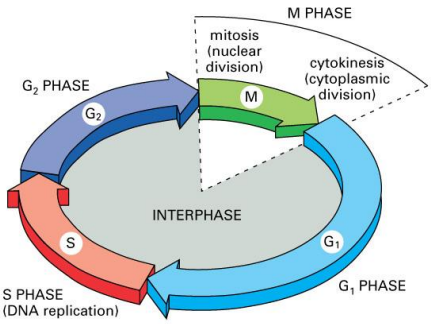
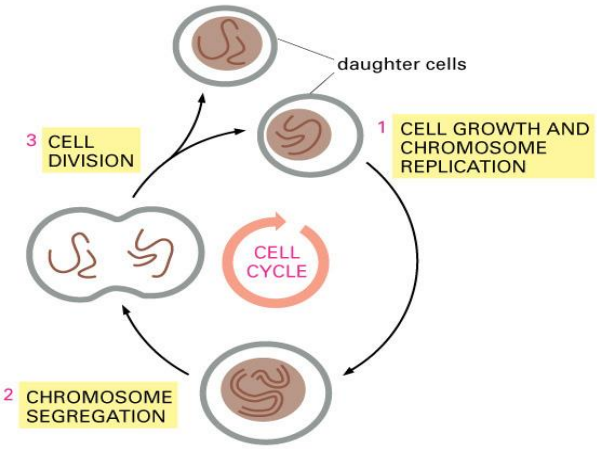
The Cell Cycle

- G_1 phase: cell prepares for DNA synthesis
- S phase: cell generates complete copy of genetic material
- G_2 phase: cell prepares for mitosis
- M phase: replicated DNA is condensed and segregated into chromosomes
- G_0 phase: resting state





Hyperplasia:
New cells produced with growth stimulus via hormones, endogenous signals Ex: hyperplasia of endometrial tissue during menstrual cycle is normal and necessary **Dysplasia**:
Replacement cells disordered in size, shape according mitosis rate Somewhat reversible, often precancerous

Neoplasia:

- abnormal growth/invasion of cells "New growth"
- Neoplasm = tumor
- Irreversible
- Cells replicate, grow without control

Neoplasms

= Tumors = groups of neoplastic cells

How faulty genes lead to cancer

Our genes pick up mistakes that occur when cells divide. These mistakes are called faults or mutations and happen throughout our lives. They are caused by the natural processes in our cells, and by various other factors. These include

- Tobacco smoke
- Radiation

- Ultraviolet radiation from the sun
- Some substances in food
- Chemicals in our environment

Sometimes people inherit certain faulty genes from their parents that mean they have an increased risk of cancer. Usually, cells can repair faults in their genes. If the damage is very bad, they may self destruct instead. Or the immune system may recognise them as abnormal and kill them. This helps to protect us from cancer. But sometimes mutations in important genes mean that a cell no longer understands its instructions, and starts to multiply out of control. It doesn't repair itself properly, and it doesn't die when it should. This can lead to cancer

There are four main types of gene involved in cell division. Most tumours have faulty copies of more than one of these types.

Genes that encourage the cell to multiply (oncogenes)

Oncogenes are genes that, under normal circumstances, play a role in telling cells to start multiplying and dividing. Normally, in adults, this would not happen very often. We can think of oncogenes as being a bit like the accelerator pedal in a car. When they are activated, they speed up a cell's growth rate. When one becomes damaged it is like the accelerator becoming stuck down. That cell, and all the cells that grow from it, are permanently instructed to divide. So a cancer develops.

Genes that stop the cell multiplying (tumour suppressor genes)

Usually, cells can repair faults in their genes. If the damage is very bad, genes called tumour suppressor genes may stop the cell growing and dividing. Mutations in tumour suppressor genes mean that a cell no longer understands the instruction to stop growing and starts to multiply out of control. This can lead to cancer. The best known tumour suppressor gene is p53. The p53 gene is damaged or missing in most human cancers.

Genes that repair other damaged genes (DNA repair genes)

The DNA in every cell in our body is constantly in danger of being damaged. But cells contain many different proteins whose job is to repair damaged DNA. Thanks to these, most DNA damage is repaired immediately, with no ill effects. But if the DNA damage occurs to a gene that makes a DNA repair protein, a cell has less ability to repair itself. So errors will build up in other genes over time and allow a cancer to form. Scientists have found these genes to be damaged in some human cancers, including bowel cancer.

Genes that tell a cell to die (self destruction genes)

Some genes normally tell a cell to self destruct if it has become too old or damaged. This is called apoptosis or programmed cell death. It is a highly complex and very important process. Cells usually die whenever something goes wrong, to prevent a cancer forming. There are many different genes and proteins involved in apoptosis. If these genes get damaged, a faulty cell can survive rather than die and it becomes cancerous.