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Web Exclusive: Cancer Biology & Genetics

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Many cancers begin as a fluke mutation in a single cell, but in less than 15 percent of cancer cases one or more mutations are passed from one generation to the next. Well-known hereditary mutations, such as BRCA1 and BRCA2 in breast and ovarian cancer, familial adenomatous polyposis in colorectal cancer and von Hippel-Lindau disease in kidney cancer, can either wreak havoc in each generation or pass silently through families.

Hereditary mutations are present in every cell, including egg and sperm, and can thus be passed to offspring. These germline mutations are often defects in tumor suppressor genes, which help to regulate cell growth, repair DNA mutations or kill off cells that have defective DNA that cannot be repaired. When the tumor suppressor gene doesn't work properly in someone with an inherited mutation, abnormal cell growth can increase dramatically.

Each person has 46 chromosomes—23 from each parent—which contain 25,000 to 30,000 genes. These chromosome pairs allow for a backup, so a mutated gene's healthy counterpart can code for the correct protein. For cancer to develop, both copies of the gene must be mutated.

Inherited mutations will only make a person susceptible to cancer. Unfortunately, because the mutation is in every cell, the healthy gene will need to mutate in only one cell to jumpstart cancer or lose the ability to suppress cancerous mutations from occurring in other genes. Because we are constantly bombarded with carcinogens (cancer-causing substances) and DNA-damaging substances, the probability of a second mutation is high.

With the BRCA genes, the risk of developing cancer can be as high as 85 percent. For other mutations, such as FAP, the risk is even greater. Researchers are developing ways to use a person's genetic information to protect them from cancer before it even starts.

For more information about cancer and genetics, go to the National Cancer Institute's [website](#).