RAD51C gene

Associated Syndrome Name: *RAD51C*-associated cancer risk (Women only)

RAD51C Summary Cancer Risk Table

CANCER	GENETIC CANCER RISK
Breast	High Risk
Ovarian	High Risk

RAD51C gene Overview

RAD51C-associated cancer risk (Women only) [1, 2, 3, 4, 5]

- Women with *RAD51C* mutations have an increased risk for ovarian cancer. Although the actual increase in risk is currently estimated to be moderate in size, there are some indications that the risk for ovarian cancer might be higher in families in which there is a past history of ovarian cancer.
- Women with *RAD51C* mutations also have an increased risk for breast cancer. Studies have shown that breast cancers in women with *RAD51C* mutations are more likely to be Triple Negative Breast Cancer (TNBC). This type of breast cancer lacks estrogen and progesterone receptors, and does not express Her2. It can be more aggressive than other types of breast cancer.
- At this time, there are no known cancer risks for men due to mutations in *RAD51C*.
- Although there are high cancer risks for patients with mutations in *RAD51C*, there are interventions that may be effective at reducing these risks. Guidelines from the National Comprehensive Cancer Network (NCCN) that may apply are listed below. Since information about the cancer risks associated with *RAD51C* mutations is relatively new, and there is still some uncertainty about the best ways to reduce these risks, it may be appropriate to interpret these results in consultation with cancer genetics experts in this emerging area of knowledge.

RAD51C gene Cancer Risk Table

CANCER TYPE	AGE RANGE	CANCER RISK	RISK FOR GENERAL POPULATION
Ovarian	To age 50 [3, 6]	0.9%, or higher if there is a family history of ovarian cancer	0.2%
	To age 80 [3, 6]	8%, or higher if there is a family history of ovarian cancer	0.9%
Female Breast	To age 80 [1, 2, 3, 6, 7]	20%, or higher if there is a family history of breast cancer	10.8%

RAD51C Cancer Risk Management Table

The overview of medical management options provided is a summary of professional society guidelines. The most recent version of each guideline should be consulted for more detailed and up-to-date information before developing a treatment plan for a particular patient.

This overview is provided for informational purposes only and does not constitute a recommendation. While the medical society guidelines summarized herein provide important and useful information, medical management decisions for any particular patient should be made in consultation between that patient and his or her healthcare provider and may differ from society guidelines based on a complete understanding of the patient's personal medical history, surgeries and other treatments.

CANCER TYPE	PROCEDURE	AGE TO BEGIN	FREQUENCY (UNLESS OTHERWISE INDICATED BY FINDINGS)
Ovarian	Bilateral salpingo-oophorectomy (BSO).[5]	45 to 50 years, or earlier if there is a family history of ovarian cancer at a younger age	NA

CANCER TYPE	PROCEDURE	AGE TO BEGIN	FREQUENCY (UNLESS OTHERWISE INDICATED BY FINDINGS)
	Other than consideration of BSO, currently there are no specific medical management recommendations for ovarian cancer risk in mutation carriers. However, the increase in risk may warrant consideration of individualized ovarian cancer risk-reduction strategies using other currently available options, such as surveillance and the use of risk-reducing agents.[5]	Individualized	NA
Female Breast	Breast awareness - Women should be familiar with their breasts and promptly report changes to their healthcare provider. Periodic, consistent breast self-examination (BSE) may facilitate breast awareness.[5]	18 years	NA
	Clinical encounter, including clinical breast exam, ongoing risk assessment and risk-reduction counseling[5]	25 years, or 5 to 10 years younger than the earliest age of breast cancer diagnosis in the family	Every 6 to 12 months
	Mammography and consideration of breast MRI with and without contrast[5]	40 years	Annually
For Patients With A Cancer Diagnosis	For patients with a gene mutation and a diagnosis of cancer, targeted therapies may be available as a treatment option for certain tumor types (e.g., PARP-inhibitors).[8]	NA	NA

Information for Family Members

The following information for Family Members will appear as part of the MMT for a patient found to have a mutation in the RAD51C gene.

This patient's relatives are at risk for carrying the same mutation(s) and associated cancer risks as this patient. Cancer risks for females and males who have this/these mutation(s) are provided below.

Family members should talk to a healthcare provider about genetic testing. Close relatives such as parents, children, brothers and sisters have the highest chance of having the same mutation(s) as this patient. Other more distant relatives such as cousins, aunts, uncles, and grandparents also have a chance of carrying the same mutation(s). Testing of at-risk relatives can identify those family members with the same mutation(s) who may benefit from surveillance and early intervention.

In rare instances, an individual may inherit mutations in both copies of the *RAD51C* gene, which may lead to a form of Fanconi anemia (FANCO). This condition has been reported with physical abnormalities, short stature and abnormal chromosome breakage test results. The children of this patient are at risk of inheriting this condition only if the other biological parent is also a carrier of a *RAD51C* mutation. This is highly unlikely, due to the rarity of *RAD51C* mutations.[9, 10]

At this time, there are no known cancer risks for men due to mutations in *RAD51C*.

References

1. Hall, M et al. 2020 Triple-negative breast cancer risk with pathogenic variants in hereditary cancer predisposition genes. Presented at San Antonio Breast Cancer Symposium 2020.
2. Breast Cancer Association Consortium, et al. Breast Cancer Risk Genes - Association Analysis in More than 113,000 Women. N Engl J Med. 2021 384:428-439. PMID: 33471991.
3. Yang X, et a. Ovarian and Breast Cancer Risks Associated With Pathogenic Variants in *RAD51C* and *RAD51D*. J Natl Cancer Inst. 2020 112:1242-1250. PMID: 32107557.
4. Shimelis H, et al. Triple-Negative Breast Cancer Risk Genes Identified by Multigene Hereditary Cancer Panel Testing. J Natl Cancer Inst. 2018 110:855-862. PMID: 30099541.
5. Daly M et al. NCCN Clinical Practice Guidelines in Oncology®: Genetic/Familial High-Risk Assessment: Breast, Ovarian, Pancreatic, and Prostate. V 3.2025. Mar 6. Available at https://www.nccn.org.

6. SEER*Explorer: An interactive website for SEER cancer statistics [Internet]. Surveillance Research Program, National Cancer Institute. [Cited 2025 Apr 1]. Available from https://seer.cancer.gov/explorer/.

7. Breast Cancer Association Consortium, et al. Pathology of Tumors Associated With Pathogenic Germline Variants in 9 Breast Cancer Susceptibility Genes. JAMA Oncol. 2022 8(3):e216744. PMID: 35084436.

8. Schaeffer E, et al. NCCN Clinical Practice Guidelines in Oncology®: Prostate Cancer. V 1.2025. Dec 4. Available at https://www.nccn.org.

9. Vaz F, et al. Mutation of the *RAD51C* gene in a Fanconi anemia-like disorder. Nat Genet. 2010 42:406-9. PMID: 20400963.

10. Blombery P, et al. Utility of clinical comprehensive genomic characterization for diagnostic categorization in patients presenting with hypocellular bone marrow failure syndromes. Haematologica. 2021 Jan 1;106(1):64-73. PMID: 32054657.

Last Updated on 03-Jun-2025