 5'UTR gross duplication is analyzed and reported. For *EPCAM*, only gross deletions encompassing the 3' end of the gene are reported. For *NTHL1*, only full-gene gross deletions and duplications are detected. For *APC*, all promoter 1B gross deletions as well as single nucleotide substitutions within the promoter 1B YY1 binding motif (NM_001127511 c.-196_-186) are analyzed and reported. Analysis of variants of somatic origin is performed for 11 genes in the homologous recombination repair pathway (*ATM, BARD1, BRCA1, BRCA2, BRIP1, CHEK2, MRE11A, NBN, PALB2, RAD51C,* and *RAD51D)* and has the same analytical range as the germline analysis.

Result Reports:

- **Germline Sequencing and Del/dup Analysis and Tumor Sequencing:** Alterations reported to be of germline origin are presumed present in the tumor unless otherwise noted on the final report. Alterations in the following classifications are always reported, and are based on the following definition and clinical recommendations:
  - **Pathogenic Mutation:** Alterations with sufficient evidence to classify as pathogenic (capable of causing disease). Target testing of at-risk relatives and appropriate changes in medical management for germline pathogenic mutation carriers recommended. Previously described pathogenic mutations, including intronic mutations at any position, are always reported when detected.
  - **Variant, Likely Pathogenic (VLP):** Alterations with strong evidence in favor of pathogenicity. Targeted testing of at-risk relatives and appropriate changes in medical management for germline VLP carriers typically recommended. Previously described likely pathogenic variants, including intronic VLPs at any position, are always reported when detected.

- **Variant, Unknown Significance (VUS):** Alterations with limited and/or conflicting evidence regarding pathogenicity. Familial testing via the Family Studies Program recommended. Medical management to be based personal/family clinical histories, not germline VUS carrier status. Note, intronic VUSs are always reported out to 5 basepairs from the splice junction when detected.
- Alterations of unlikely clinical significance (those with strong/very strong evidence to argue against pathogenicity) are not routinely included on results reports. These include findings classified as “likely benign” and “benign” alterations.
- Additional variants of somatic origin may have also been detected and can be released upon request.

- **Therapeutic Information:** Only those FDA approved PARP inhibitor therapies are reported, which are indicated in epithelial ovarian cancer, and this list is not comprehensive Not all FDA-approved therapies may be reported.

Assay Information Continued on Next Page

ASSAY INFORMATION *(Supplement to Test Results - Continued)*

Resources: The following references are used in variant analysis and classification when applicable for observed genetic alterations.

1. The 1000 Genomes Project Consortium. An integrated map of genetic variation from 1092 human genomes. *Nature*. 2012;491:56-65.
2. ACMG Standards and guidelines for the interpretation of sequence variants. *Genet Med.* 2015 May;17(5):405-23.
3. Ambry Genetics Variant Classification Scheme. http://www.ambrygen.com/variant-classification.
4. Berkeley Drosophila Genome Project [Internet]. Reese MG et al. *J Comp Biol.* 1997;4:311-23. http://www.fruitfly.org/seq_tools/splice.html.
5. Database of Single Nucleotide Polymorphisms (dbSNP) [Internet]. Bethesda (MD): National Center for Biotechnology Information, National Library of Medicine (dbSNP Build ID:135) Available from: www.ncbi.nlm.nih.gov/SNP. Accessed Jan 2012).
6. ESEfinder [Internet]. Smith PJ, et al. (2006) *Hum Mol Genet.* 15(16):2490-2508 and Cartegni L, et al. *Nucleic Acid Research.* 2003;31(13):3568-3571. http://rulai.cshl.edu/cgi-bin/tools/ESE3/esefinder.cgi?process=home.
7. Exome Variant Server, NHLBI Exome Sequencing Project (ESP) [Internet], Seattle WA. Available from: evs.gs.washington.edu/EVS.
8. Grantham R. Amino acid difference formula to help explain protein evolution. *Science.* 1974;185(4151):862-864.
9. HGMD® [Internet]: Stenson PD et al. *Genome Med.* 2009;1(1):13. www.hgmd.cf.ac.uk.
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11. Online Mendelian Inheritance in Man, OMIM®. McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University (Baltimore, MD), Copyright® 1966-2012. World Wide Web URL: http://omim.org.
12. Feng BJ. PERCH: A Unified Framework for Disease Gene Prioritization. *Hum Mutat.* 2017 Mar;38(3):243-251.
13. Exome Aggregation Consortium (ExAC) [Internet], Cambridge, MA. Available from: http://exac.broadinstitute.org.
14. Genome Aggregation Database (gnomAD) [Internet], Cambridge, MA. Available from: http://gnomad.broadinstitute.org.
15. Lek M et al. Analysis of protein-coding genetic variation in 60,706 humans. *Nature*. 2016 Aug 17;536(7616):285-91. PMID: 27535533
16. Mu W et al. *J Mol Diagn*. 2016 Oct 4. PubMed PMID: 27720647

Disclaimer: This test was developed and its performance characteristics were determined by Ambry Genetics Corporation. DNA isolation, NGS and Sanger sequencing, and deletion/duplication analyses are performed at Ambry Genetics 7 Argonaut, Aliso Viejo, CA (CLIA# 05D0981414). Pathology review of stained slides is performed at Ambry Genetics 15 Argonaut, Aliso Viejo, CA (CLIA#: 05D2115857). It has not been cleared or approved by the US Food and Drug Administration. The FDA does not require this test to go through premarket FDA review. It should not be regarded as purely investigational or for research. This test should be interpreted in context with other clinical findings. This report does not represent medical advice. Any questions, suggestions, or concerns regarding interpretation of results should be forwarded to a genetic counselor, medical geneticist, or physician skilled in interpretation of the relevant medical literature. This laboratory is certified under the Clinical Laboratory Improvement Amendments (CLIA) as qualified to perform high complexity clinical laboratory testing. The sequencing and germline gross deletion duplication portion of this test analyzes the following types of mutations: nucleotide substitutions, small deletions (up to 25 bp), small insertions (up to 10 bp), small indels, gross deletions/duplications, and targeted gross rearrangements known to be associated with somatic tumor development and progression. It is not intended to analyze the following types of mutations: uncharacterized gross rearrangements, deep intronic variations, Alu element insertions, and other unknown abnormalities. The assay is validated to detect single nucleotide variants (SNVs) and short indels with variant frequency at 5% or greater, the variant frequency represents the percentage of sequencing reads from tumor specimen DNA containing the variants and has not been corrected for the estimated fraction of tumor cells in the specimen. The pattern of mutation types varies with the gene tested and this test detects a high but variable percentage of known and unknown mutations of the classes stated. A negative result from the analysis cannot rule out the possibility that the tested individual carries a rare unexamined mutation or mutation in the undetectable group. This test is designed and validated to be capable of detecting >99% of described abnormalities in the genes and chromosome regions represented on the test (analytical sensitivity). The clinical sensitivity of this test may vary widely according to the specific clinical history, histopathological subtypes of each tumor and tumor heterogeneity. Mutations in other genes or the regions not tested by this test can also play a role in tumorigenesis. Although molecular tests are highly accurate, rare diagnostic errors may occur. Possible diagnostic errors include sample mix-up, erroneous paternity identification, technical errors, clerical errors, and genotyping errors. Genotyping errors can result from trace contamination of PCR reactions, rare genetic variants that interfere with analysis, germline or somatic mosaicism, presence of pseudogenes, technical difficulties in regions with high GC content or homopolymer tracts, active hematologic disease, a history of allogenic bone marrow or peripheral stem cell transplant, or from other sources. Rare variants present in the human genome reference sequence (GRCh37.p5/hg19) or rare misalignment due to presence of pseudogenes can lead to misinterpretation of patient sequence data.

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Clinician Management Resource for *ATM*

This overview of clinical management guidelines is based on this patient's positive test result for an *ATM* gene mutation. Unless otherwise stated, medical management guidelines used here are limited to those issued by the National Comprehensive Cancer Network® (NCCN®)[1] in the U.S. Please consult the referenced guideline for complete details and further information.

Clinical correlation with the patient's past medical history, treatments, surgeries and family history may lead to changes in clinical management decisions; therefore, other management recommendations may be considered. Genetic testing results and medical society guidelines help inform medical management decisions but do not constitute formal recommendations. Discussions of medical management decisions and individualized treatment plans should be made in consultation between each patient and his or her healthcare provider, and may change over time.

SCREENING/SURGICAL CONSIDERATIONS	AGE TO START	FREQUENCY
Female Breast Cancer		
Breast awareness • Women should be familiar with their breasts and promptly report changes to their healthcare provider.	18 years old	Periodic and consistent
Clinical Breast Exam	30 years old, or 10 years before the earliest known breast cancer in the family	Every 6-12 months
Breast Screening[1] • Mammography with consideration of tomosynthesis • Consider breast MRI with contrast	40 years old, or 5-10 years before the earliest known breast cancer in the family	Every 12 months
For consideration of risk-reducing mastectomy manage based on family history	Individualized	N/A
Pancreatic Cancer[2]		
No specific screening guidelines exist at this time	N/A	N/A
Prostate Cancer[3]		
No specific screening guidelines exist at this time	N/A	N/A
Other[1]		
Counsel for risk of autosomal recessive condition in offspring • If both parents have an *ATM* mutation, each of their children have a 25% chance to have ataxia telangiectasia	Individualized	N/A

1. NCCN Clinical Practice Guidelines in Oncology®. Genetic/Familial High-Risk Assessment: Breast and Ovarian. V1.2018. Available at nccn.org.
2. Canto MI, *et al*. International Cancer of the Pancreas Screening (CAPS) Consortium summit on the management of patients with increased risk for familial pancreatic cancer. Gut. 2013 Mar;62(3):339-47.
3. Pritchard CC, *et al*. Inherited DNA-Repair Gene Mutations in Men with Metastatic Prostate Cancer. N Engl J Med. 2016 Aug 4; 375(5):443-53.

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Ambry Genetics®

Understanding Your Positive *ATM* Genetic Test Result

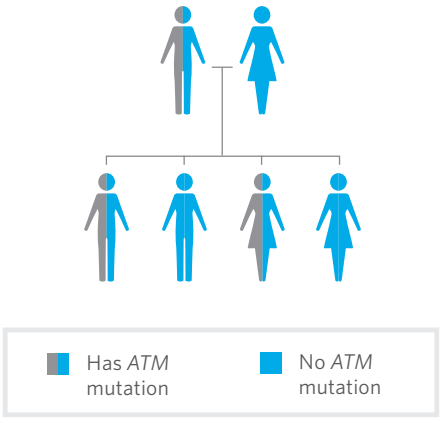
INFORMATION FOR PATIENTS WITH A **PATHOGENIC MUTATION** OR **VARIANT, LIKELY PATHOGENIC**

4 Things To Know

1	*ATM* mutation	Your testing shows that you have a pathogenic mutation or a variant that is likely pathogenic in the *ATM* gene.
2	Cancer risks	You have an increased chance to develop female breast cancer, pancreatic cancer, and possibly other types of cancer.
3	What you can do	There are risk management options to detect cancer early or lower the risk to develop cancer. It is important to discuss these options with your doctor, and decide on a plan that best manages cancer risks.
4	Family	Family members may also be at risk – they can be tested for the *ATM* mutation that was identified in you.

ATM Mutations in the Family

There is a 50/50 random chance to pass on an *ATM* mutation to your sons and daughters. The image to the right shows that both men and women can carry and pass on these mutations.



Understanding Your Positive *ATM* Genetic Test Result

INFORMATION FOR PATIENTS WITH A **PATHOGENIC MUTATION** OR **VARIANT, LIKELY PATHOGENIC**

Result	**MUTATION**	Your testing shows that you have a pathogenic mutation (a disease-causing change in the gene, like a spelling mistake) or a variant that is likely pathogenic in the *ATM* gene. Both of these results should be considered positive.
Gene	***ATM***	Everyone has two copies of the *ATM* gene, which we randomly inherit from each of our parents. Mutations in one copy of the *ATM* gene can increase the chance for you to develop certain types of cancer in your lifetime.
Cancer Risks	**INCREASED**	You have an increased chance to develop female breast cancer (about 2-4 times higher than the average woman), pancreatic cancer, prostate cancer, and possibly other cancers. Some specific *ATM* mutations may cause a higher chance for female breast cancer (up to 52-69%).
Other Medical Concerns	**MAY BE PRESENT**	Individuals with ATM mutations may have an increased risk (25%) to have a child with ataxia telangiectasia, but only if their partner also carries a mutation in the *ATM* gene. Ataxia telangiectasia is a rare condition that can cause enlarged blood vessels under the skin (telangiectasias), uncoordinated movements, and other neurological symptoms.
Management Options	**FOR WOMEN**	Options for early detection and prevention for women may include: breast exam, mammogram, breast MRI, and options for preventive surgery. Talk to your doctor about what options may be right for you.
Management Options	**FOR MEN & WOMEN**	Options for screening and early detection may include pancreatic or other types of cancer screening. Talk to your doctor about what options may be right for you.
Risk Management	**VARIES**	Risk management decisions are very personal, and the best option depends on many factors. Screening typically begins earlier than the general population and is often more frequently performed. It is important to discuss these options with your doctor
Family Members	**50/50 CHANCE**	Your close relatives (like your parents, brothers, sisters, children) have a 50/50 random chance of inheriting the *ATM* mutation that you carry, and other family members (like your aunts, uncles, cousins) may also inherit it. Your relatives can be tested for this same mutation. Depending on the family history, those who DO NOT have it may not have an increased chance (above the general population) to develop cancer.
Next Steps	**DISCUSS**	It is recommended that you share this information with family members so they can learn more and discuss this with their healthcare providers.
Reach Out	**RESOURCES**	• Ambry's Hereditary Cancer Site for Families patients.ambrygen.com/cancer • American Cancer Society cancer.org • FORCE facingourrisk.org • Genetic Information Nondiscrimination Act (GINA) ginahelp.org • National Society of Genetic Counselors nsgc.org • Canadian Society of Genetic Counsellors cagc-accg.ca

Please discuss this information with your healthcare provider. The cancer genetics field is continuously evolving, so updates related to your *ATM* result, medical recommendations, and/or potential treatments may be available over time. This information is not meant to replace a discussion with a healthcare provider, and should not be considered or interpreted as medical advice.

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