Cancer Families

However, cancer is nowaday quite common disease, just less than 10% of cases are connected with some inherited genes and are trasmitted through the generations. Cancer in older people (older than 65 years) is common and it is usually not connected with family history, because the **early disease outbreak** is typical for cancer families.

There is a special type of mutation connected to the families - **germline mutation**. Mutation is the way how a normal cell transforms into a cancer cell, however, there should be more than one mutation for cancer outbreak. Some special groups of genes have to be affected, we called them oncogenes and tumour suppressor genes.

Each of our body´s cells was created from the mother one, which resulting from the connection of sperm and egg during fertilization. In germline type of mutation this mother cell includes the affected genetic information, so thanks to a mitosis, all of the cells going to have a change, which leads to a cancer. And this cancer pattern is passed from the generation to the next one.

Characteristics of Familial Cancers

1. Early onset.
2. Positive family history – more than two affected close relatives.
3. Multiple incidence of tumours.

Family-Transmitted Cancer

Hereditary Non-polyposis Colorectal Cancer (HNPCC)

HNPCC is the most common hereditary disease of bowel. For HNPCC is typical an increased risk to another cancer too - cancer of the ovaries, womb, stomach, bladder or pancreas. This connection we called **Lynch syndrome** and we need to think about it in person affected by HNPCC, especially women.

HNPCC is caused by the mutation of **mismatch repair genes**, which usually protect us from a cancer mutation. Each of us has two copies of these genes (one from mother and one from father) and their **inheritance is dominant**, so if just one of them is faulty and the second one is in order, we still suffer from HNPCC. The next generation of affected person has 50% risk of HNPCC.

Breast/Ovarian Cancer

About 3/4 of breast cancer have familiar cause – genes *BRCA1* (about 50%) and *BRCA2* (about 20%). We still do not know how it exactly works, but there are few facts, which can help us to decide if the origin is familiar or sporadic (non-familiar): developing before menopause, bilateral focuses and positive family history .

Pancreatic Cancer

Pancreatic cancer has one of the highest mortality rates at all. The incidence of familiar form is not as high as the sporadic one, but it still increases. The mutation of *PRSS1* trypsinogen leads to pancreatitis, which is one of the risk factor of pancreatic cancer.

Brain Cancer

Familial Adenomatous Polyposis (FAP)

FAP also runs in families, although it is just a **predisposition for the bowel cancer**. The bowel is covered by hundreds or thousands or small polyps (adenomas), which can later transfor into the cancer. The only prevention is a treatment, usually surgical (resection of the affected part of the bowel). The incidence of familial version is 66% and 33% have some somatic mutation.

Diagnosis and Prognosis

DNA analysis is the main method of diagnosis in cancer families. **Predicting testing** is a situation, when some member of a family suffer from cancer and we know the original mutation. Then we can test also other family members before they have any symptoms. It does not take so much time, because we do not have to investigate whole genome if we know the possition of gene. The possitive result leads to increased risk. The genetic counseling is recommended to cancer families, because the earlier we find the carriers of mutation, the more effective treatment will be.

Links

Related articles

- Mutation
- Oncogenes
- Tumour Suppressor Genes
- Gametogenesis
- Genetic Counseling
- Lynch Syndrome
- Laws of Inheritance
- Mismatch Repair Genes
- DNA Diagnostic Direct Methods
- DNA Diagnostic Indirect Methods

External links

- Cancer support (http://www.macmillan.org.uk/Cancerinformation/Causesriskfactors/Genetics/Cancergenetics/Cancergenetics.aspx)

Bibliography

- KUMAR,, et al. *Robbins Basic Pathology.* 8th edition. 2007. ISBN 978-0-8089-2366-4.