

# LECTURE 4

❖ **RADIOBIOLOGY**

❖ **RADIOLOGICAL PROTECTION**

Faculty of Health Sciences  
Powiślański University Radiology  
semester II  
Bachelor's degree - nursing

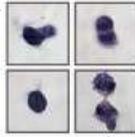


Moderator: Dr. Jan Lica

# "TREE OF LIFE"

## PRIMITIVE CELLS

**SELF-MODERN AND ASYMMETRIC DIVISION**  
 VERY SMALL OR SMALL STEM CELL  
*-HAYFLICK'S LIMIT?-*



Light microscope images. Cells stained with MGG.

**SAMMOODNOWA? AND ASYMMETRIC DIVISION**  
 PNIA CELL  
*-HAYFLICK'S LIMIT?-*



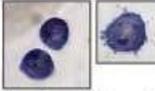
**ASYMMETRIC DIVISION**  
 MULTIPOTENT PROGENITOR  
*-DIVERSITY-*



**ASYMMETRICAL DIVISION**  
 OLIGOIPOTENT PROGENITOR AND  
*-DIVERSITY-*



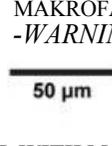
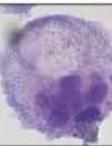
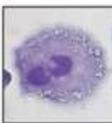
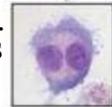
**ASYMMETRICAL DIVISION**  
 OLIGOIPOTENT PROGENITOR II  
*-DIVERSITY-*



**ASYMMETRIC DEPLOIDIZATION**  
 LINEARLY SPECIFIC PRECURSOR - BLAST  
*-DEVELOPMENT-*



**NO SUBJECT.**  
 HEMOPLASMOCYTES  
*-ADOLESCENCE AND AGING TOWARD OSTEOCLASTS-*

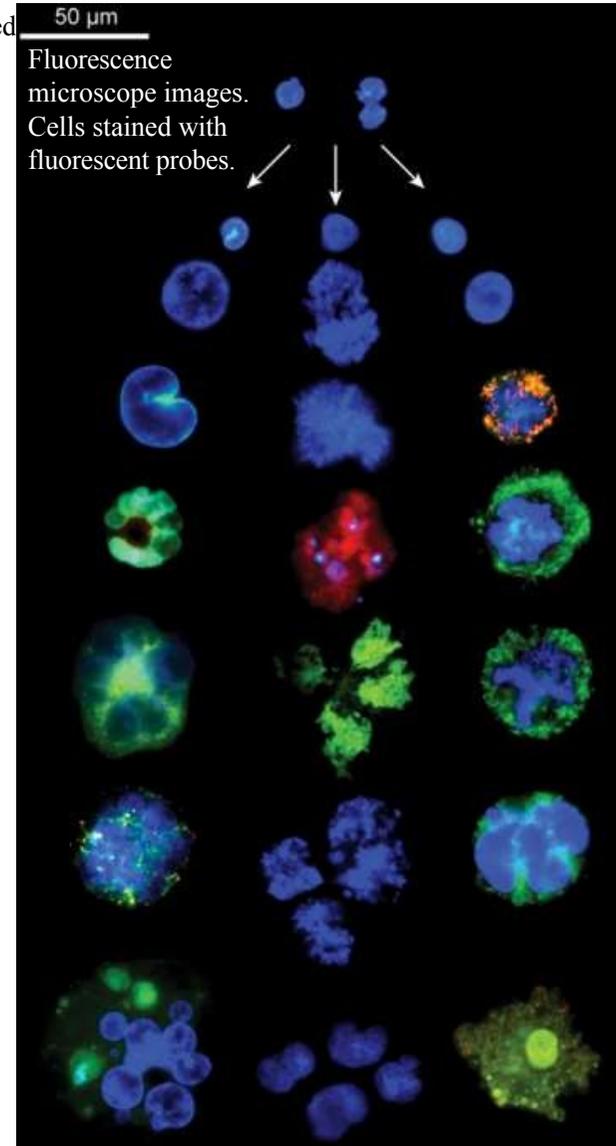


**NO SUBJECT.**  
 MAKROFAG  
*-WARNING-*

50 µm

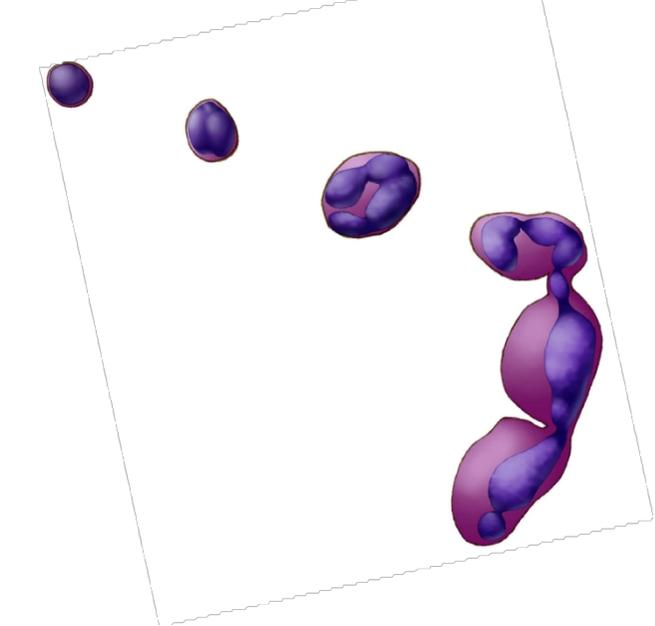
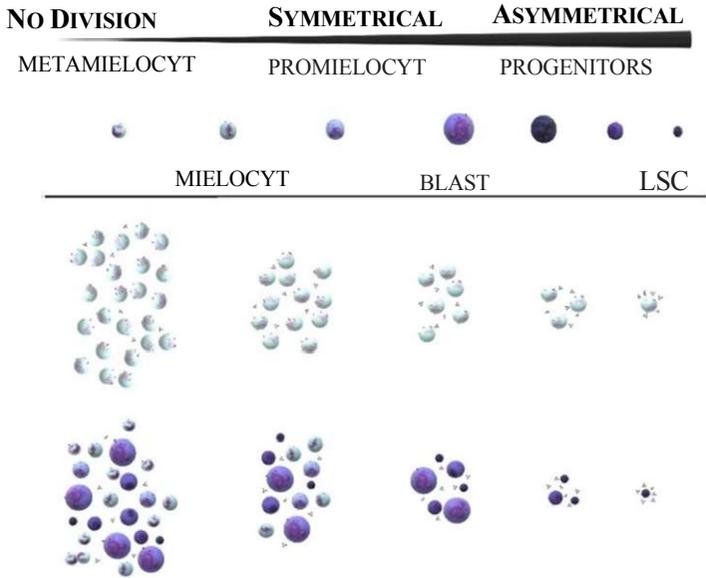
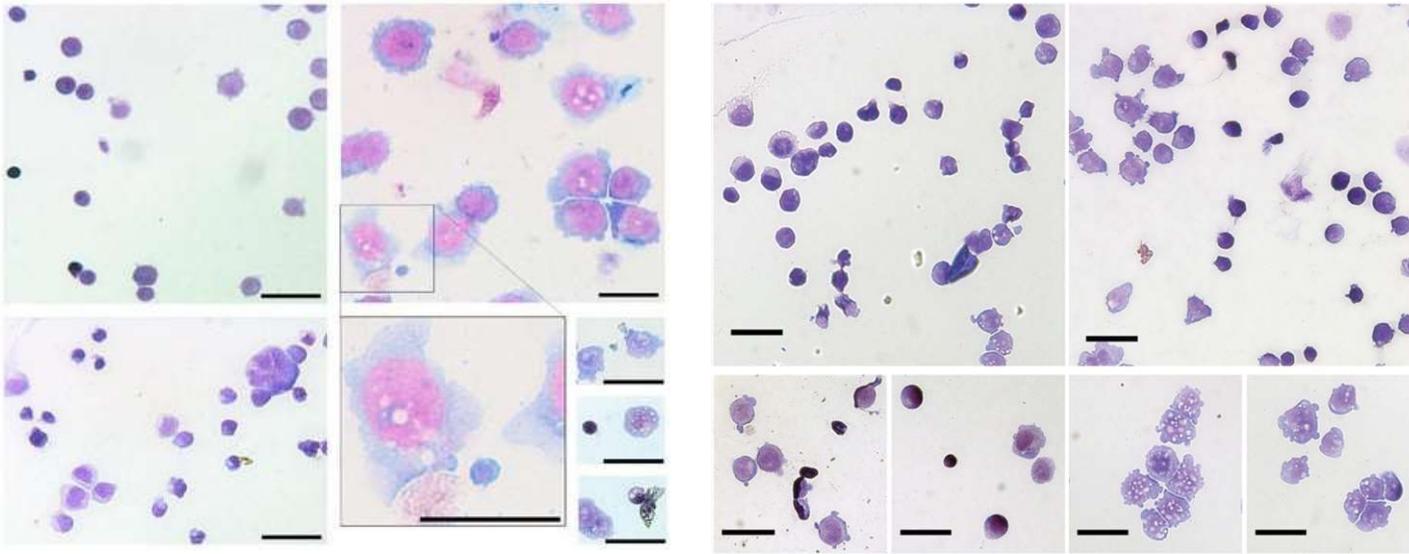
**NON-STOP**  
 MEGACARIOCYT  
*-CARE-*

**SYMMETRICAL WITH MATURATION**  
 GRANULOCYTAR  
*-DEVELOPMENT-*

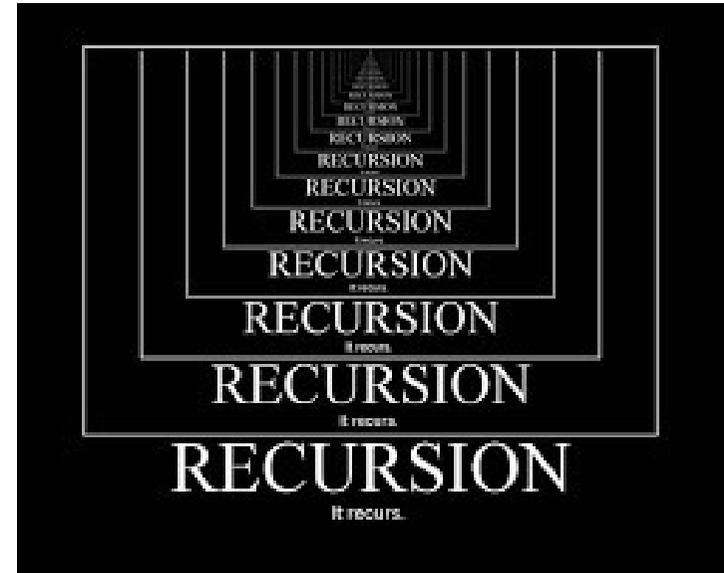


Crawling Neutrophil Chasing a Bacterium  
[https://www.youtube.com/watch?v=I\\_xh-bkiv\\_c&ab\\_channel=AndresTrevino](https://www.youtube.com/watch?v=I_xh-bkiv_c&ab_channel=AndresTrevino)

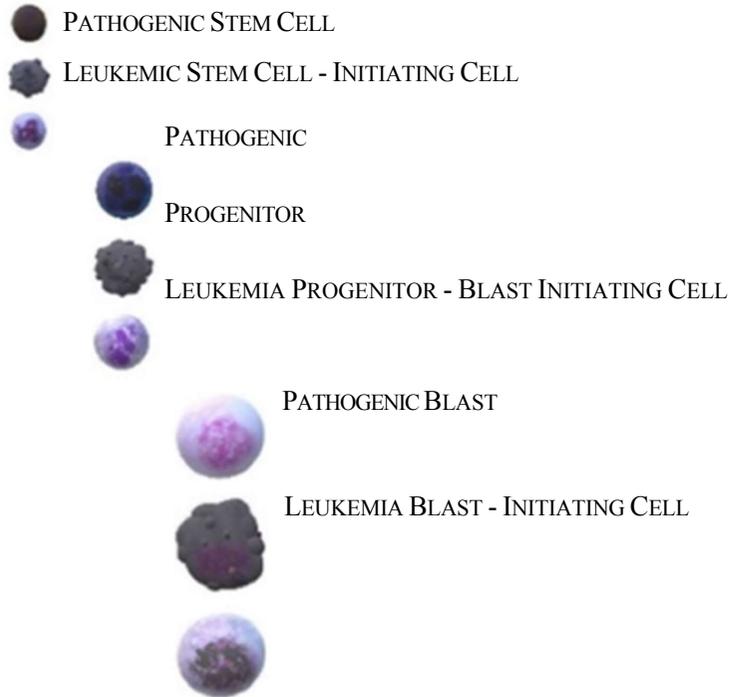
# PANIA CELLS



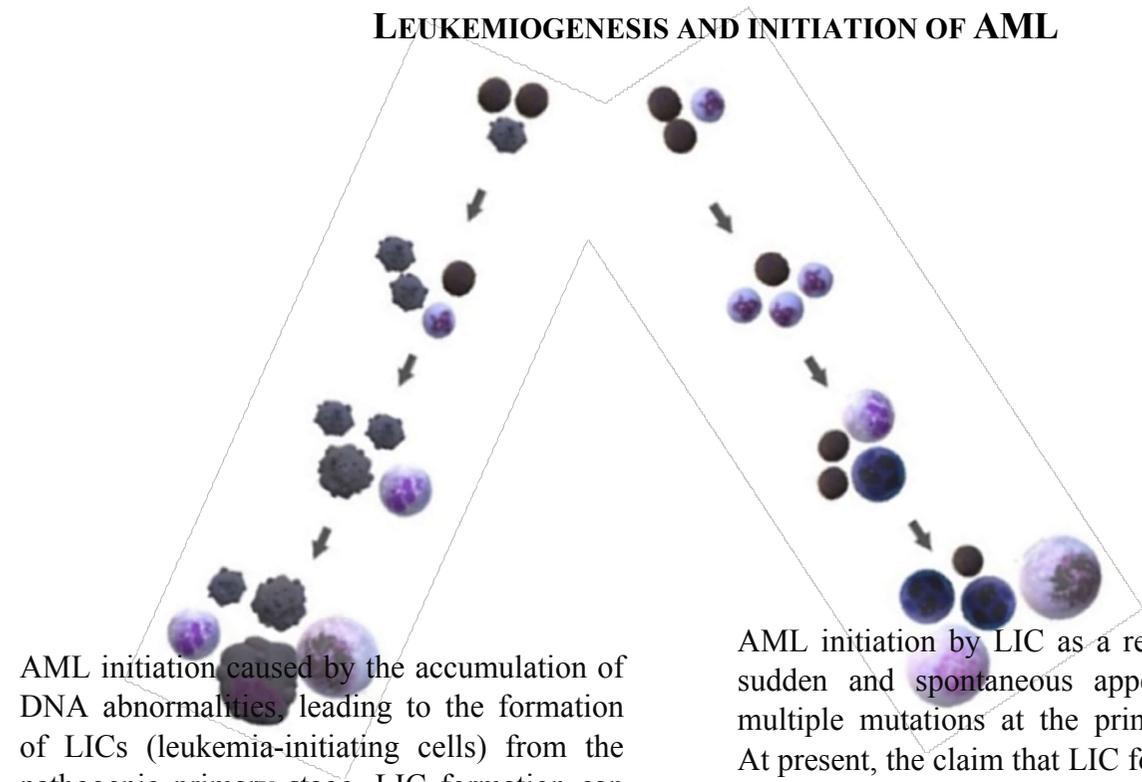
# RECURSION



## DEVELOPMENTAL STAGES OF LEUKEMIA



## LEUKEMIOGENESIS AND INITIATION OF AML

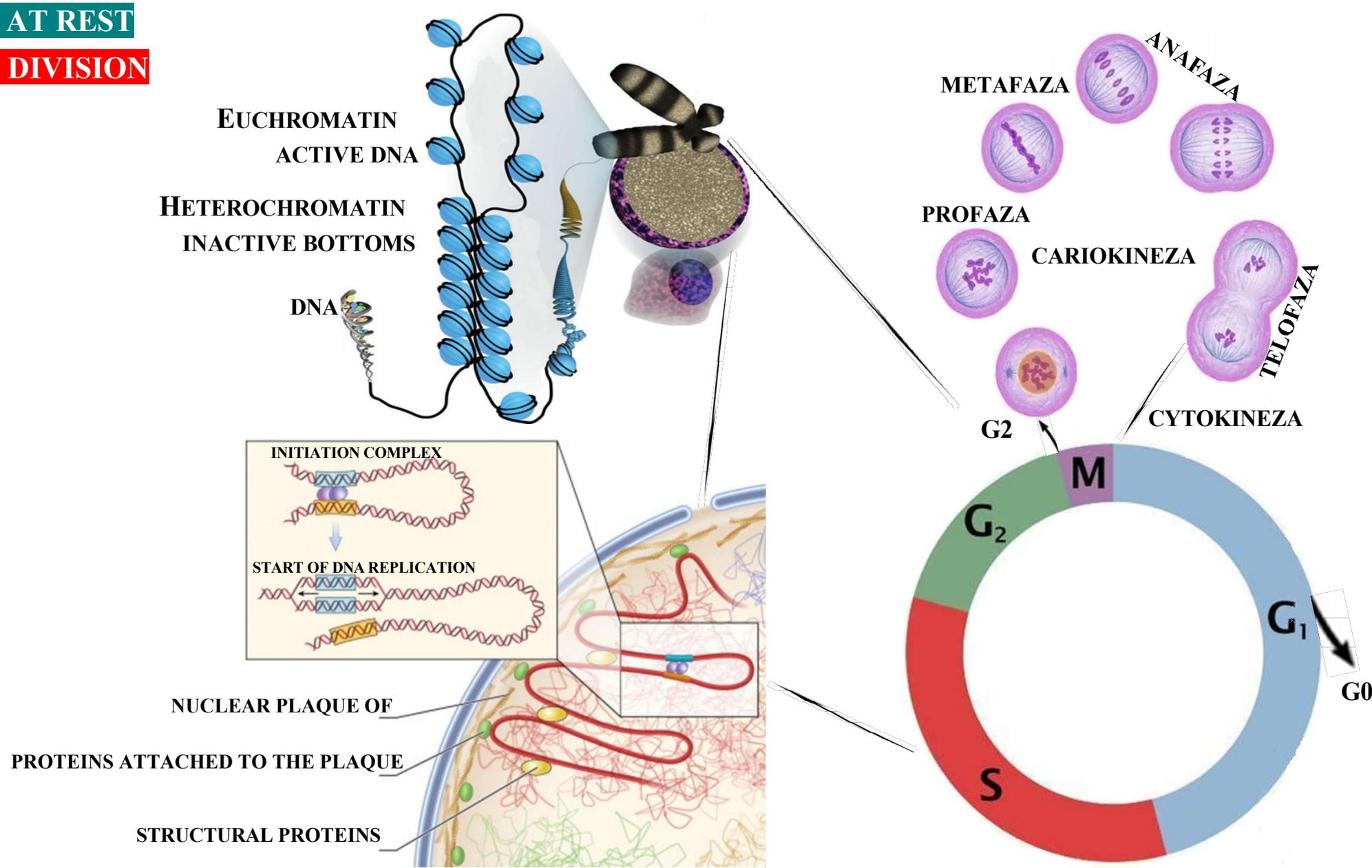


AML initiation caused by the accumulation of DNA abnormalities, leading to the formation of LICs (leukemia-initiating cells) from the pathogenic primary stage. LIC formation can occur independently in different cells and at different stages of their development.

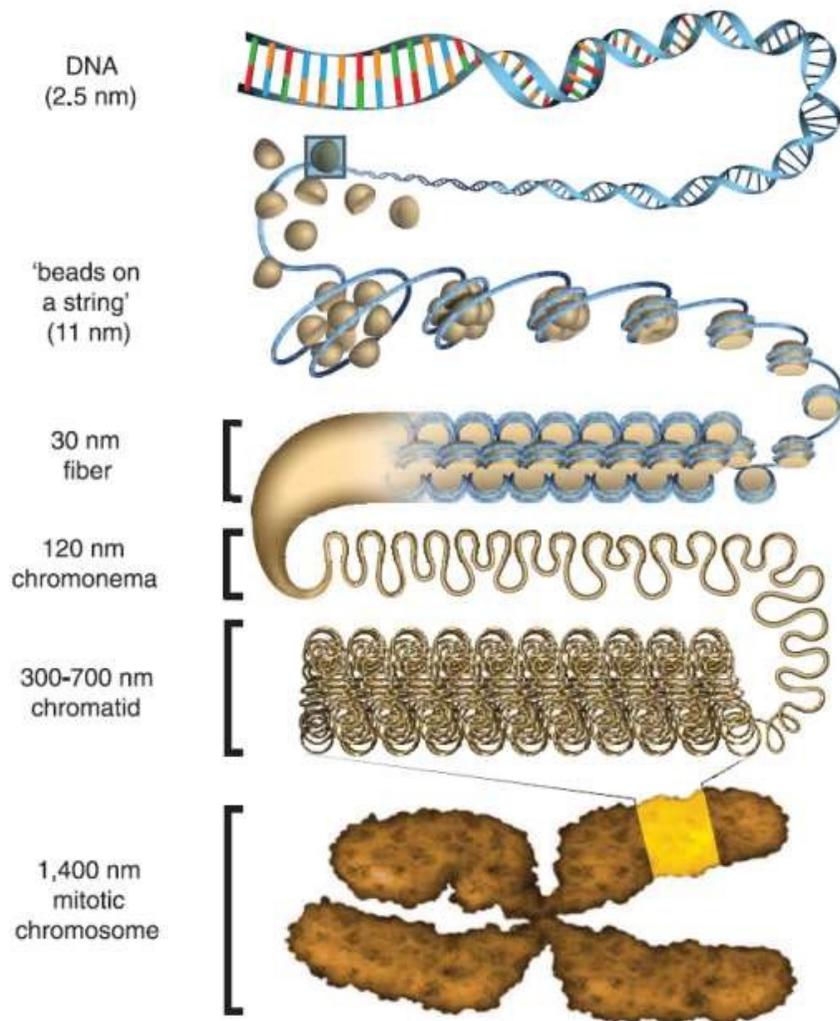
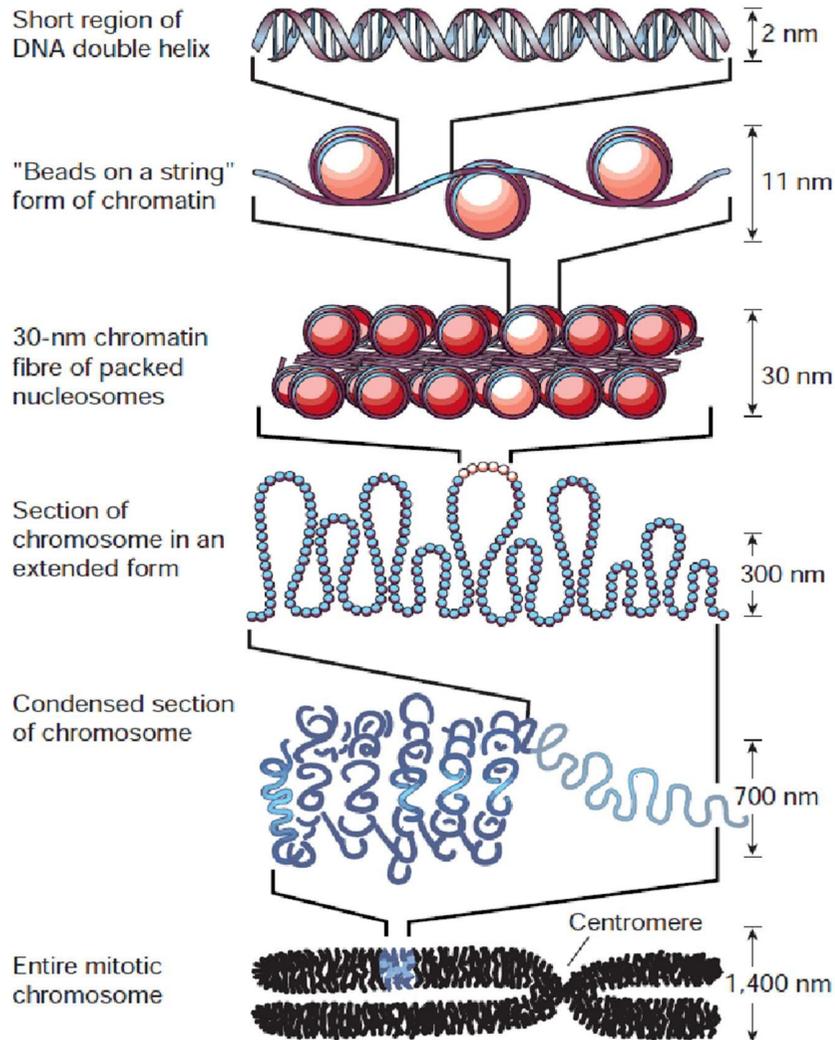
AML initiation by LIC as a result of the sudden and spontaneous appearance of multiple mutations at the primary stage. At present, the claim that LIC formation at a stage other than the knockout cells is debatable.

# BOTTOMS AT REST

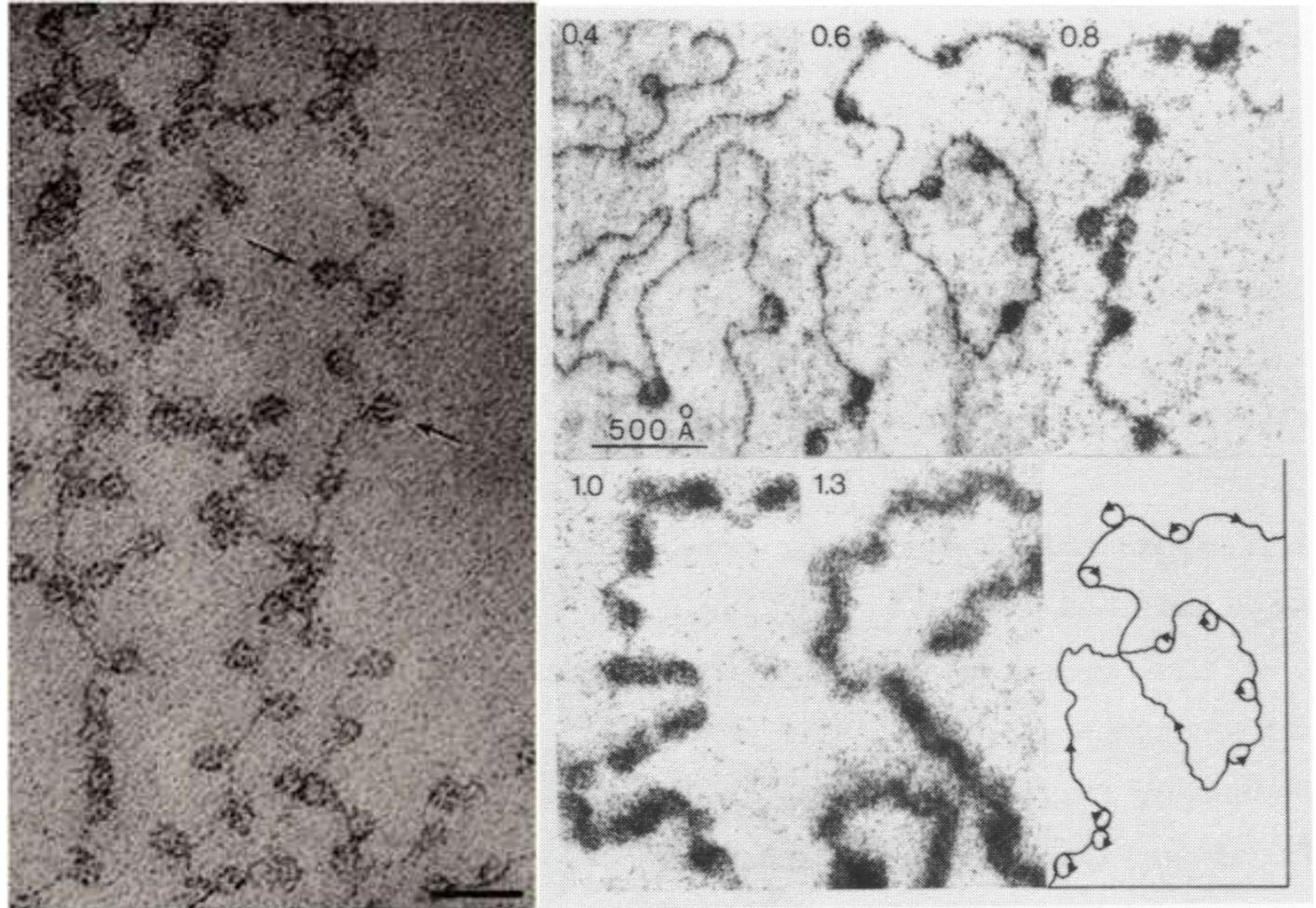
# AND CELL DIVISION



## SPATIAL ORGANIZATION OF THE BOTTOM

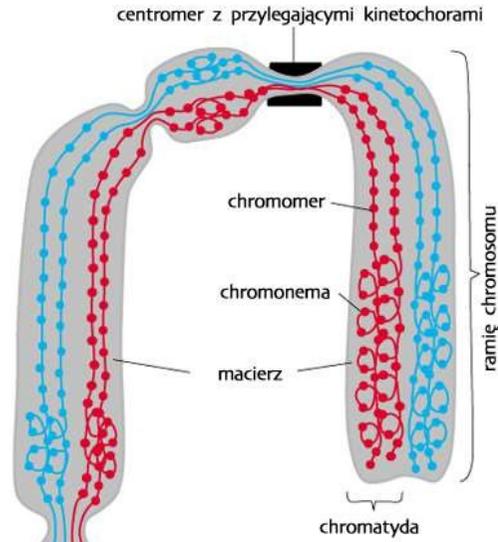
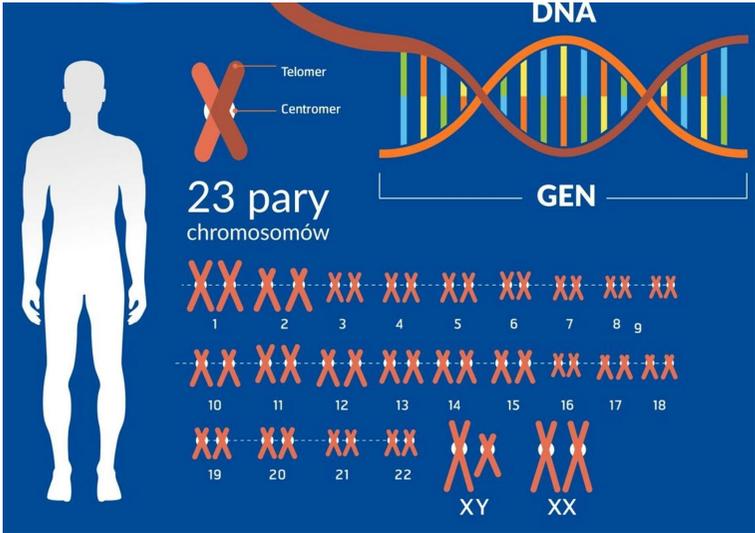


## SPATIAL ORGANIZATION OF THE BOTTOM



**Electron micrograph of chromatin: the beads on a string.** In this micrograph, nucleosomes are indicated by arrows.  
<https://www.nature.com/>

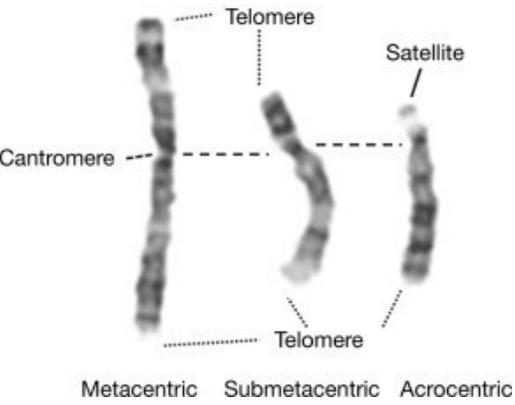
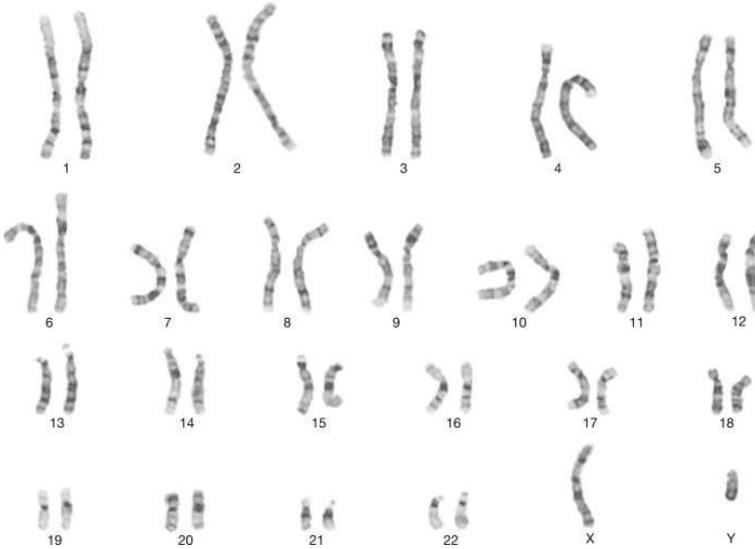
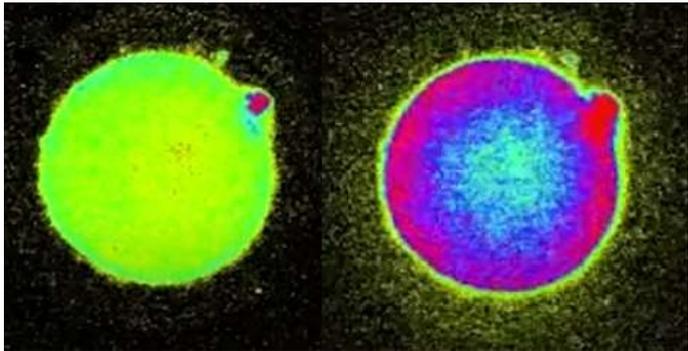
# CHROMOSOME STRUCTURE



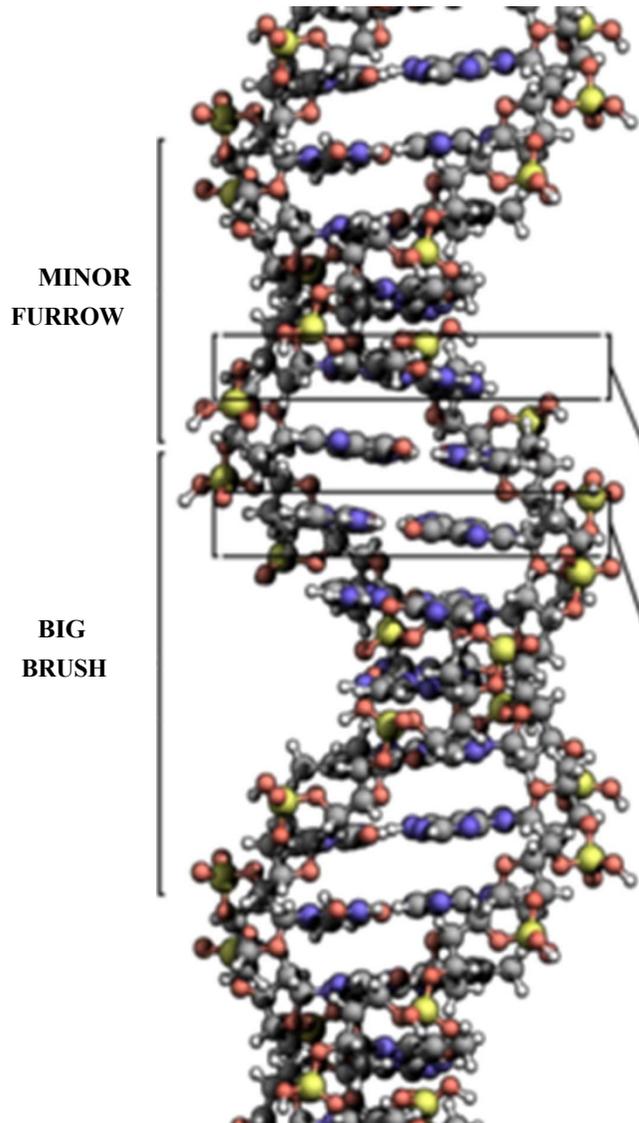
## COMBINATION OF SPERM AND EGG

When Sperm Meets Egg

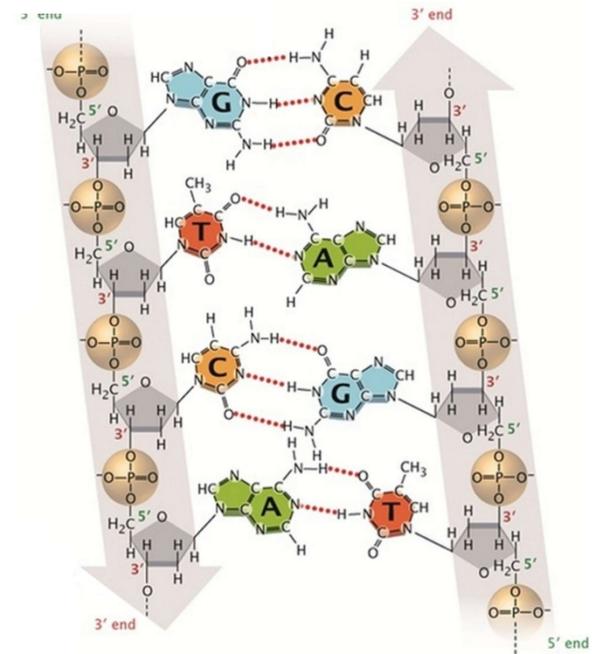
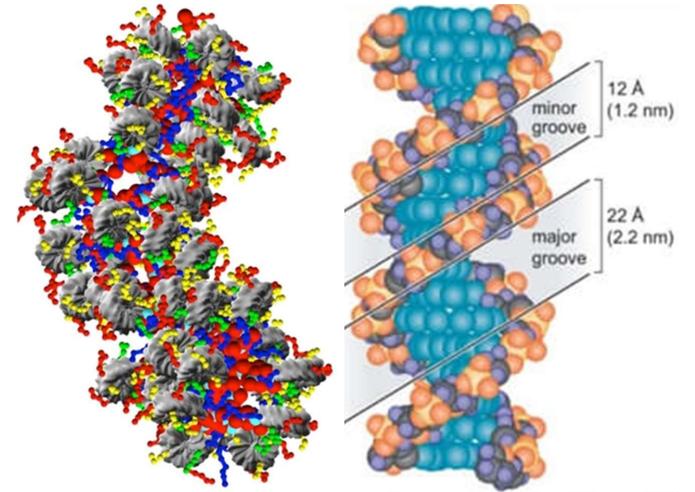
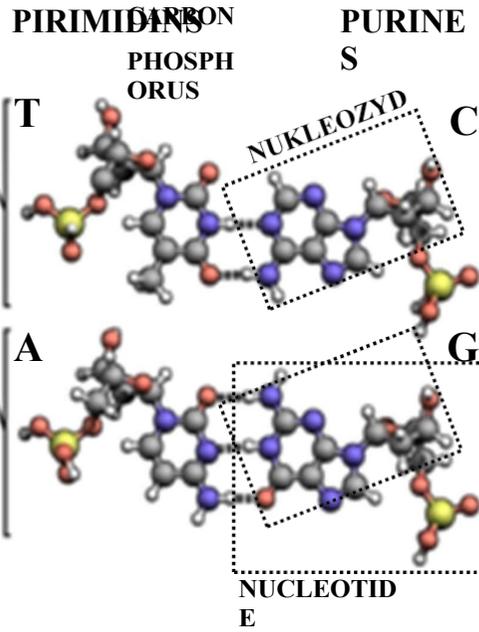
[https://www.youtube.com/watch?v=BJ2x\\_5MSuyg&t=31s&ab\\_channel=Slate](https://www.youtube.com/watch?v=BJ2x_5MSuyg&t=31s&ab_channel=Slate)



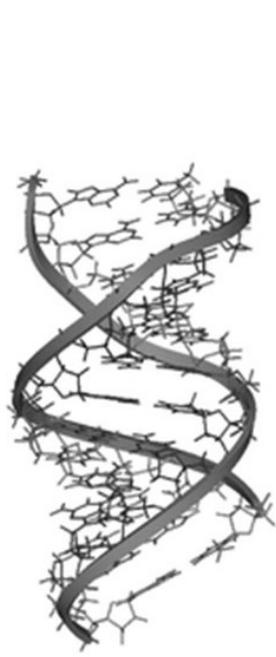
# DNA CONSTITUTION



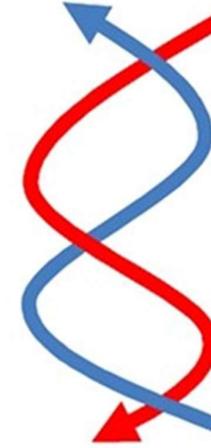
- HYDRO
- GEN
- OXYGE
- N
- NITROG
- EN



# CONFORMATIONAL VARIANTS OF THE BOTTOM



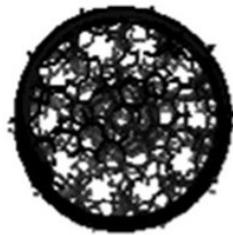
**B-DNA**



**Z-DNA**



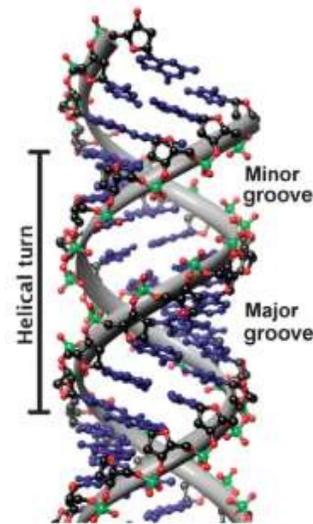
**A-DNA**



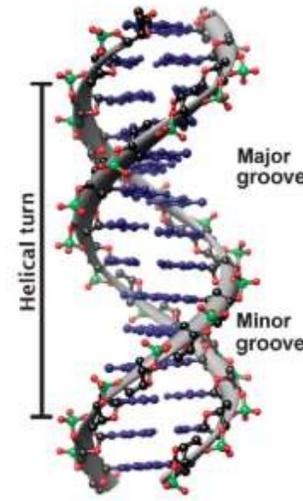
**B-DNA**



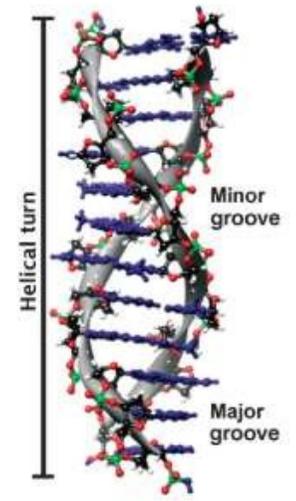
**Z-DNA**



**A-DNA**

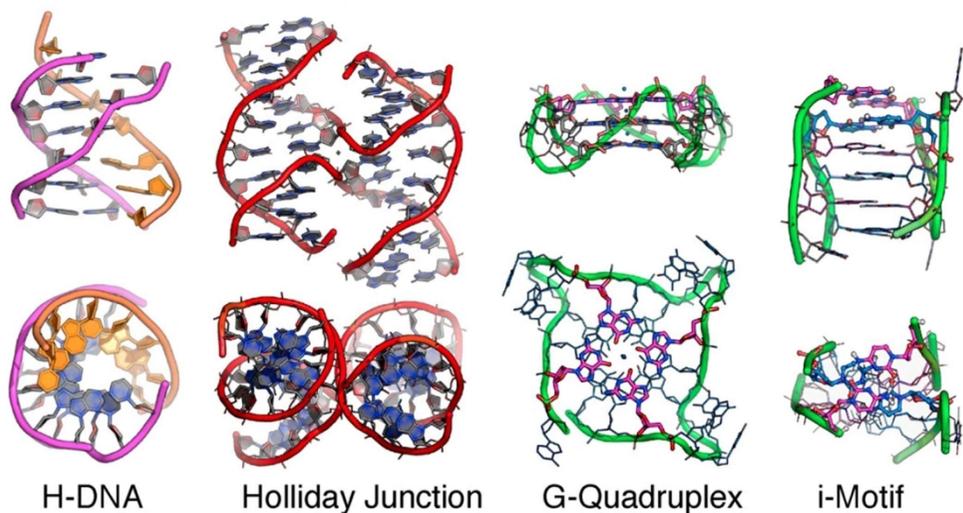
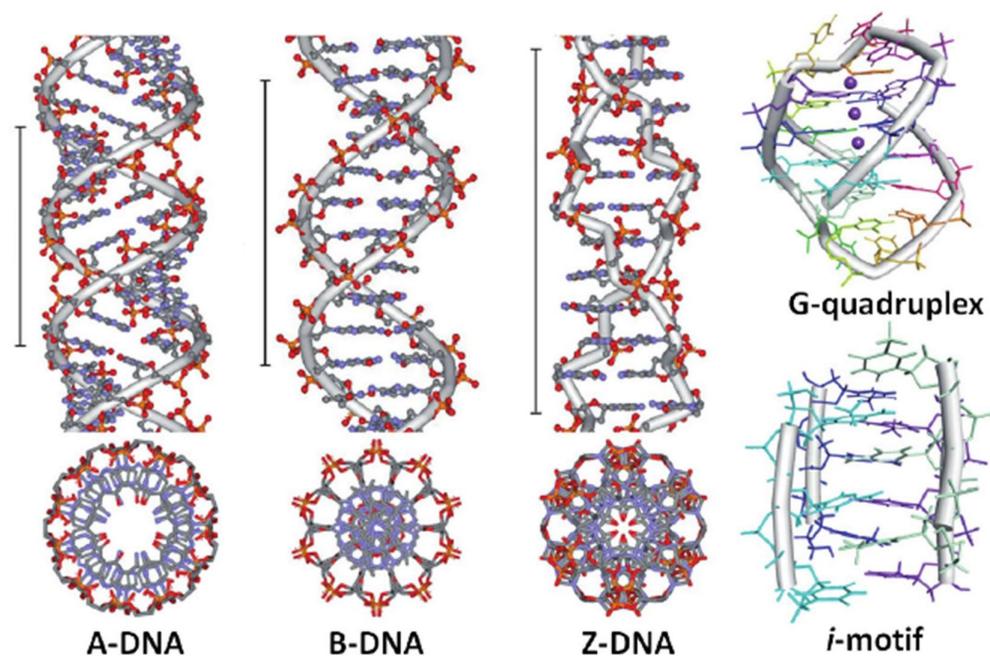


**B-DNA**



**Z-DNA**

## CONFORMATIONAL VARIANTS



### DNA Form A:

The A form is one of the alternative conformations of DNA strands. It is characterized by a greater number of ribose helices compared to the B form, leading to a shortening of the distance between DNA helices. The A-form is often observed in lower moisture conditions, such as crystallization conditions or in association with ligands.

### DNA Form B:

The B-form is the most common conformation of DNA strands under physiological conditions. It is characterized by a helical structure in which the DNA helices are spaced apart in a way that allows access to proteins and other factors that regulate gene expression. It is stable and found in most cells of eukaryotic organisms.

### DNA form Z:

The Z-form is a DNA conformation that features a left-handed helix, as opposed to the B-form, which is right-handed. It can occur in regions of GC-rich sequence, especially under high stress conditions. The biological functions of the Z form are not fully understood, but its potential role in regulating gene expression and DNA recombination has been suggested.

### G-quadruplex DNA:

is a structure that consists of repeated sequences of guanine (G). In this structure, four guanine molecules form a square loop, curling around a central axis. G-quadruplexes can occur at various locations in the genome and serve a variety of biological functions, such as regulating gene expression and stabilizing chromosome ends (telomeres).

### DNA motif I:

is a class of motifs in DNA sequences that contain repeated sequences complexed with guanine (G) and adenine (A). These motifs are often associated with gene promoter regions, binding sites for proteins that regulate gene expression, and other areas that play key roles in regulating biological processes related to gene expression. The "DNA i" motif may have an important function in processes such as transcription, replication and DNA repair.

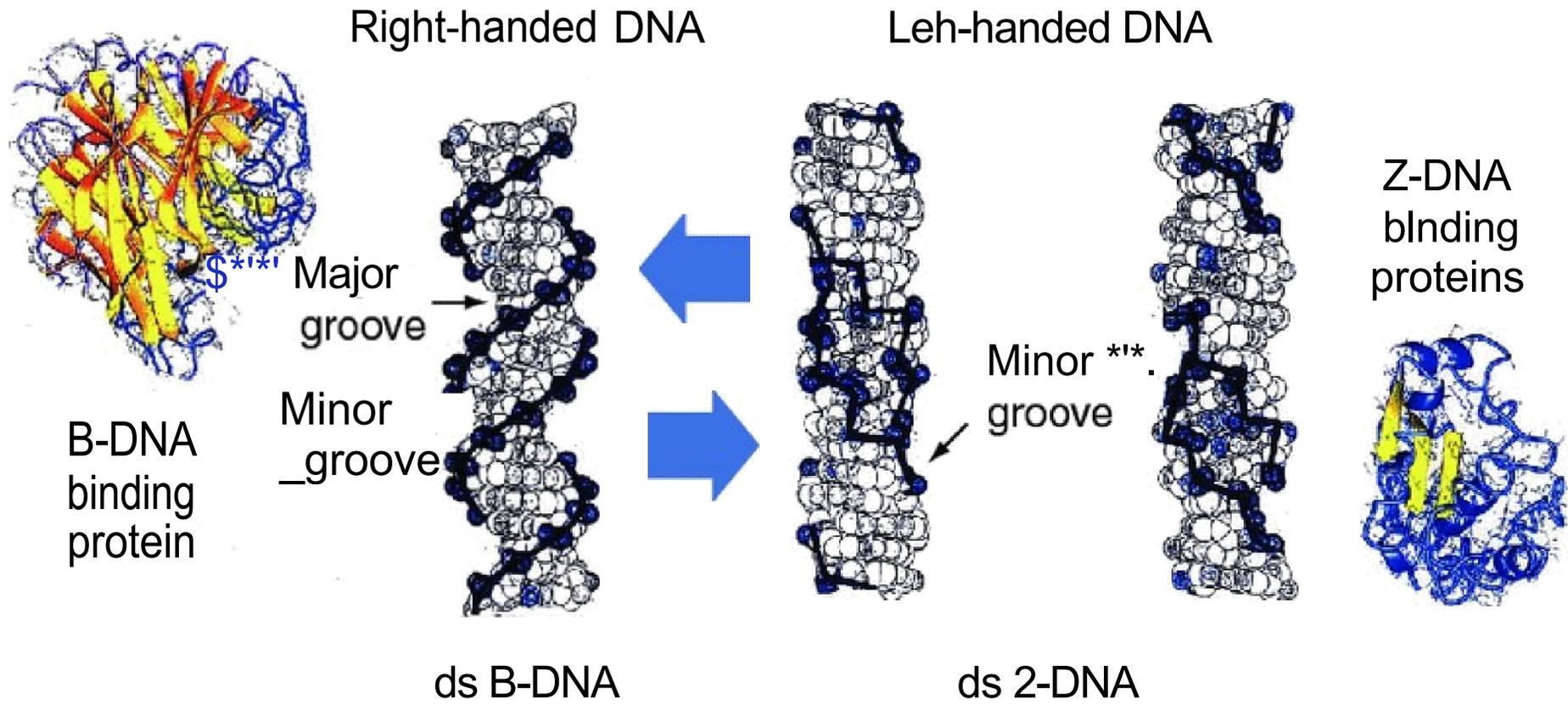
### DNA form H:

H-DNA is a specific structure of triple-stranded DNA, which is a conformational form found in sequences with a high concentration of pyrimidine sequences (Cytosines and Thymines). It is characterized by the fact that fragments of so-called H-DNA motifs will appear in the sequence, which form an unconventional double helix structure. In the H-DNA structure, one of the strands is unusually bent, leading to a three-dimensional structure resembling the letter "H." The H-DNA structure was discovered in the 1970s and is considered one of the conformational forms of DNA. The formation of H-DNA can affect gene expression, DNA replication and other molecular processes. It is also the subject of scientific research to understand its functions and potential importance in cell biology.

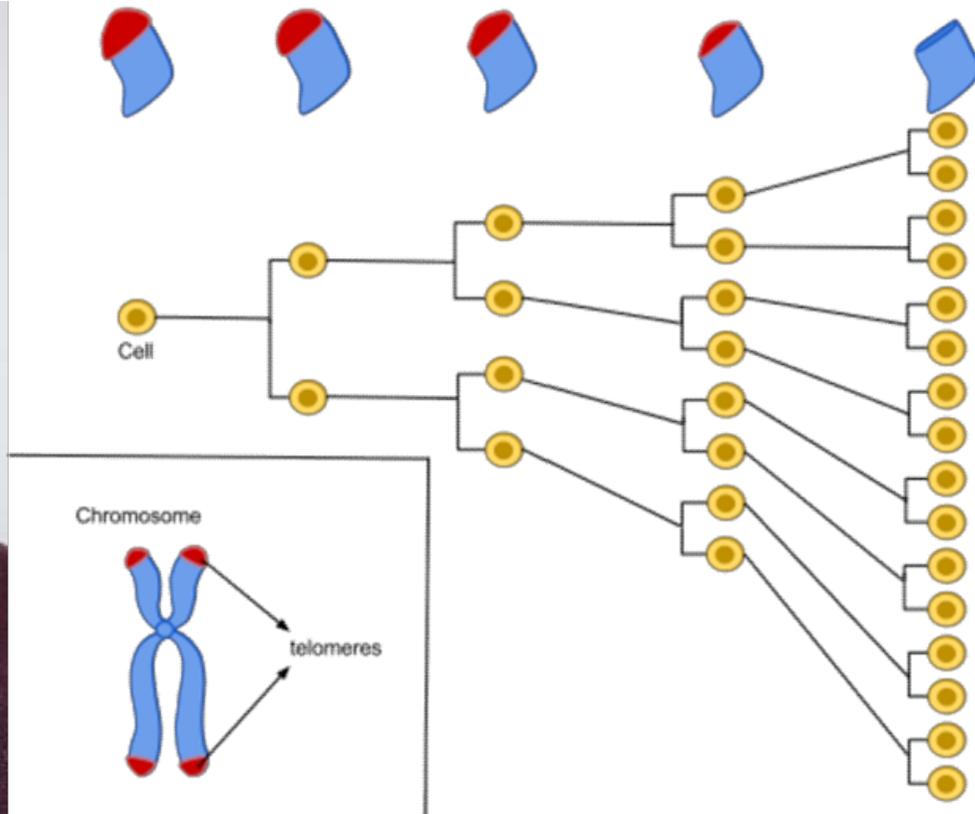
### Holliday connection:

In a Holliday junction, the two DNA strands are twisted and exchange fragments with each other. This results in cross-structures that can be seen as a point contact between them. These cross-structures can be resolved in different ways, leading to different recombination products. The Holliday junction is a key step in the homologous recombination process, which allows the exchange of genetic material between two chromosomes or two different regions of a single chromosome.

# Transitions in DNA structure



# HAYFLICK LIMIT



<b>Struktura komórkowa</b>	Number of $^{137}\text{Cs}$ 1 particles
	DłśA
DNA	1
RNA	44
Białko	700
inne cząsteczki materii organicznej	4000
Lipidy	7000
Makrocząsteczki	6, fl JD'
Woda	1.2 JD'

Products of water radiolysis are characterized, as the experiments have established, by a high level of reactivity and strong biological action. Of particular importance is the catalytic effect of hydrogen peroxides, especially  $\text{H}_2\text{O}_2$ , which is probably the most important member of the radiochemical reaction.

## RADIATION VS. CELLS

### CELLULAR SENSITIVITY

Sensitivity to ionizing radiation depends on the phase of the cell cycle. During these phases, the DNA of the cell undergoes intensive processes of replication, segregation and division. Various types of damage, mutations or structural anomalies are possible.

Damage to DNA during these phases can lead to serious consequences, such as loss or alteration of genetic information, resulting in cellular dysfunction or disease. Therefore, cells take special care and activate DNA repair mechanisms to minimize the damage from possible damage.

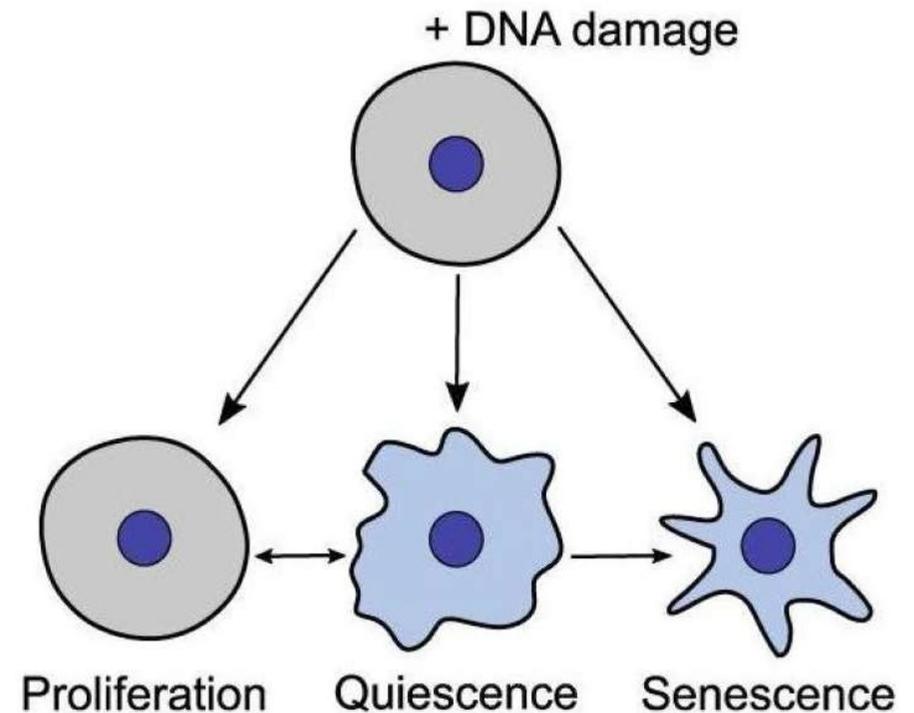
### VERY SENSITIVE

The cells that are most sensitive to ionizing radiation are those in the dividing/cell division phase, that is, in the phases of mitosis (in somatic cells) or meiosis (in reproductive cells). This is because during cell division DNA is most vulnerable to damage, and repair processes can be disrupted by radiation.

Cells in the G<sub>2</sub> phase (before the M phase) are the most sensitive to radiation, because in this phase the DNA is already replicated, but cell division has not yet occurred. This sensitivity is due to the fact that the double amount of DNA increases the potential radiation damage, while at the same time the cell has limited capacity to repair damage before division.

### LESS SENSITIVE

In contrast, cells in the G<sub>0</sub> phase (i.e., resting phase) may be less sensitive to ionizing radiation because they are in a state of "quiescence" and do not proceed to cell division.



# BERGON AND TRIBONDEAU PRINCIPLE

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In 1906, French radiation therapists Bergonie' and Tribondeau noted that:

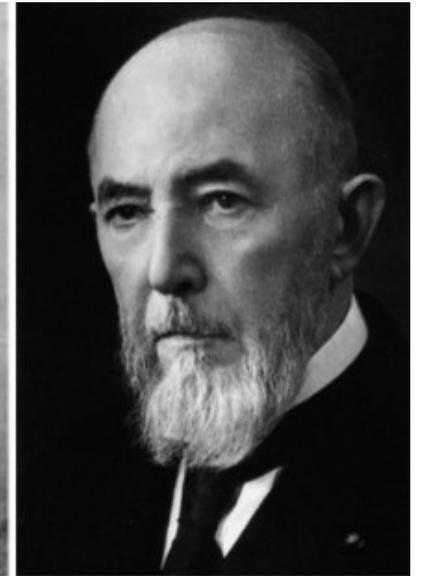
- THE RADIO-SENSITIVITY OF MAMMALIAN CELLS IS PROPORTIONAL TO THE RATE OF CELL DIVISION
- AND INVERSELY PROPORTIONAL TO THE DEGREE OF THEIR DIFFERENTIATION



Jean Alban Bergonié  
1857 – 1925

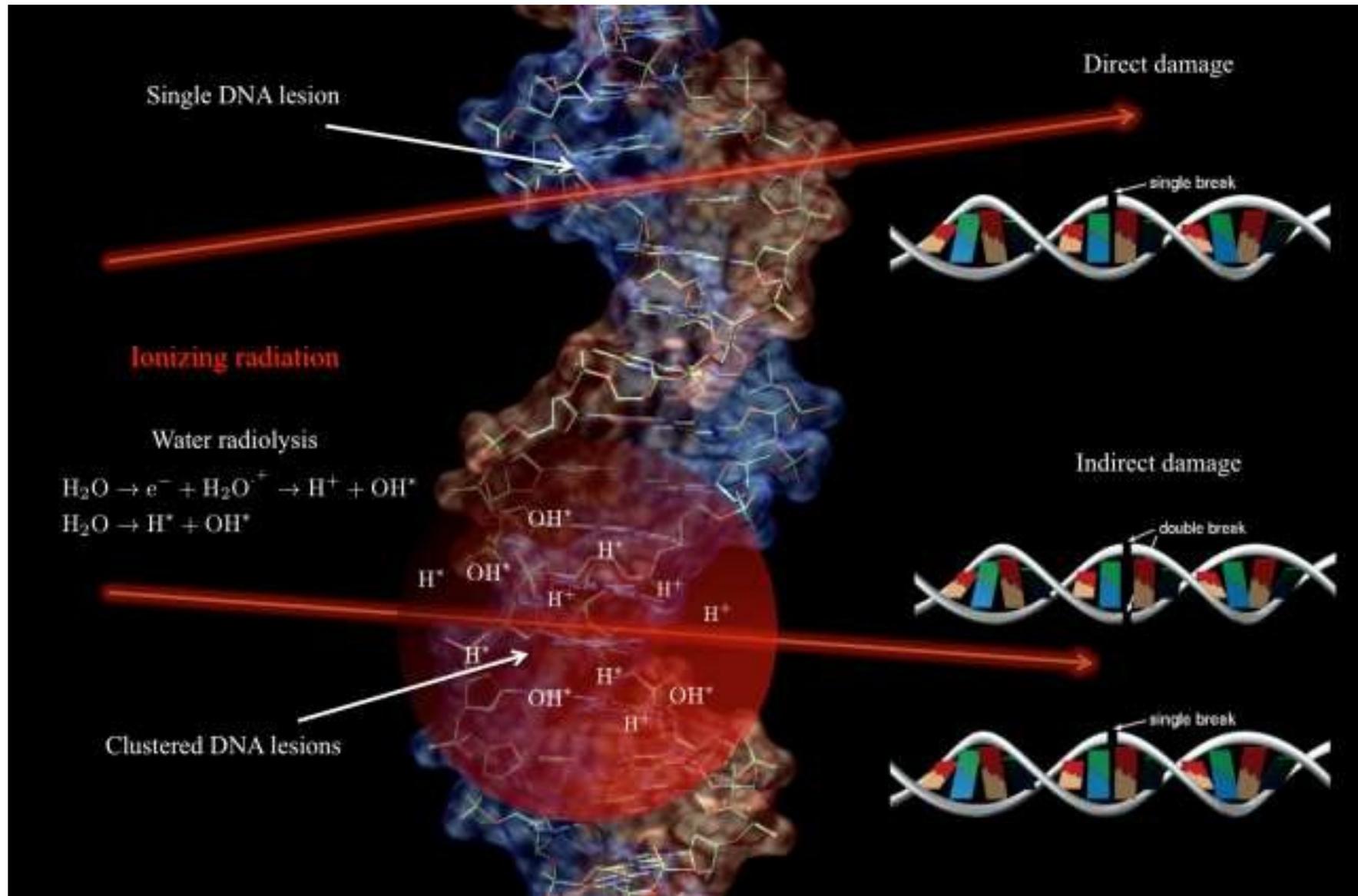


Louis Tribondeau  
1872 – 1918

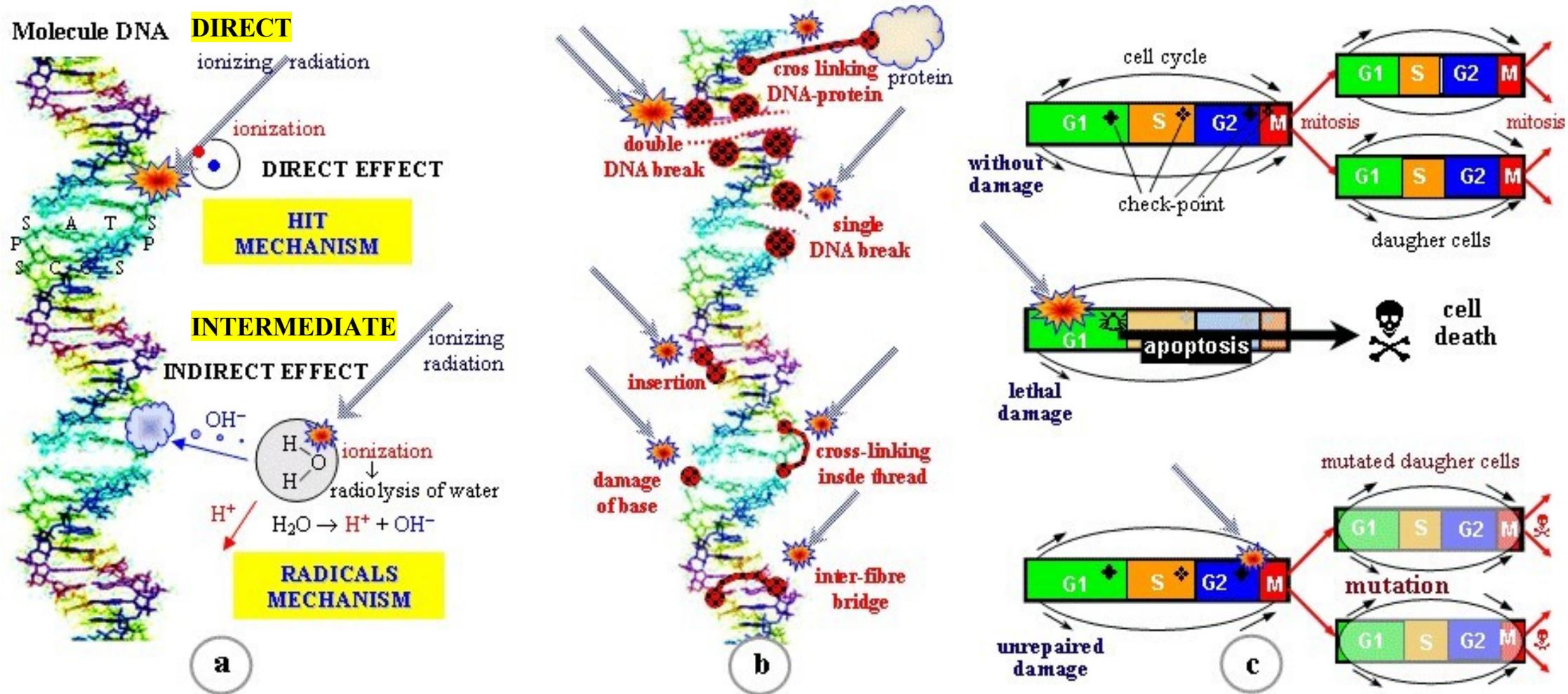


Claudius Regaud  
1870 - 1940

## DAMAGE TO THE BOTTOM CAUSED BY IONIZING RADIATION

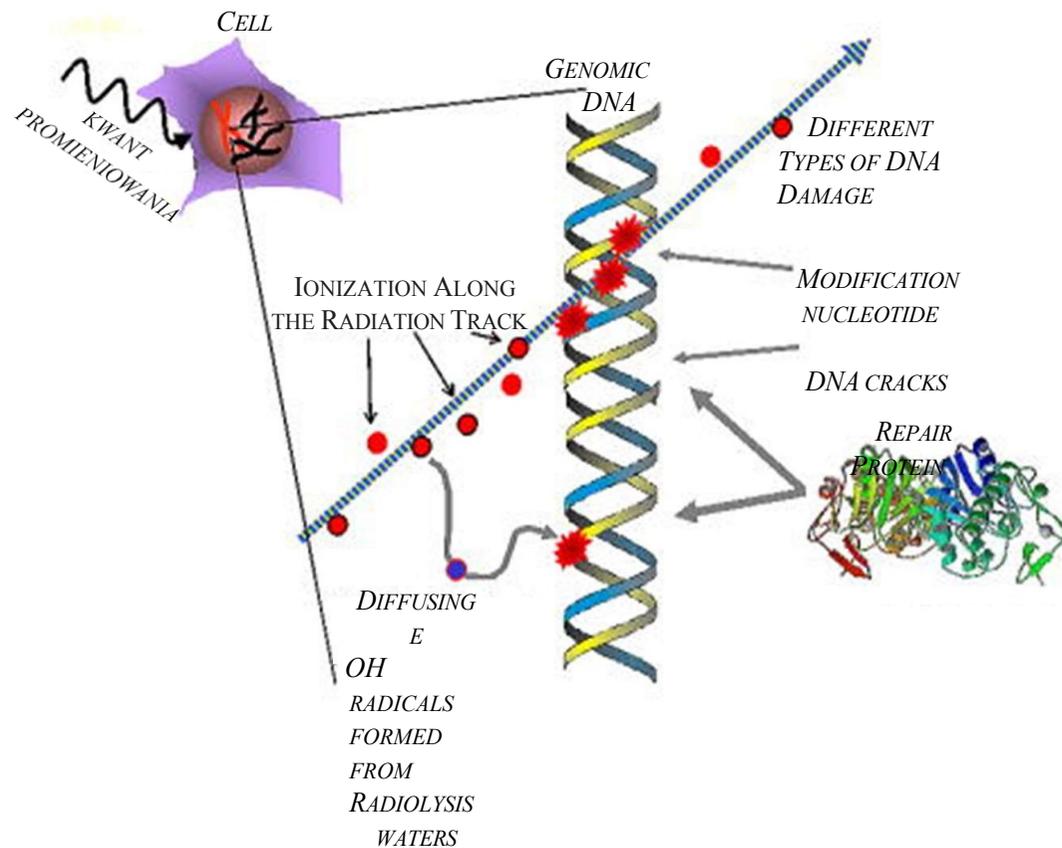


# DAMAGE TO THE BOTTOM CAUSED BY IONIZING RADIATION



# DNA REPAIR MECHANISMS

Cells take special care and activate DNA repair mechanisms to minimize the damage from any damage.



**Repair by Nucleotide Excision Repair (NER):** This repair mechanism removes damaged DNA nucleotides, such as thymine dimer formed by UV exposure or other damaging agents. NER is divided into two main pathways: global NER, which acts on the entire genome, and transcription-related NER, which removes damage to the gene during transcription.

**Repair by compatibility with unpaired bases (BER - Base Excision Repair):** This mechanism removes single bases damaged by oxidation, alkylation or deamination. In BER, the damaged base is first removed by DNA-glycosylase, and then the sugar residue and phosphate residue are removed, and the missing fragment is replenished by DNA polymerase and ligase.

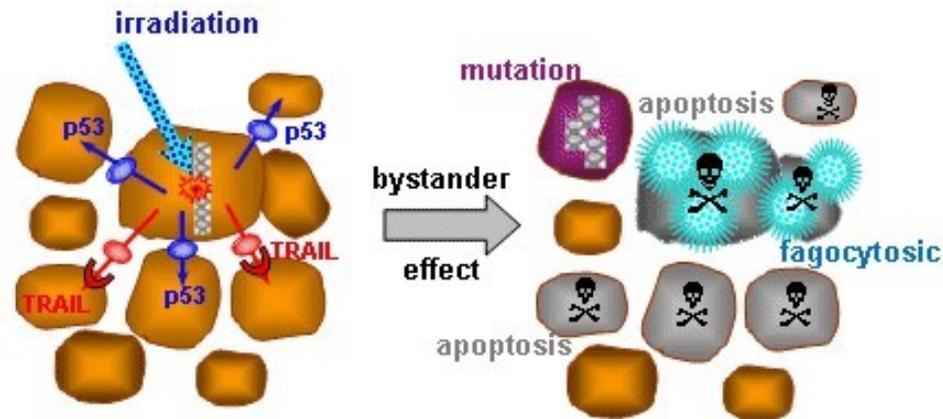
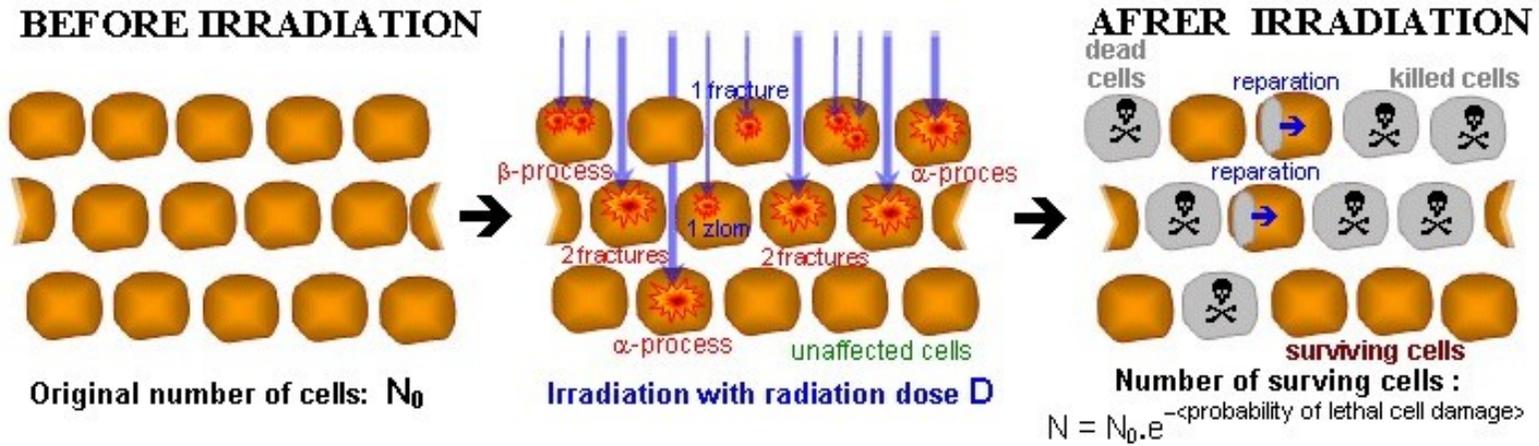
**Repair by Non-Homologous End Joining (NHEJ):** This mechanism works in situations where the two ends of damaged DNA do not have identical sequences. In NHEJ, the damaged ends are joined without the need for sequence identity, which can lead to insertions or deletions.

**Repair by Homologous Recombination (HR):** This mechanism repairs double-stranded DNA damage, such as double-strand breaks, by using an identical or similar DNA sequence as a template for repair. HR is particularly important during repair of damage in the S-phase of the cell cycle, when sister chromatin is available.

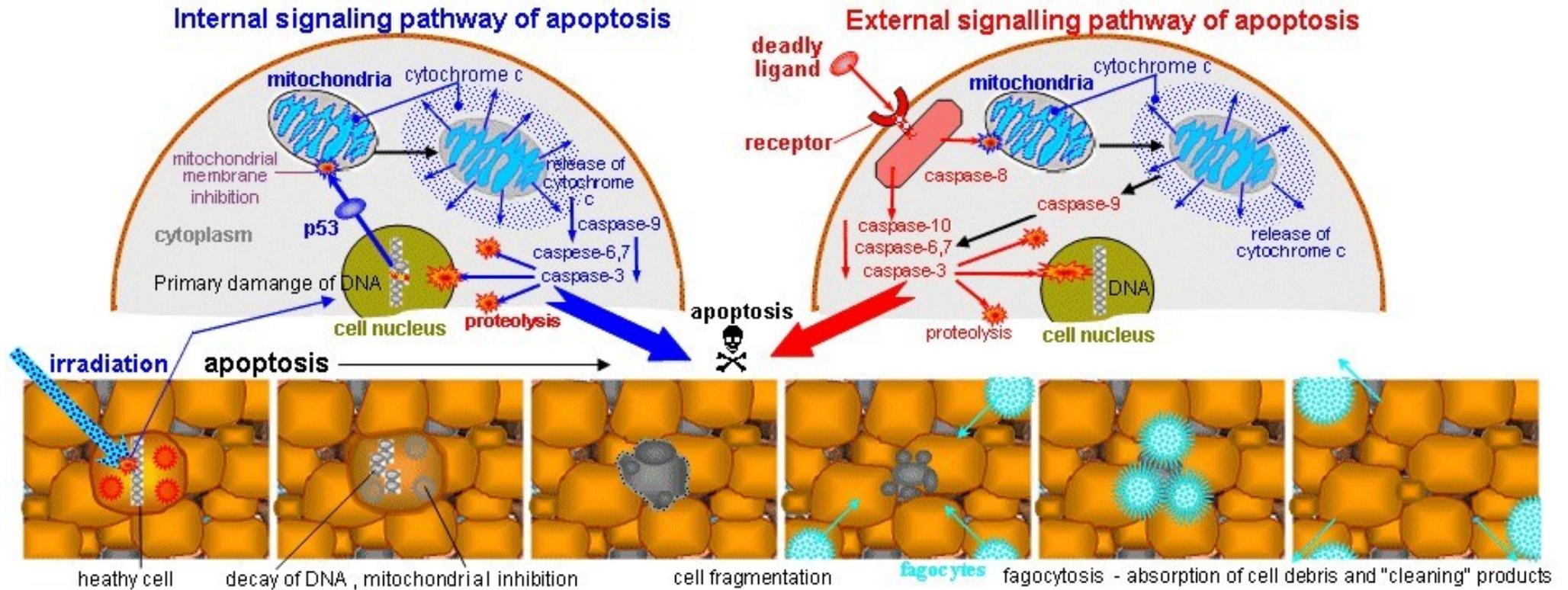
**Mismatch compatible base-pairing repair (MMR):** This mechanism repairs base-pairing errors that can arise during DNA replication or gene rearrangement. MMR removes improperly paired nucleotides and then fills in the gaps using DNA-polymerase.

These DNA repair mechanisms are crucial for maintaining the integrity of the genome and preventing mutations that can lead to diseases, including cancer. Cells use these mechanisms to repair DNA damage caused by various environmental and endogenous factors.

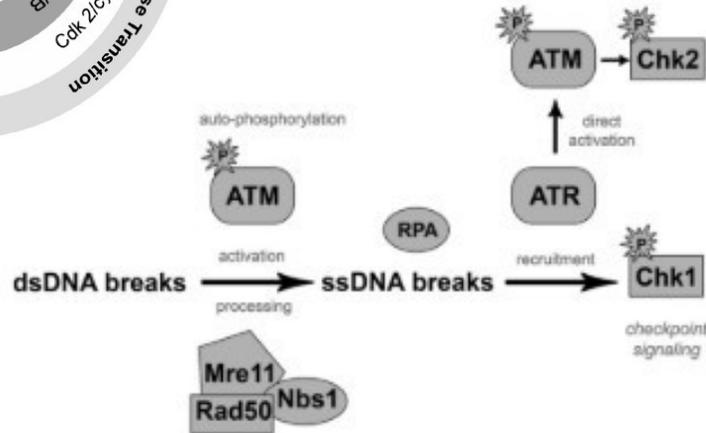
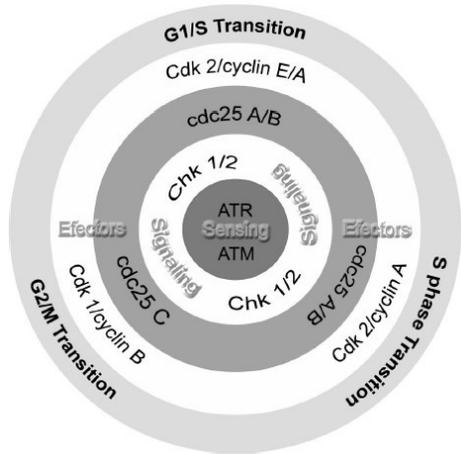
# CELLULAR EFFECT



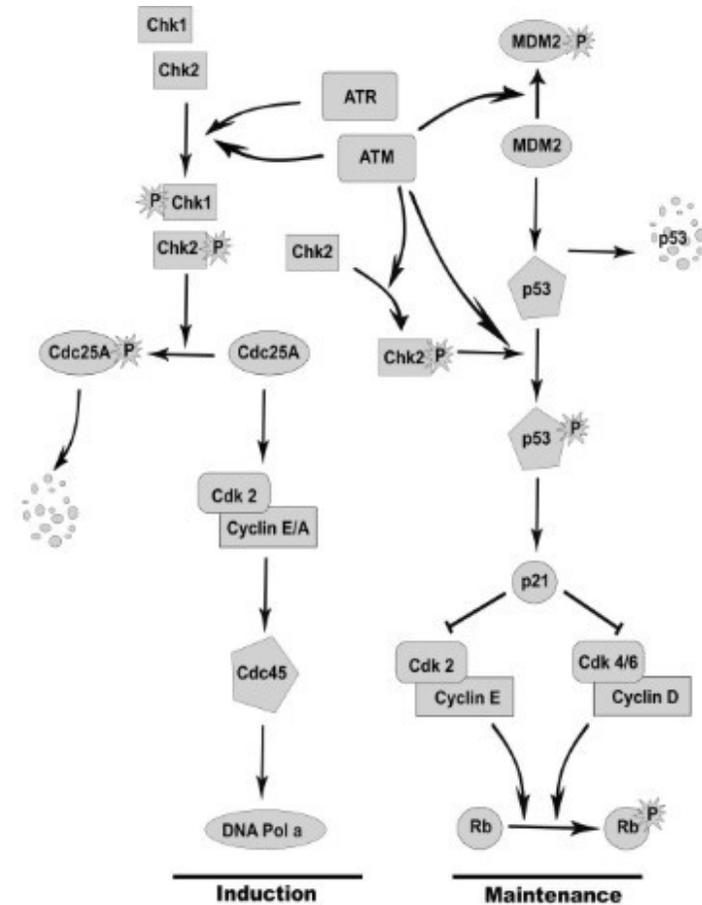
# CELL DEATH - APOPTOSIS



## MOBILE CHECK-POINTS



Interaction of ATR and ATM pathways in DNA damage checkpoint activation. DNA double-strand breaks are detected by the ATM kinase, leading to its activation, but can also be processed by the MRN complex (Mre11- Rad10-Nsb1) into single DNA strand breaks, which, after binding to RPA proteins, form ssDNA-RPA structures. These structures facilitate ATR recruitment and activation and subsequent activation of the Chk1 signaling kinase. The activated ATR can also directly phosphorylate and activate ATM and enhance checkpoint signaling through activation of Chk2 kinase.



Activation of G1/S and intra-S checkpoints in response to genotoxic stress. DNA damage is detected by ATM/ATR kinases, which activate Chk1/2 checkpoint signaling kinases. This leads to phosphorylation of Cdc25A phosphatase and its degradation, which in turn prevents activation of S-phase-specific kinases Cdk2/cyclin E and Cdk2/cyclin A. As a result, S-phase DNA replication is blocked by inhibiting the Cdc45-dependent cargo of DNA polymerase  $\alpha$  on chromatin. At the same time, phosphorylation of p53 by ATM/Chk2 kinase leads to its stabilization, by preventing Mdm2-dependent degradation, and increased expression of p21, an inhibitor of Cdk2/cyclin E and Cdk4/6/cyclin A activity. Decreased phosphorylation of pRb protein by Cdks blocks activation of S-phase-specific DNA expression.

# SUPPRESSOR PROTEINS

also known as tumor suppressor proteins, are a group of proteins responsible for inhibiting the growth of cancer cells by regulating various cellular processes such as proliferation, differentiation, apoptosis and DNA repair. Here are some examples of the main tumor suppressor proteins:

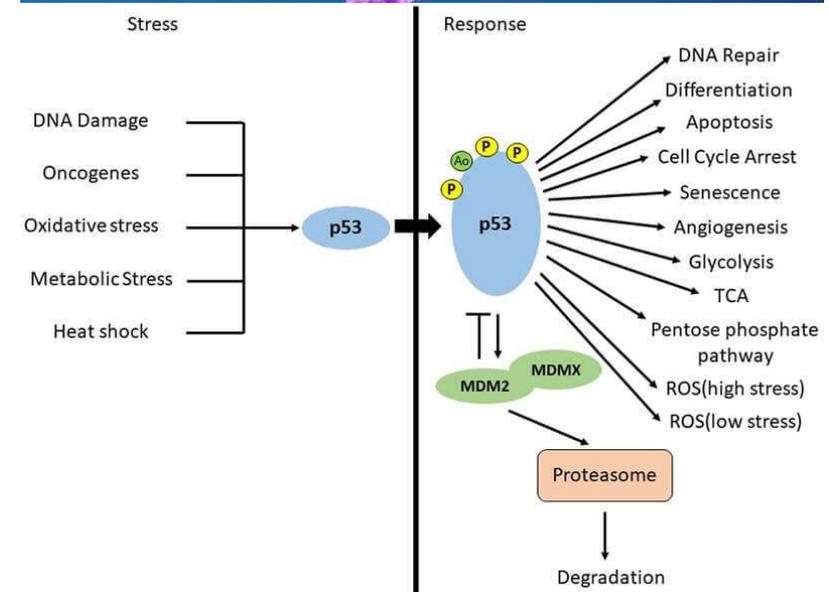
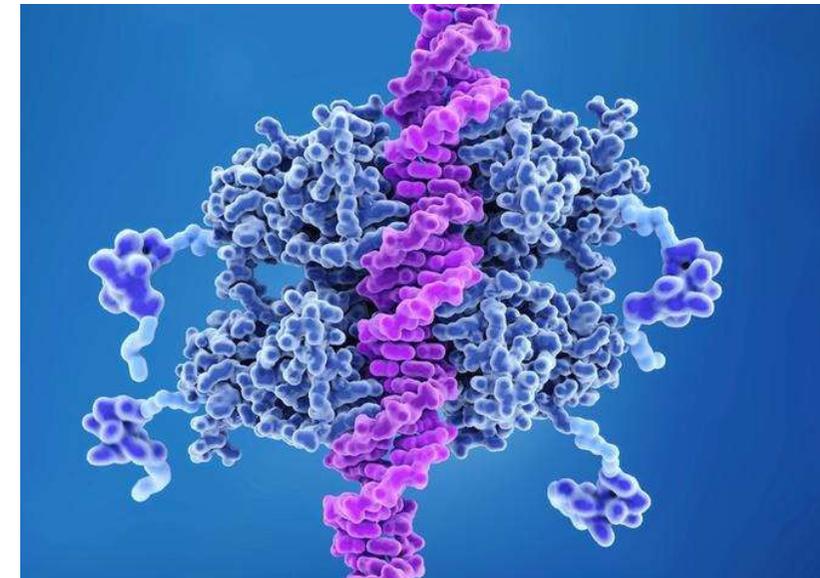
**The tumor suppressor protein p53:** This is one of the best-known tumor suppressor proteins, also known as the "guardian of the genome." P53 acts as a transcription factor that activates many DNA repair genes, inhibits the cell cycle and induces apoptosis of cells that are damaged or have abnormal proliferation.

**RB (retinoblastoma) proteins:** RB proteins control the cell cycle by inhibiting cell cycle-related transcription factors such as E2F proteins. In an inactive state, RB proteins allow the cell to enter the S phase of the cell cycle, while activated RB proteins inhibit this process, leading to cell cycle arrest.

**PTEN (Phosphatase and Tensin Homolog) Proteins:** PTEN is a phosphatase that regulates signaling pathways involved in cell proliferation and apoptosis. It acts by degrading the second-order lipid, phosphatidylinositol triphosphate (PIP3), leading to inhibition of the PI3K/AKT pathway, important in regulating cell growth.

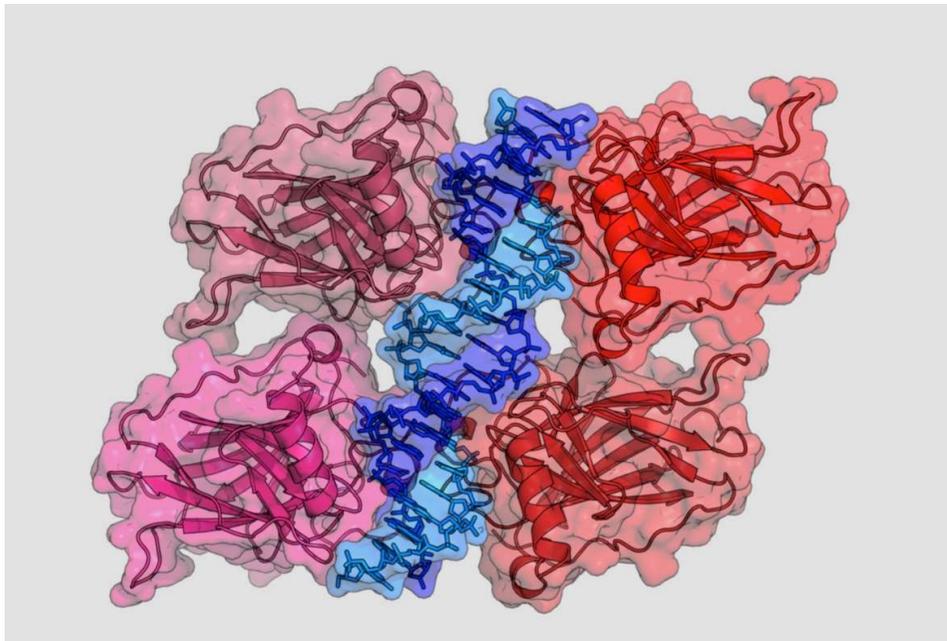
**BRCA1 and BRCA2 tumor suppressor proteins:** These proteins are involved in DNA repair, particularly the removal of double-stranded DNA damage. Mutations in the BRCA1 and BRCA2 genes increase the risk of developing breast and ovarian cancer.

**APC (adenomatous polyposis coli) suppressor proteins:** APC is involved in the regulation of signaling pathways involved in the regulation of cell proliferation, cell adhesion and cell movement. Mutations in the APC gene are associated with colorectal polyposis and colorectal cancer.



## THE P53 PROTEIN

**p53** plays a key role as a guardian of the genome, the integrity of the cellular genome and the DNA damage response, which is important for preventing cancer development and maintaining cellular homeostasis. p53 does not repair DNA damage directly. Its main function is to control the DNA damage response by regulating processes such as cell cycle arrest, induction of apoptosis and regulation of DNA repair by other mechanisms. Thus, in the case of severe damage, p53 can influence the elimination of cells through apoptosis to prevent potential mutations and the development of cancer. The mechanism of action of the p53 protein is complex and involves several key cellular processes. Here are the main elements of p53's mechanism of action:



**Activation in response to DNA damage:** such as double helix breaks, radiation damage, or point mutations. Under normal conditions, p53 is present in cells at low levels, but when DNA damage is detected, its concentration increases.

**Cell cycle regulation:** Activated p53 acts as a transcription factor that regulates the expression of many genes, including cell cycle inhibitory genes. p53 can activate the expression of cell cycle inhibitory genes, such as p21, leading to cell cycle arrest in the G1 phase, allowing damaged DNA to be repaired before cell division.

**Induction of apoptosis:** If DNA damage is too extensive or irreversible, p53 can also induce apoptosis, or controlled cell death. P53 regulates the expression of apoptosis-related genes such as BAX, PUMA and NOXA, leading to the activation of the apoptotic cascade and elimination of cells that cannot be repaired.

**Regulation of DNA repair:** p53 can affect DNA damage repair by regulating the expression of genes associated with various repair mechanisms, such as unpaired base-compatible repair (NHEJ) or repair by fragment exchange (NER).

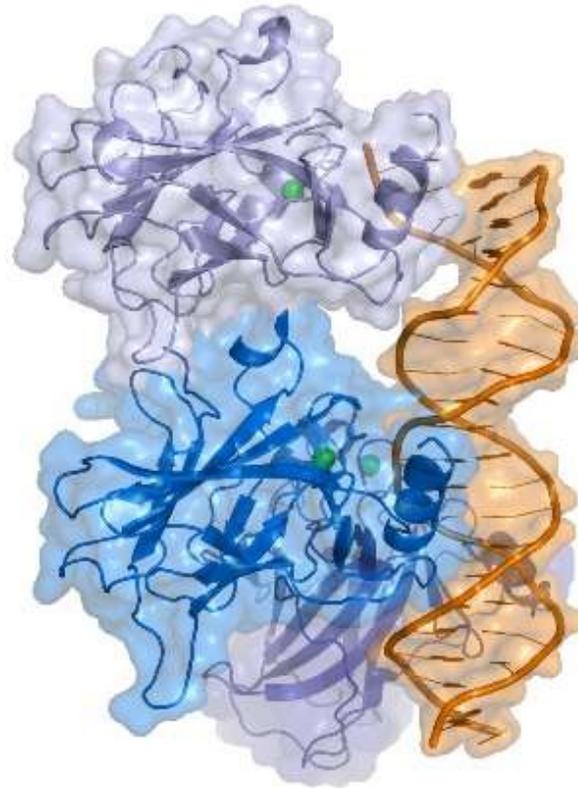
**Other functions:** In addition to cell cycle regulation, apoptosis and DNA repair, p53 is also involved in other cellular processes, such as regulation of angiogenesis, metabolic reprogramming of tumor cells and response to oxidative stress.

# THE P53 PROTEIN

The TP53 gene is a tumor suppressor gene that encodes a protein for cell cycle regulation, DNA repair and apoptosis induction. - p53. Mutation of TP53 is an important factor in the process of cancer formation. It is mutated in more than 50% of cancer types, resulting in the loss of cell growth "brakes" in the p53 signaling pathway. The wild-type p53 protein is at the intersection of cellular and death signals, playing a critical role in cell growth, differentiation, aging, stress response and apoptosis. At the same time, it has complex relationships with other signaling pathways intracellular.

Signaling pathways directly related to p53 activity: p21, MDM2, CD95/fas, Bax, WT1, p53AIP1, PAG608, HER2, PCNA, cyclin G, etc.

Its level of intracellular localization is kept relatively low, and the p53 signaling network is "turned off." When cells are stressed or damaged, p53 levels are increased, activating a "brake" function on growth and DNA repair of damaged cells and mutants, preventing cancer. Activation of p53 following min: ultraviolet (UV) radiation, ionizing radiation.



Most of the above activation signals are caused by cell damage and mutation, and the p53 signaling network is activated to prevent damage or accumulation of mutant cells. Among them, there are many causes of DNA damage, such as UV irradiation, chemotherapy and ionizing radiation. Different types of DNA damage activate different downstream signals. For example, ATM (ataxia telangiectasia-mutated) and ATR (ATM and Rad3-related) in response to ionizing radiation and UV irradiation, respectively, are members of phosphoinositol-3-kinase-related kinases (PIKKs) that initiate a series of phosphorylation reactions, ultimately causing p53 to stabilize and accumulate intracellularly. Primary tumor gene activation and hypoxia are also stimulated by a series of phosphorylation reactions that transmit signals to p53, ultimately inhibiting p53 degradation, maintaining high levels of p53 and its functions intracellularly.

## The role of P53 in cancer prevention

The Role of p53 in Cancer

[https://www.youtube.com/watch?v=2RG9caushi0&ab\\_channel=MajaDivjak](https://www.youtube.com/watch?v=2RG9caushi0&ab_channel=MajaDivjak)

Protooncogenes are normal, healthy genes that can turn into oncogenes (i.e. cancer genes) as a result of mutation or over-activation. When protooncogenes undergo such a change, they can begin to promote uncontrolled cell growth, which can lead to cancer. Examples of protooncogenes include genes encoding growth factors, cell receptors, signaling proteins and transcription factors, which are normally involved in regulating cellular processes such as cell growth, differentiation and survival. However, when they are changed into oncogenes, they can contribute to the formation of cancer.

Gene: c-Myc Product: Myc protein Function: Regulates the expression of genes related to growth, differentiation and apoptosis.

Gene: c-Ras Product: Ras protein Function: Involved in the regulation of signaling pathways that control cell growth and differentiation.

Gene: c-ErbB (EGFR) Product: cell growth factor receptor (EGFR) Function: Participates in transduction of cell growth signals in response to cell growth factors.

Gene: c-Kit Product: hemopoiesis-stimulating factor receptor (Kit) Function: Involved in the regulation of hematopoietic cell differentiation and other cellular processes.

Gene: c-Src Product: Src tyrosine kinase Function: Involved in signaling pathways controlling cell adhesion, migration and proliferation.

Gene: c-Fos Product: Fos protein Function: Involved in the transcriptional regulation of genes related to stress responses and cell growth.

Gene: Bcl-2 Product: Bcl-2 protein Function: Participates in the regulation of apoptosis (programmed cell death), protecting cells from apoptosis. Its overexpression or mutation can lead to unregulated cell survival, which is characteristic of many cancers.

TNBC is an aggressive subtype of breast cancer that is characterized by a lack of expression of estrogen receptor (ER), progesterone receptor (PR) and human growth factor receptor 2 (HER2/neu). This means that the cancer cells do not respond to hormone therapies or HER2-targeted treatments, making them difficult to treat. The following is some key information about triple-negative breast cancer:

**Molecular features:** Triple-negative breast cancer is defined by the lack of expression of ER, PR and HER2 receptors. Consequently, therapies that target these receptors, such as hormonal therapies (e.g., tamoxifen) or therapies that target HER2 (e.g., trastuzumab), are ineffective in treating this subtype of cancer.

**Prevalence:** TNBC accounts for about 10-15% of all breast cancer cases. It is more common in younger women, people of lower socioeconomic status, African-American women and people with mutations in BRCA1 genes.

**Prognosis:** Triple-negative breast cancer typically has a worse prognosis than other breast cancer subtypes. This is partly due to the lack of options for therapies targeting ER, PR or HER2 receptors. However, recent years have seen advances in the treatment of TNBC, particularly with therapies targeting biological pathways such as PARP inhibitors and immunotherapy.

**Treatment:** Treatment of triple-negative breast cancer usually includes chemotherapy, as cancer cells of this type are usually sensitive to chemotherapeutics. In addition, clinical trials of new therapies, such as PARP inhibitors and immunotherapy, are continuing to improve treatment outcomes.

**Recurrence risk and monitoring:** Patients with triple-negative breast cancer often undergo post-treatment monitoring for early detection of possible recurrence. Imaging tests, such as mammograms and MRIs, may be performed regularly, as well as blood tests to monitor tumor markers.

Triple-negative breast cancer presents clinical challenges due to its aggressive nature and limited treatment options. However, research into new therapies continues to improve treatment outcomes for TNBC patients.

- Changes in p53 and BRCA 1 and 2 may be present

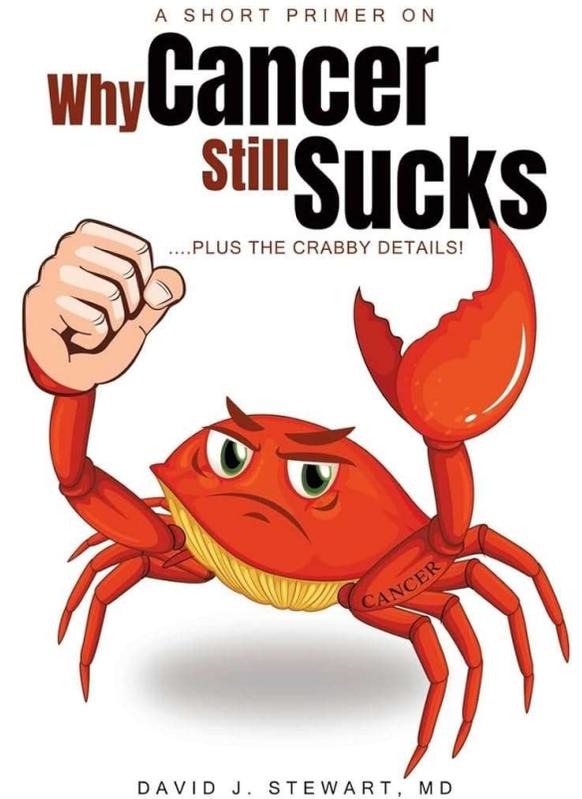
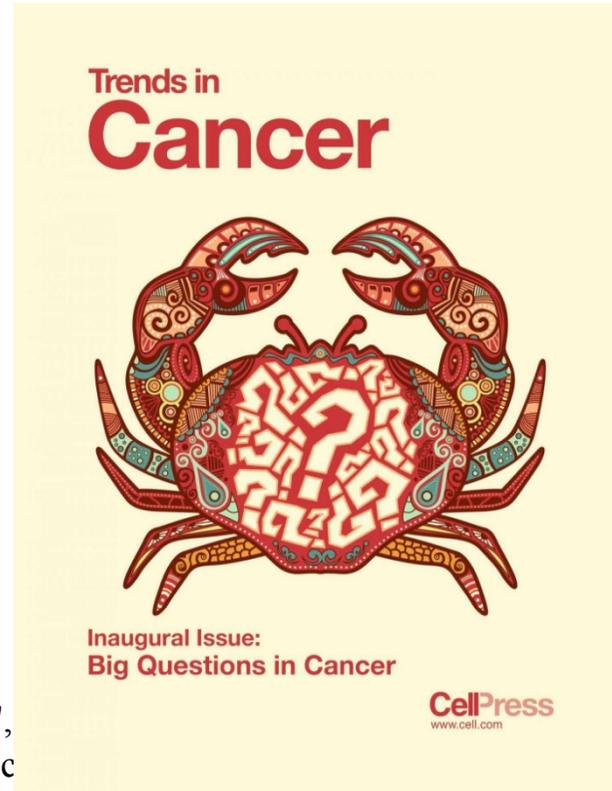
### **DNA damage response - breast cancer**

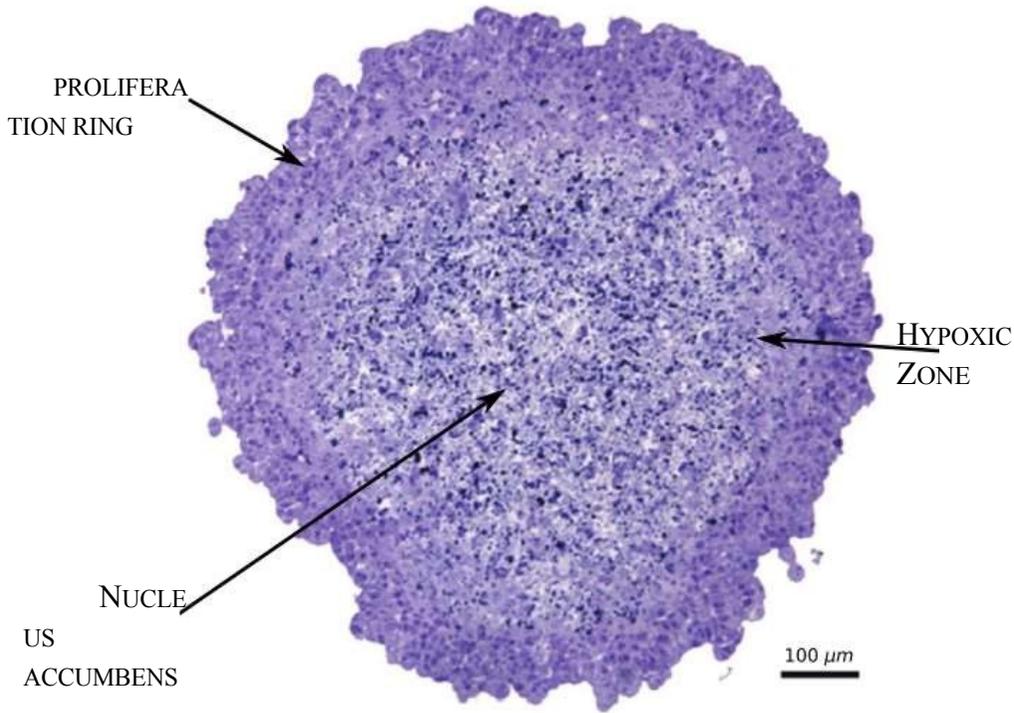
#### DNA Damage Response

[https://www.youtube.com/watch?v=2yI\\_SoOWFMU&ab\\_channel=Nymus3D](https://www.youtube.com/watch?v=2yI_SoOWFMU&ab_channel=Nymus3D)



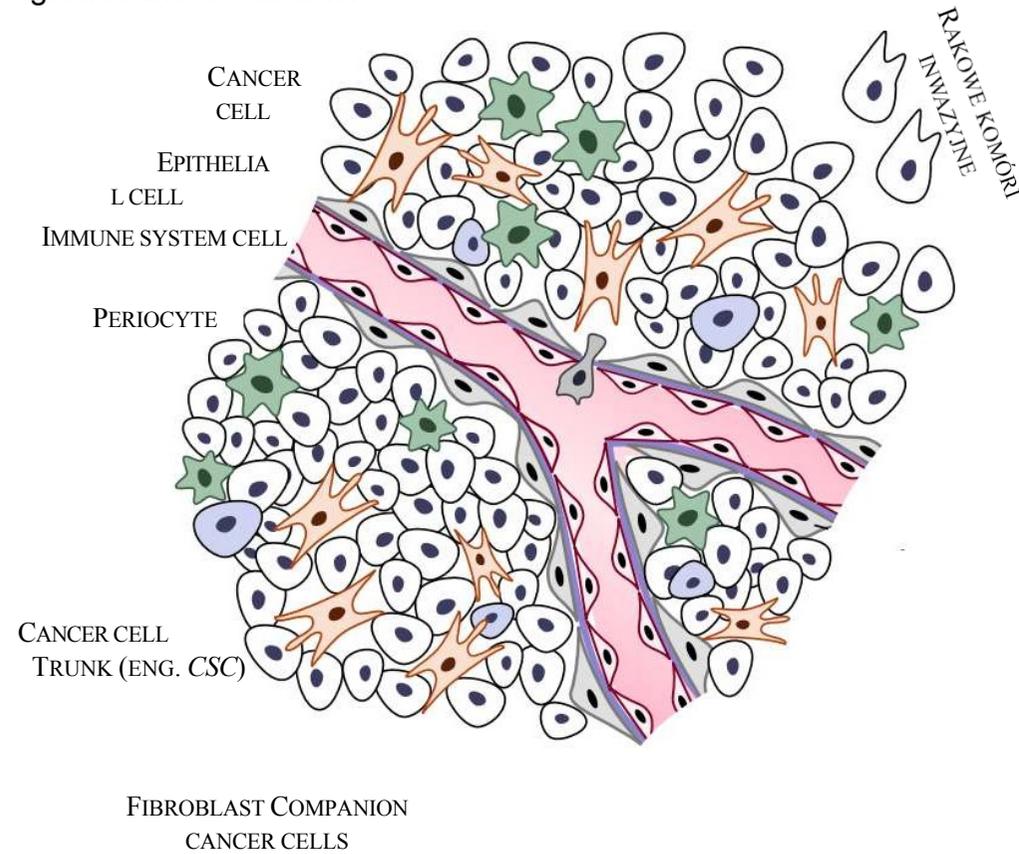
Latin *carcinoma*, from Latin *cancer* - "cancer", "crab",  
From the Greek *καρκινος* /karkinos/ - "crayfish", "sea c



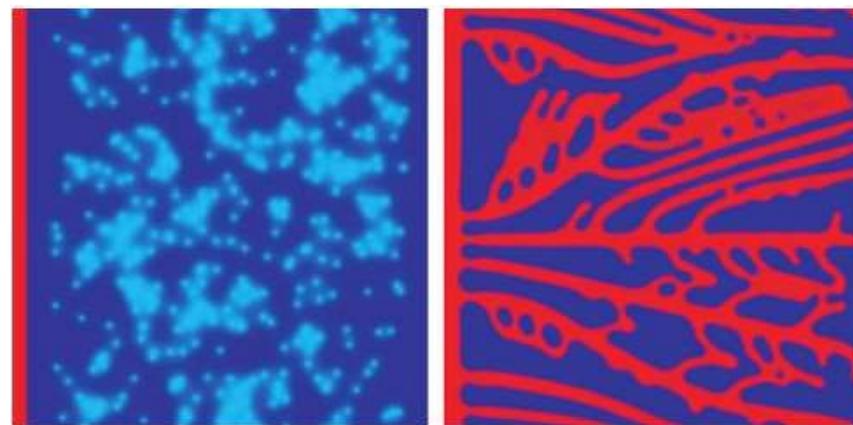
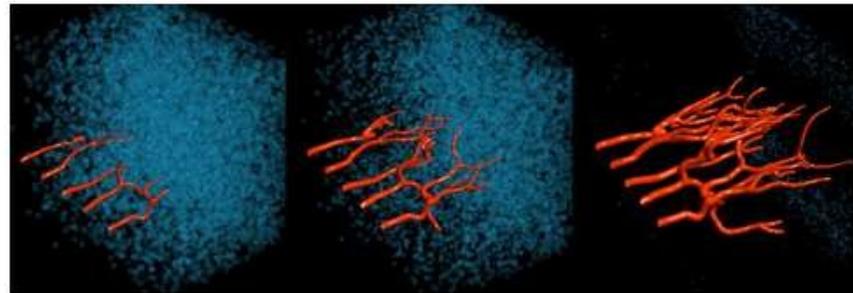
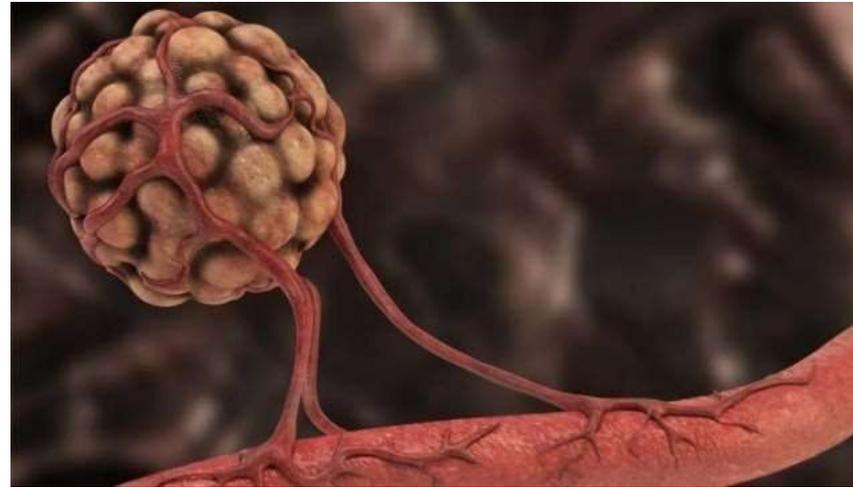
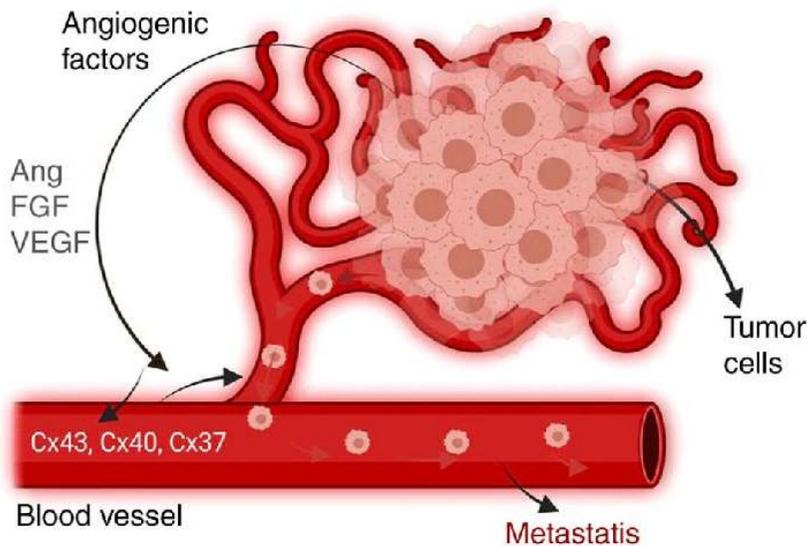
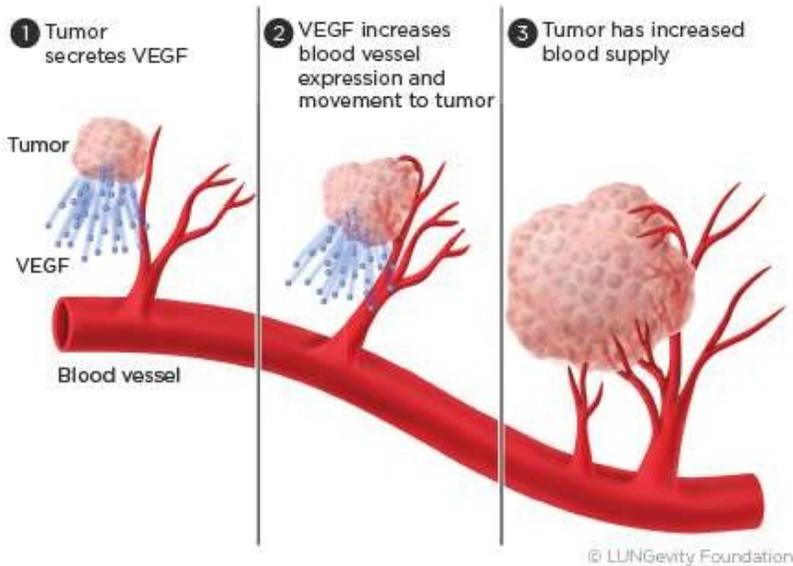


Sectioning of a nonvascular tumor. The non-vascularized tumor reaches a stable configuration with three main distinguishable areas: the proliferative margin, the hypoxic zone and the necrotic nucleus.

Tumor lining. Tumor cells recruit other cells from their microenvironment to form the tumor lining. Tumor cells associated with newly formed vessels, activate fibroblasts to produce pro-inflammatory cytokines, e.g.: IL-6, IL-8, tumor necrosis factor alpha (TNF- $\alpha$ ) or growth factors (e.g. TGF- $\beta$ ). This promotes inflammation in the tumor microenvironment and promotes tumor growth and metastasis.

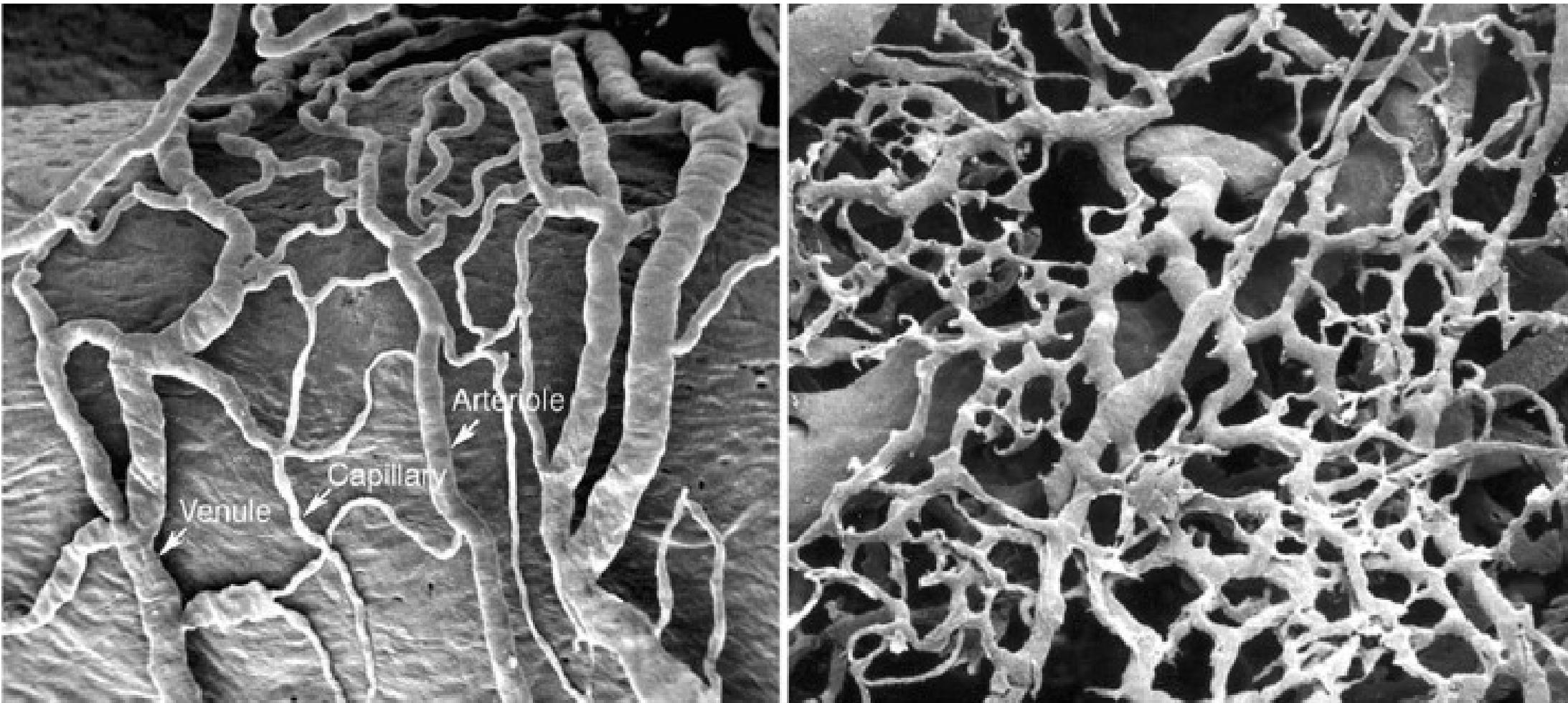


## Blood Vessel Overgrowth on Cell



A three-dimensional model based on discrete Agents, which takes into account the interactions between vascular end cells (TECs) and the extracellular matrix (ECM).

Hybrid A model of tumor-induced angiogenesis. Left: Tumor angiogenic factor (light blue) promotes angiogenesis from the initial vessel (red). Right: The model takes into account the formation of a new vascular network.



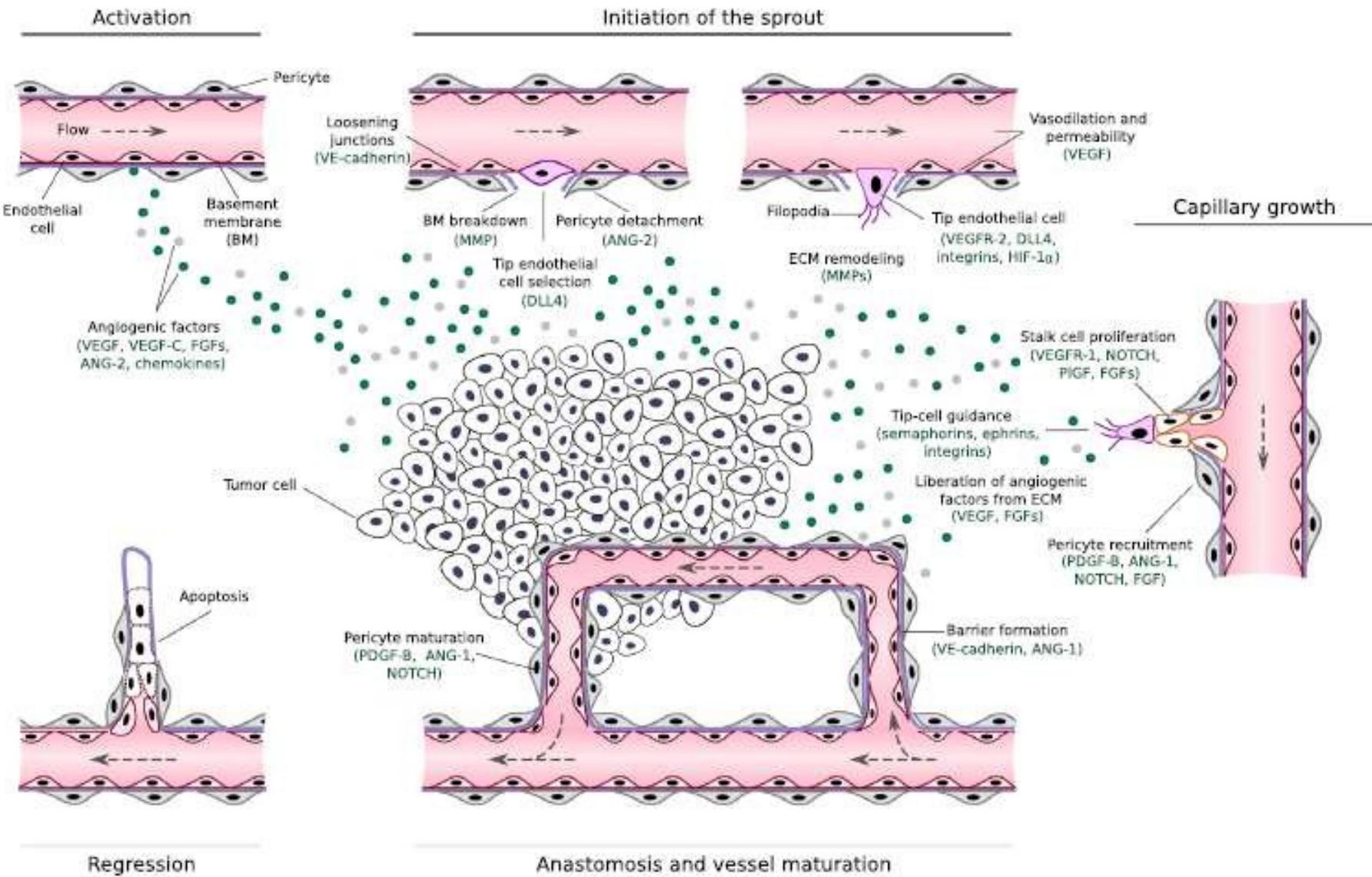
Physiological versus tumor vessels. Unlike vessels formed under physiological conditions (left panel), tumor vessels (right panel) show a marked loss of vessel hierarchy and structure, significant morphological deviation, and disordered and numerous branches.

**THE PROCESS OF ANGIOGENESIS**

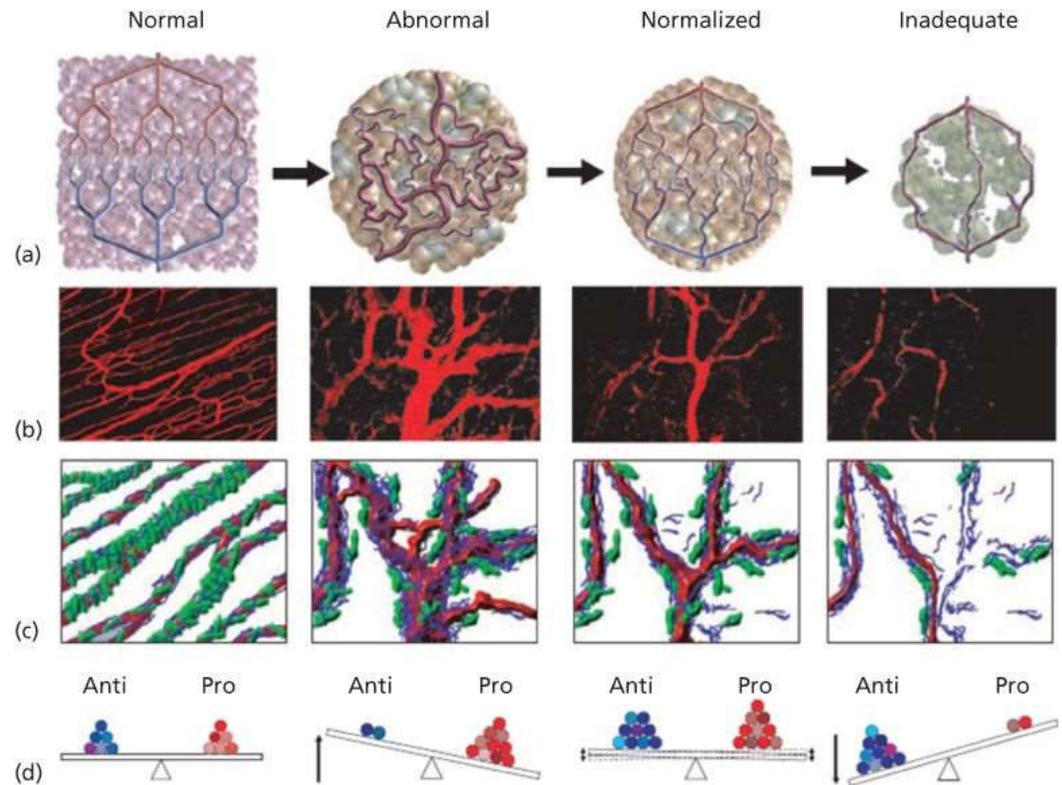
Angiogenesis begins with the activation of endothelial cells by the factors angiogenic factors of the tumor. The resulting bud, already freed from under the vascular sheath, grows toward the source of tumor angiogenic factors.

The capillary elongates through proliferation

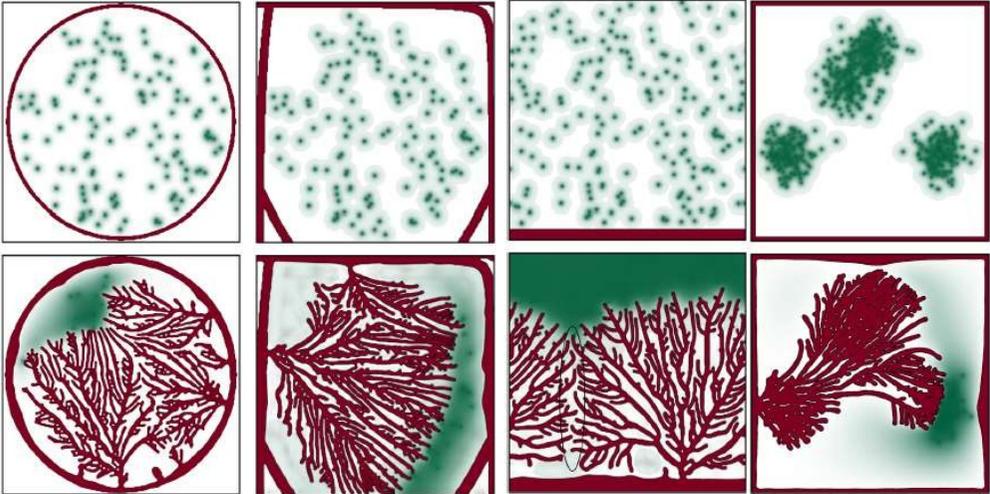
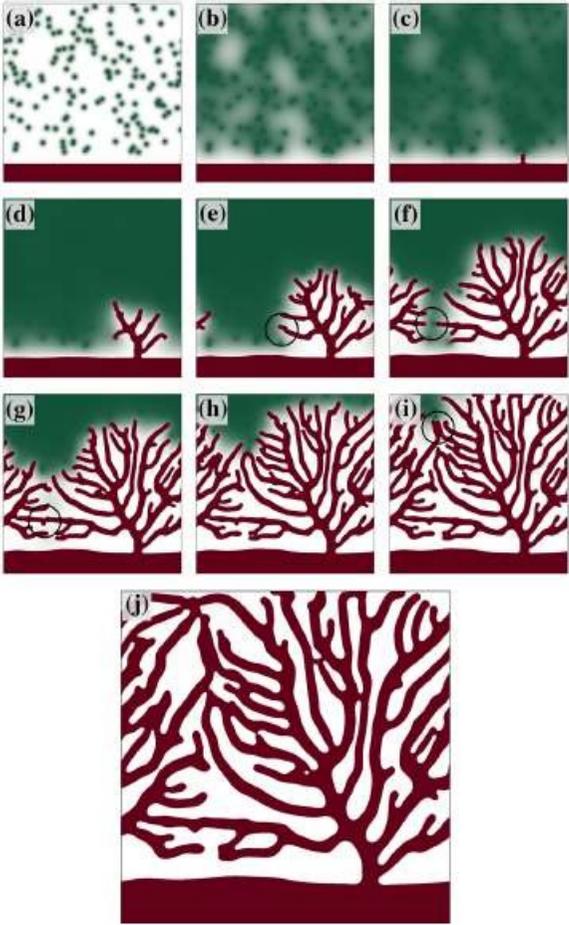
endothelial cells. Growth continues until two capillaries anastomose. The new vessel matures, and blood flows through it, irrigating the tissue. In addition, in the absence of stimuli, immature capillaries may also regress.



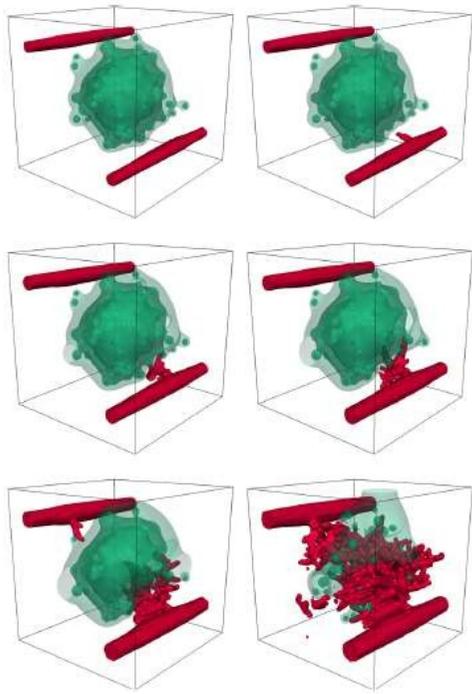
Changes in tumor vasculature during treatment with anticancer drugs. (a) The tumor vascular network is structurally and functionally abnormal. Anticancer therapies may initially improve both the structure and function of the tumor vasculature. Continued or aggressive anti-cancer treatment regimens may eventually lead to an inadequate vascular supply that is unable to support tumor growth. (b) Normalization of the vasculature by VEGFR2 inhibition. On the left is a two-photon image showing normal blood vessels in skeletal muscle; the next representative images show the vasculature of colon cancer in mice at day 0, day 3 and day 5 after treatment with a VEGFR2-specific antibody. (c) Diagram illustrating simultaneous changes in basement membrane coverage (blue) and endothelial cells (red) during vascular normalization. (d) These changes in the vasculature may reflect changes in the balance of pro-angiogenic and anti-angiogenic factors in the microenvironment.



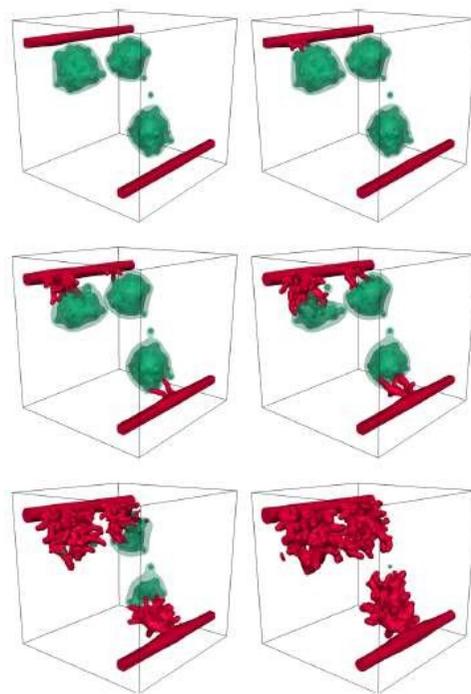
Vascular network formation. Two hundred randomly distributed hypoxic cells release tumor angiogenic factor (in green), which induces angiogenesis from a pre-existing capillary (in red). New capillaries guided by terminal endothelial cells grow and form a new vascular network. As they grow, they consume angiogenic factor and fuse with other capillaries. The resulting vascular pattern presents a defective structure characteristic of tumor-induced angiogenic vascular networks.



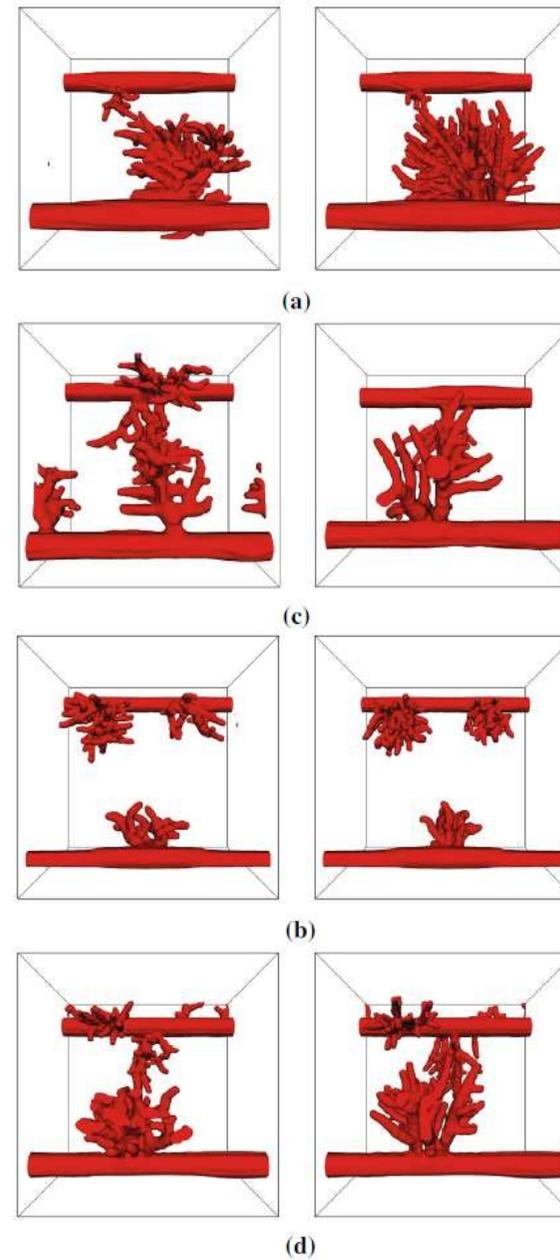
The impact of initial conditions in the larger field. Initial conditions (top) and advanced stage of angiogenesis (bottom) of four simulations. The domain represents a tissue measuring 875 micrometers by 875 micrometers. The first three simulations start with the same random distribution of hypoxic cells, but differences in the initial capillaries lead to different arrangements of blood vessels. In the last simulation, the multi-point distribution of hypoxic cells generates a vascular network that encompasses each focus.



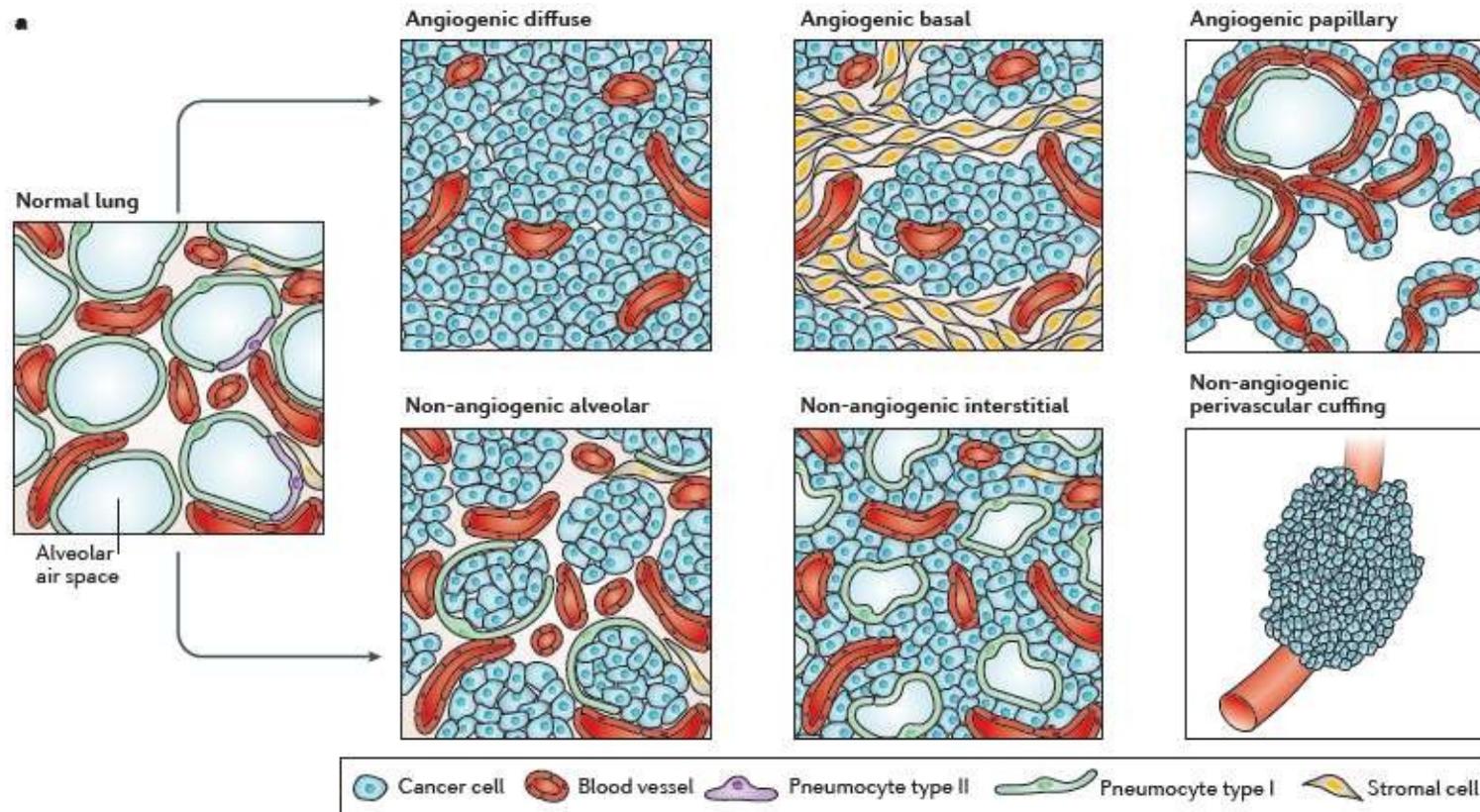
Simulation of three-dimensional angiogenesis from two parent capillaries. Angiogenesis is triggered by the tumor angiogenic factor (green iso-surfaces), which is released by hypoxic cells arranged to form a tumor-like structure. The endothelial cells that guide bud growth migrate by chemotaxis and haptotaxis. A new vascular network develops from the two parent capillaries and penetrates the tumor, leaving no cells under hypoxic conditions. The initial capillaries are connected through the new network, allowing blood to flow between them.



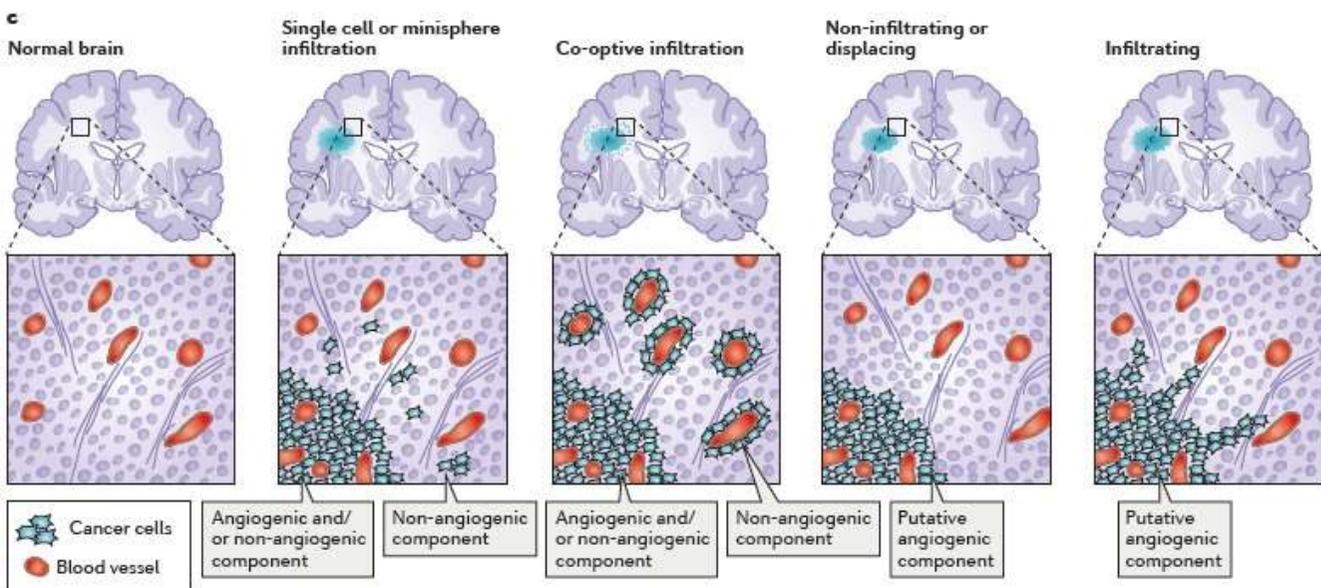
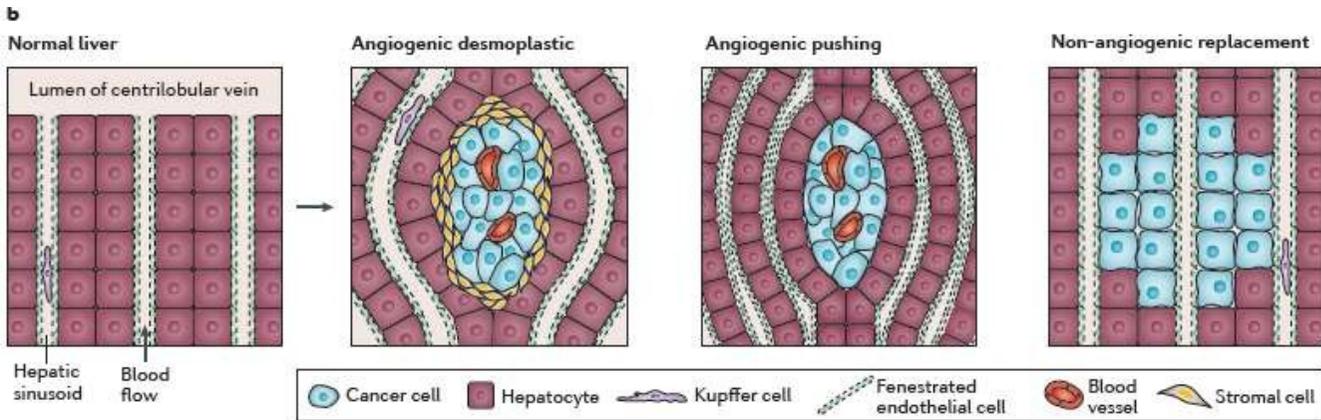
Multi-point tumor stimulates angiogenesis in three dimensions. Three clusters of hypoxic cells, which mimic the multipoint tumor, induce the formation of a new vascular network. The resulting vascular network forms three structures, which satisfy the oxygen demand of the clusters, but are independent and unconnected to each other.



Effect of haptotaxis in angiogenesis. Each subfigure shows the final pattern produced by a model with haptotaxis (left side) and a model without haptotaxis (right side). Each simulation begins with two initial capillaries parallel to each other. We describe the initial hypoxic cell state in detail in each caption. Note that the left snapshots in Figure 18a, c correspond respectively to Figures 16 and 17. vessel patterns blood vessels for each model vary in their complexity, number of anastomosis events (connections), and length of capillaries. a One central tumor. b Three clusters of hypoxic cells. c One hundred randomly distributed hypoxic cells. d Two hundred randomly distributed hypoxic cells.



a | Schematic representation of tumor growth patterns in the lung. In normal alveolar parenchyma of the lung, alveolar spaces are filled with air and are bounded by alveolar walls. Inside the alveolar walls are blood vessels surrounded by a thin layer of extracellular matrix. Type I and type II epithelial cells line the alveolar walls and separate the vessels from the air space. Three basic patterns of tumor growth angiogenesis are observed in the lung, which can occur in both primary and metastatic lesions. The first is called "diffuse," in which the tumor grows without a clear architectural structure, replacing normal parenchyma. The second is "basal," in which nests of cancer cells are surrounded by connective tissue containing blood vessels and inflammation (which replaces normal parenchyma). The third is "papillary," in which papillae made up of a thin fibrous shaft, containing a new vessel, are lined by tumor cells to form a neoplasm. This latter growth pattern is mainly associated with primary alveolar carcinoma, and some remnants of normal lung structure can be seen in some areas of the tumor. Three non-angiogenic patterns of tumor growth can also be seen. The first pattern is called "alveolar" and is the most common type of non-angiogenic malignant lung lesion in both primary and secondary cancers, in which tumor cells fill the alveolar spaces and then pneumocytes disappear. It could be argued that in the initial stage, the cancer cells absorb the epithelium instead of the vasculature. The second pattern is "intercellular," which occurs only in lung metastases, where metastatic cells enter and grow in the alveolar walls, but do not enter the alveolar space. The third non-angiogenic pattern observed in the lung is "vascular encirclement" (also observed only in metastases), in which cells grow like a cuff around a larger vessel.



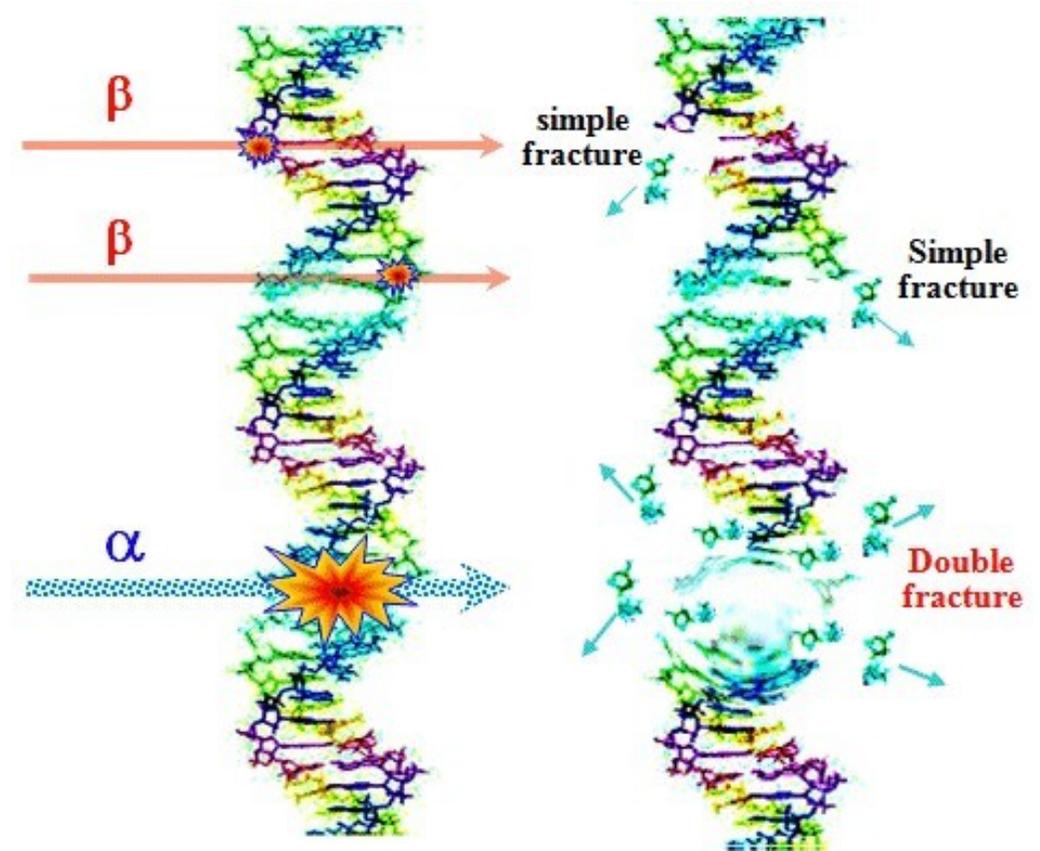
b | Schematic representation of tumor growth patterns in the liver. In the liver, blood flows from the portal vein and branches of the hepatic artery (located in the portal spaces), through the sinusoids to the central vein of the lobule. The hepatic sinusoids are vascular channels lined by specialized endothelial cells containing numerous pores (fenestrae) and scattered cells with phagocytic capacity, known as Kupffer cells. Between the endothelium and liver cells is the Disse space, where fluids can carry molecules from the hepatocytes into the blood flow and vice versa. Here, there are two types of tumor growth angiogenesis. One is known as "desmoplastic," in which tumor cells induce the growth of new vessels and are surrounded by a desmoplastic framework that separates them from normal liver parenchyma. The second is known as "pressure," in which the tumor also induces the growth of new vessels and presses directly on normal liver tissue, leading to compression of liver beads. In the type of non-angiogenic replacement growth seen in both primary and secondary liver tumors, tumor cells effectively replace hepatocytes, but preserve the liver architecture and interact with existing sinusoidal vessels. c | Schematic representation of tumor growth patterns in the brain. At least four different growth patterns have been described for brain tumors that use both vascular cooption and angiogenesis, or potentially mixtures of both mechanisms. In the "single cell or minisphere infiltration" pattern, single cells or small clusters of cells spread through the brain parenchyma without contacting existing vessels and without inducing the formation of new vessels. In the pattern of "co-optive infiltration" of tumor cells at the periphery of the lesion, or even away from the main lesion, progress by using normal vessels (vascular encirclement). It is currently unclear whether deeper areas of brain tumors with these two patterns can also activate angiogenesis. Two other growth patterns in the brain are potentially angiogenic: the "non-infiltrative or displacement" pattern, which has a discrete boundary between the tumor and the parenchyma, while another pattern, dubbed "infiltrative," has irregular edges with irregular columns of cells that infiltrate the parenchyma and induce the formation of new vessels. All of these patterns can be observed in both primary and metastatic brain tumors.

The lack of electric charge of neutrons means that they do not interact electromagnetically with atoms in a material in the same way as alpha or beta rays. For this reason, neutrons can penetrate matter, bypassing most obstacles such as electrical charges in atoms.

**Neutron energy:** Higher neutron energy usually means a greater penetration range, since neutrons of higher energy can penetrate the material more deeply, with less likelihood of absorption or scattering.

**Material:** Different materials behave differently toward neutrons. Materials with higher atomic weight or density may absorb neutrons better, limiting their penetration range. For example, lead is a good neutron absorber.

**Lack of charge of neutrons:** The lack of electric charge of neutrons means that they do not interact electromagnetically with atoms in a material in the same way as alpha or beta rays. For this reason, neutrons can penetrate matter, bypassing most obstacles such as electrical charges in atoms.



If we assume that neutrons and gamma rays have similar energy, a beam of neutrons will penetrate deeper through the material.

A neutron beam will penetrate a material to a greater depth than gamma rays because neutrons are electrically inert particles, which means they do not interact with material atoms through electromagnetic force as gamma rays do. Gamma rays can be inhibited or absorbed by electromagnetic interaction with material atoms, especially if the material has large amounts of electrons. Neutrons, on the other hand, can penetrate a material without much interference unless they undergo absorption or scattering by nuclear reactions.

Lead, due to its properties, can help shield gamma radiation, but is not an effective material for shielding neutron radiation. Lead can even increase the effects of neutron radiation by converting some thermal neutrons into intermediate and fast neutrons, which are more harmful to the body.

Therefore, other materials, such as those rich in hydrogen, are usually used to shield neutron radiation.

The conversion of thermal neutrons into fast neutrons, also known as fast neutrons, can occur during collisions with atomic nuclei in a high-mass material, such as a neutron-absorbing material such as a moderator in a nuclear reactor.

Thermal neutrons are low-energy neutrons that travel at speeds close to those of particles under normal conditions. When thermal neutrons collide with atomic nuclei, absorption of the neutron by the atomic nucleus can occur. As a result of this collision and the absorption of neutron energy by the nucleus, the nucleus may undergo a nuclear reaction, leading to the release of fast neutrons.

The fast neutrons that result from these reactions have higher energies than thermal neutrons. They can have sufficient energy to initiate subsequent nuclear reactions, which is important in the context of maintaining a chain reaction in nuclear reactors.

A material in which gamma rays will pass farther than other materials is one that has a lower atomic density and a lower atomic number. Atomic density refers to the number of atoms per unit volume, and atomic number tells the number of protons in the atomic nucleus of an element.

Typically, materials with lower atomic density and lower atomic number tend to inhibit or absorb gamma radiation more weakly. Examples of such materials include organic materials such as plastics, wax or wood.

Therefore, in organic materials, which have relatively low atomic density and atomic number, gamma rays can penetrate farther than in more dense materials such as lead or concrete. However, the penetration range of gamma rays also depends on other factors, such as the energy of the radiation and its characteristics. In summary, lead shields needed to protect against neutron radiation will typically be thicker than those that are effective in protecting against gamma radiation of the same energy, due to the different mechanisms by which these types of radiation interact with the shielding material.

Gamma radiation tends to interact with electrons in the body and with electric and magnetic fields. It can lead to damage in tissues at the cellular level, including DNA damage, which can in turn lead to cancer and genetic diseases.

Neutron radiation mainly interacts with atomic nuclei in the body through nuclear reactions. This can lead to the formation of radioactive isotopes and direct damage at the cellular level, which can also lead to cancer and other diseases.

Which radiation is more harmful can depend on a variety of factors, such as tissue type and sensitivity to radiation, exposure time, type of radiation (e.g., gamma or neutron), and other individual factors. It is also important to note that radiation risk assessments usually take into account different exposure scenarios and doses, not just the type of radiation.

The bulk nature of neutrons can pose some obstacles to their penetration range, as neutrons can be scattered and absorbed by atomic nuclei in the material through which they pass.

However, if they have sufficient energy and encounter low atomic mass material they can penetrate the material much deeper than gamma rays. The penetration range of neutrons depends not only on their mass, but also on their energy and the properties of the material through which they pass.

A material in which gamma rays will pass farther than other materials is one that has a lower atomic density and a lower atomic number. Atomic density refers to the number of atoms per unit volume, and atomic number tells the number of protons in the atomic nucleus of an element.

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# RADIOTHERAPY - WITH NEUTRONS

## Neutron Therapy

Neutron Therapy at Fermilab

[https://www.youtube.com/watch?v=X3SfQwMyjtw&ab\\_channel=NorthernIllinoisUniversity](https://www.youtube.com/watch?v=X3SfQwMyjtw&ab_channel=NorthernIllinoisUniversity)

## Boron Neutron Therapy.

Manufacturing Excellence at SHI vol8 Boron Neutron Capture Therapy (BNCT) system\_CG

[https://www.youtube.com/watch?v=NrGtA2Cd3\\_k&ab\\_channel=SumitomoHeavyIndustriesCorporateCommunications](https://www.youtube.com/watch?v=NrGtA2Cd3_k&ab_channel=SumitomoHeavyIndustriesCorporateCommunications)

## Animation of Boron Neural Therapy

Boron Neutron Capture Therapy Animation

[https://www.youtube.com/watch?v=2yI\\_SoOWFMU&ab\\_channel=Nymus3D](https://www.youtube.com/watch?v=2yI_SoOWFMU&ab_channel=Nymus3D)

## Demonstration of Neutron Boron Therapy Apparatus

Demonstrating Boron Neutron Capture Therapy

[https://www.youtube.com/watch?v=xTO54SaJQu8&ab\\_channel=TheJapanTimes](https://www.youtube.com/watch?v=xTO54SaJQu8&ab_channel=TheJapanTimes)

## BNCT - a new radiation therapy technology

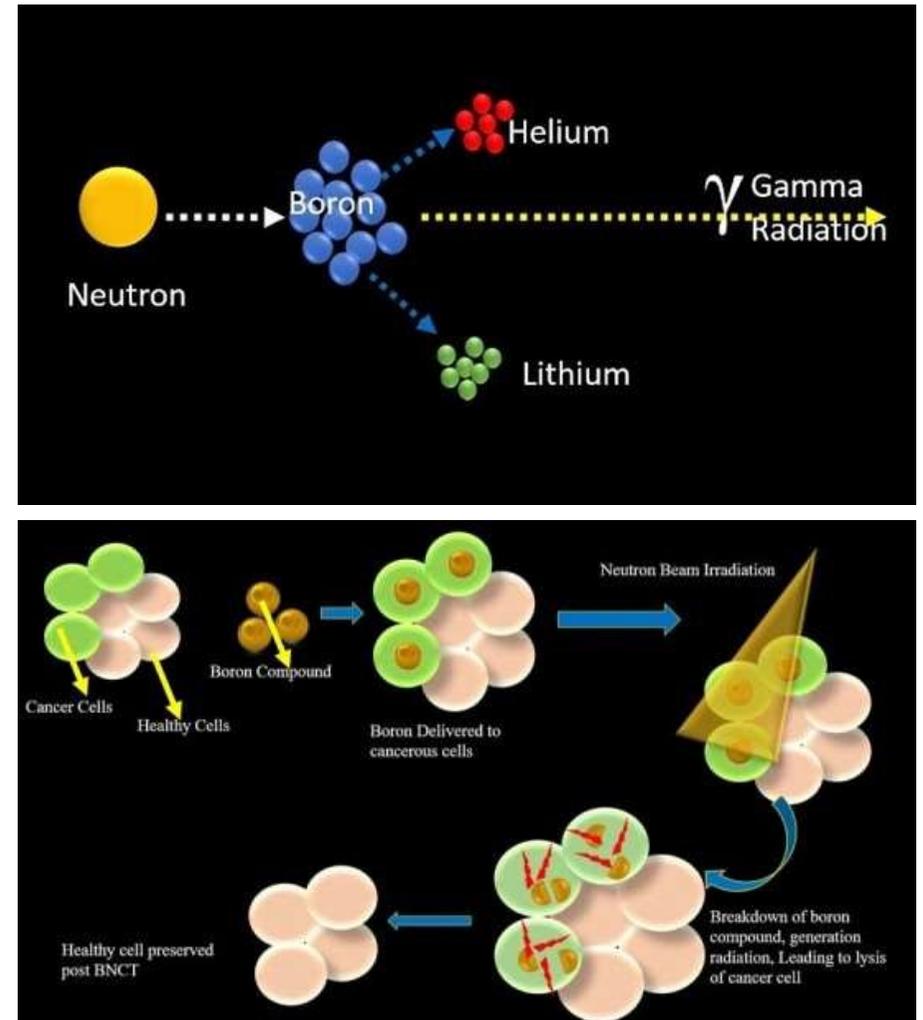
BNCT - the newest 4th generation cancer therapy

[https://www.youtube.com/watch?v=qTxO2-QJEhc&ab\\_channel=CurostechInc](https://www.youtube.com/watch?v=qTxO2-QJEhc&ab_channel=CurostechInc)

## Lecture - Dr. James Smith

Boron Neutron Capture Therapy (BNCT) of Cancer

[https://www.youtube.com/watch?v=7\\_ZgM\\_VkZ\\_s&ab\\_channel=JamesSmith](https://www.youtube.com/watch?v=7_ZgM_VkZ_s&ab_channel=JamesSmith)



## **RADIOTHERAPY - WITH PROTONS**

### **How does proton therapy work?**

How does Proton Therapy work?

[https://www.youtube.com/watch?v=MS590Xtq9M4&ab\\_channel=ProtonTherapyIBA](https://www.youtube.com/watch?v=MS590Xtq9M4&ab_channel=ProtonTherapyIBA)

### **Proton Tarapia - Varian**

Varian Medical Systems: Proton Center Tour

[https://www.youtube.com/watch?v=rSvEvvLsPU8&ab\\_channel=QuietIsland](https://www.youtube.com/watch?v=rSvEvvLsPU8&ab_channel=QuietIsland)

### **Proton therapy - Mayo Clinic**

Proton Beam Therapy - Mayo Clinic

[https://www.youtube.com/watch?v=OTd5dv3VDws&ab\\_channel=MayoClinic](https://www.youtube.com/watch?v=OTd5dv3VDws&ab_channel=MayoClinic)

## **RADIATION THERAPY - PROM. X**

### **How does the electra linear accelerator work?**

How a Linear Accelerator Works - Elekta

[https://www.youtube.com/watch?v=jSgnWfbEx1A&ab\\_channel=Elekta](https://www.youtube.com/watch?v=jSgnWfbEx1A&ab_channel=Elekta)

### **How the cyber knife works**

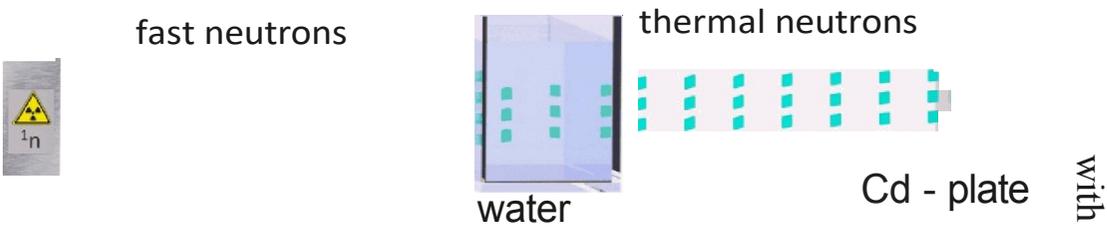
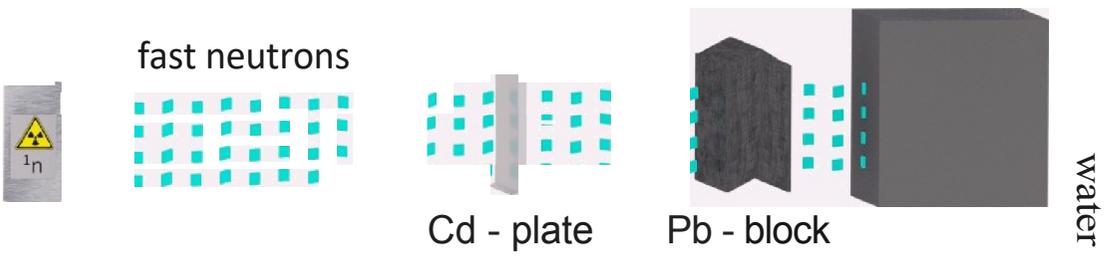
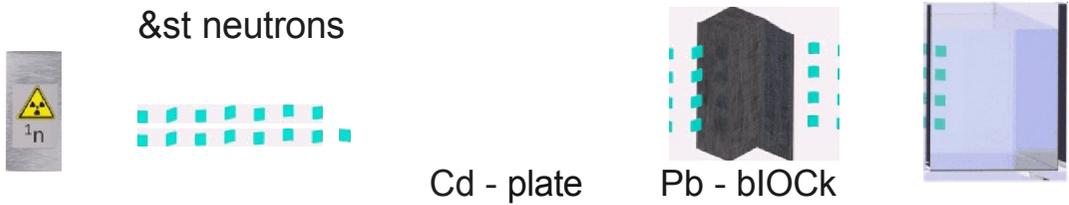
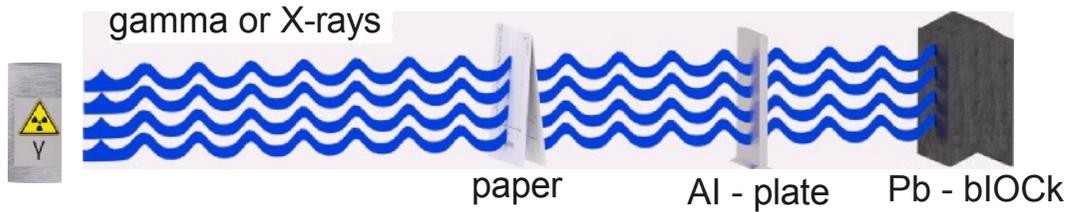
How CyberKnife Works

[https://www.youtube.com/watch?v=mcC0\\_azJ0rw&ab\\_channel=MountMiriamCancerHospital](https://www.youtube.com/watch?v=mcC0_azJ0rw&ab_channel=MountMiriamCancerHospital)

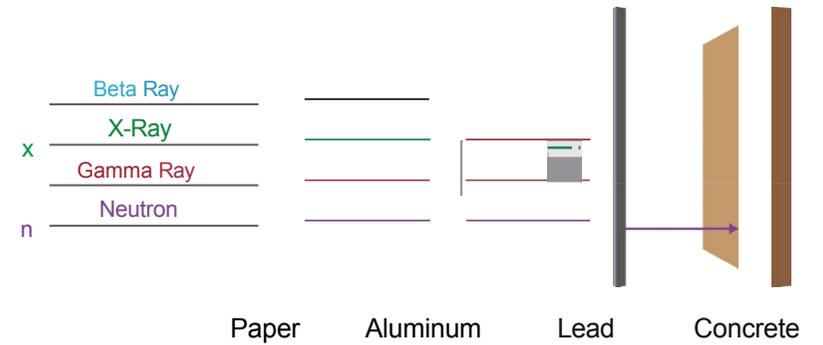
### **How gamma cybersurgery works - Elektra radiosurgery**

How a Leksell Gamma Knife® radiosurgery system works - Elekta

[https://www.youtube.com/watch?v=pxikNfTbtzw&ab\\_channel=Elekta](https://www.youtube.com/watch?v=pxikNfTbtzw&ab_channel=Elekta)



### Penetrating Power of Different Types of Radiation



### Neutron Energy Categories

Neutron Name/Title	Energy (eV)
Cold Neutrons	$0 < 0.025$
Thermal Neutrons	$0.025$
Epithermal Neutrons	$0.025 < 0.4$
Cadmium Neutrons	$0.4 < 0.6$
Epicadmium Neutrons	$0.6 < 1$
Slow Neutrons	$1 < 10$
Resonance Neutrons	$10 < 300$
Intermediate Neutrons	$300 < 1,000,000$
Fast Neutrons	$1,000,000 < 20,000,000$
Relativistic Neutrons	$> 20,000,000$

IAEA

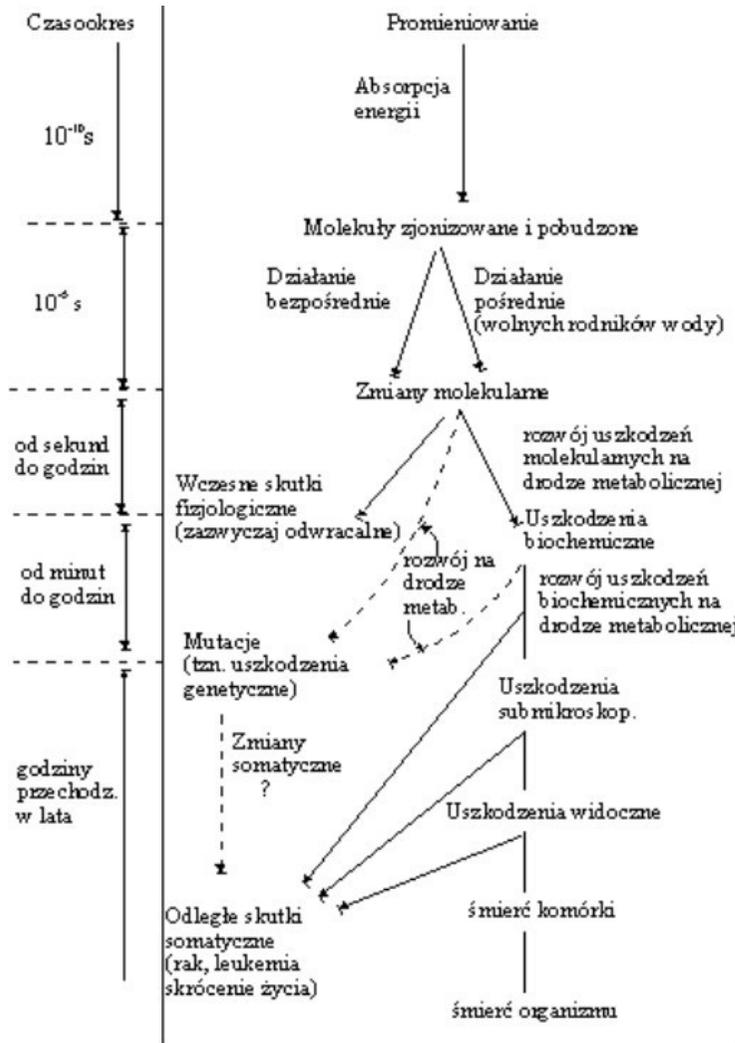
concrete

In some cases, the terminology "palliative radiation therapy" is used to refer to treatment that is used to alleviate the discomfort associated with advanced stages of cancer, or when the goal of treatment is symptom control rather than a complete cure.

"Symptomatic radiation therapy" can be used to refer to treatment that is used solely to alleviate specific symptoms associated with the cancer, without regard to the overall stage of the disease.

Despite these subtle differences in terminology, both approaches focus on improving the patient's quality of life by controlling cancer symptoms. It is important that the treatment plan is tailored to the patient's individual needs and condition, taking into account both the therapeutic goals and the expectations of the patient and family.

# TIME VS. BIOLOGICAL EFFECT



**Drogi odnowy**

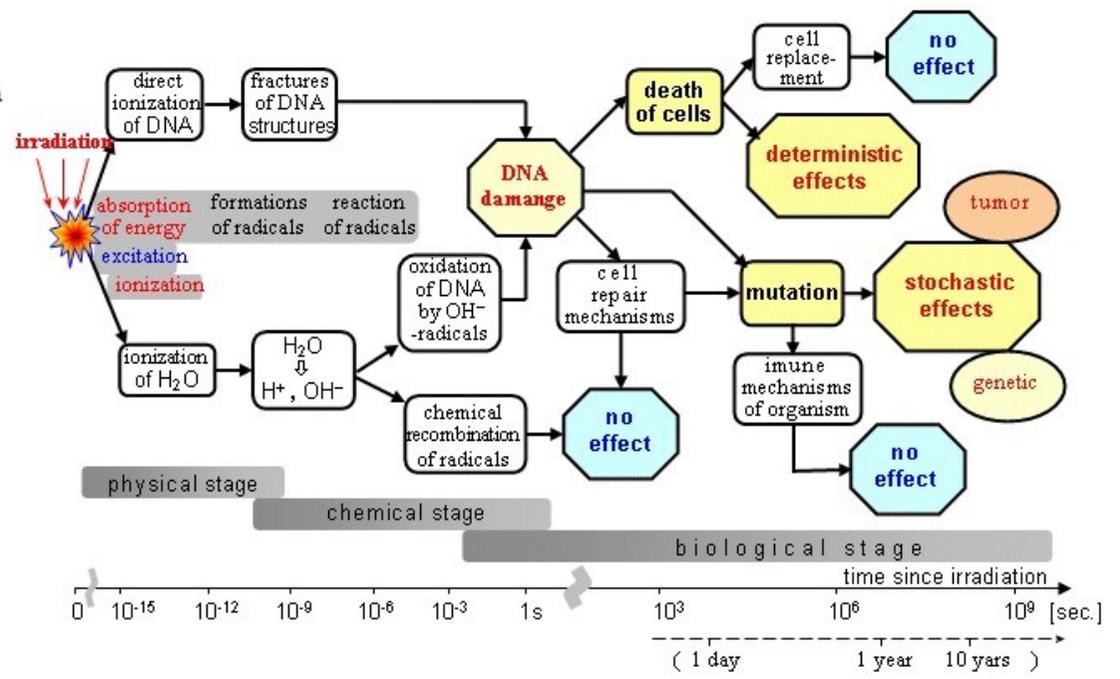
Modyfikacja uszkodzeń

Za pomocą chemicznych substancji ochronnych (efekt tlenowy)

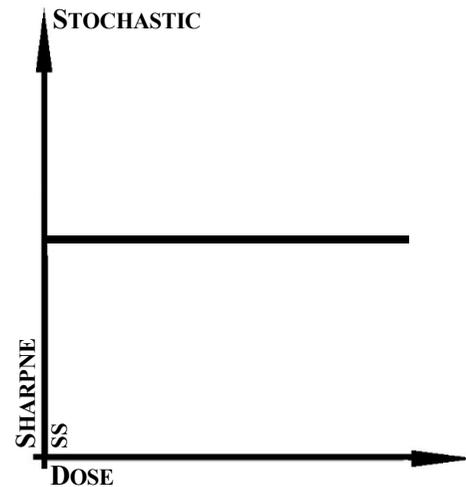
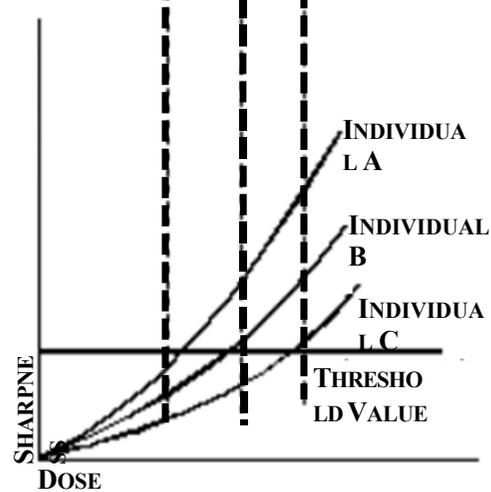
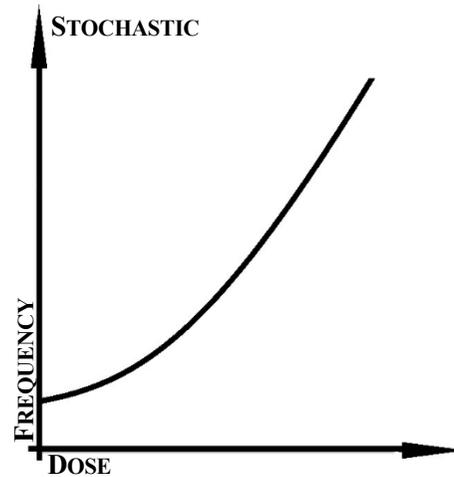
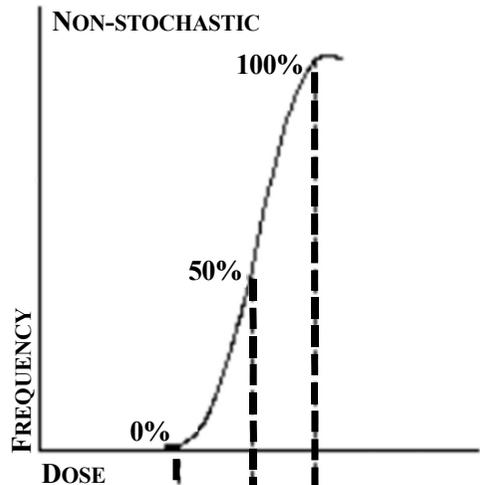
na drodze zmiany uszkodzeń biochemicznych

na drodze odnowy wewnątrzkomórkowej (również w odniesieniu do mutacji)

na drodze w ymiany kom. Regeneracja samoistna lub przeszczepienie



## EFFECTS OF RADIATION IN LIVING ORGANISMS



**Skutki deterministyczne (niestochastyczne)** to takie, których zarówno częstość jak i stopień ciężkości ulega wzrostowi z dawką promieniowania. Można określić dla nich dawkę progową. Należą do nich np. wszystkie dobrze znane powikłania radioterapii.

**Skutki stochastyczne**, to te, których częstość występowania ulega jedynie zwiększeniu ze wzrostem dawki. Są to zjawiska probabilistyczne. Nie istnieje dla nich dawka progowa. Przebieg wywołanej choroby nie jest już przez dawkę determinowany.

Organizm	Dawka [Gy]
Człowiek	4
Małpa	5 ÷ 6
Osiół	7,8
Świnia	3,5 ÷ 4,2
Koza	3,5
Kura	6 ÷ 10
Świnka morska	1,7 ÷ 4,9
Szczur	5,9 ÷ 9,7
Królik	7,5 ÷ 8,2
Mysz	4,5 ÷ 6,5
Żaba	7
Żółw	15
Ślimak	80 ÷ 200
Nietoperz	150
Drosophila	800
Pantofelek	3000
Bakteria: Micrococcus radiodurans	7000

LD<sub>50</sub> - tzw. dawka śmiertelna. Jest to dawka, która powoduje śmierć 50% liczby napromieniowanych osobników w ciągu 30 dni.

&GDRATICNR



1. Choroba popromienna  
b) przewlekła  
2. Miejscowe uszkodzenie skóry

1. Zmętnienie  
2. abezzotions  
somatycznych  
3. Nowotwory złośliwe  
4. Niepłodność  
5. Zachamowanie w zrostu i rozwoju

Dawka [Gy]	Narząd lub tkanka	
0,1	Plód	reatgeua
0,15	Jądra	Periodovvabeiplodo c
0,5	Szypik kostny	Zabmzenwbmoogovv
0,5	Cale ciało	Wymioty
0.5 ÷ 2	Soczewka	Zmętnienie
3	Skóra	Depilacja, rumień
2.5 - 6	Jajniki	B ãóoo
30-6	Jądra	BezbJro b
	Soczewka	Zaémz
10	Płuca	ZapaleoiepAzc..w:s':soaóiiü
10	Tarczycyca	Niedocsyoo%Ltamzycj:

Tj zzwomot-ófi and uaziejsrowiexie	R-ccie,tny okrrs utajenia (lata)	hliniznal period utajenia (lata)
Białaczki	7-10	2 - 3
Mięsaki kości	10 - 15	3 - 4
Mięsaki tkanek miękkich	12	
Imózp 'Dm	14	ok. 10 lat
No* trcz/://	20	dla pozostałych
No pr	20	nowotworów
Raki gruczołu piersiowego	2?	
Raki płuc	?5	
Guzy mozgu (glejaki)	27	

## ACTIVITY AND DOSAGE

### ACTIVITY (A)

The activity of a radioactive source **A** is defined by the number of nuclear transformations occurring in this source per unit time:

$$A = -\frac{dN}{dt}$$

Where **N** is the number of radioactive nuclei, and **dt** is the time interval during which the number of these nuclei decreased by **dN**. The minus sign just indicates the decreasing number of nuclei due to the radioactive decay taking place.

In the SI system, the unit of activity is the becquerel [Bq]. A becquerel is the unit of activity of a source, in which one nuclear transformation occurs in one second

### EXPOSURE DOSE (X)

**X** is a measure of the degree of ionization of air by passing radiation. The degree of ionization is defined as the absolute value of the sum of the charges of ions of one sign **dQ** produced by the radiation in a unit mass of air **dm**:

$$X = \frac{dQ}{dm}$$

In the SI system, the unit of exposure dose is the coulomb per kilogram [C·kg<sup>-1</sup>].

### DOSE ABSORBED (D)

Ionizing radiation as it passes through a medium loses its energy. The measure of radiation absorption is the so-called absorbed dose. Thus, the absorbed dose **D** is the amount of energy **dE** of ionizing radiation absorbed by a unit mass **dm** of matter:

$$D = \frac{dE}{dm}$$

In the SI system, the unit of absorbed dose is the gray [Gy]. 1 Gy = 1 J · kg<sup>-1</sup>

### DOSE EQUIVALENT (H)

The concept of absorbed dose **D** refers to the absorption of radiation by various materials, including the tissues of the human body. During the irradiation of living organisms, biological processes that depend on the type of radiation occur in their tissues in addition to physical phenomena. Therefore, in order to determine the degree of energy absorption by a living organism taking into account the biological effects that different types of ionizing radiation cause, the concept of dose equivalent was introduced. The dose equivalent **H** is expressed by the formula:

$$H = Q \cdot D$$

Where **Q** is the so-called radiation quality factor depending on the type of radiation. The SI unit of dose equivalent is the sievert [Sv]. 1 [Sv] = 1 [Gy] · Q

Rodzaj promieniowania	Współczynnik Q
X i γ powyżej 30 keV	1
β powyżej 30 keV	1
β trytu	2
α, neutrony, protony, jony ciężkie	25
neutrony termiczne	4,5

## DOSAGE POWER

*If the dose value changes over time, it is convenient to use the concept of dose power.*

### EXPOSURE DOSE STRENGTH

The power of exposure dose **X** is defined as the increase in exposure dose **dX** over time **dt**:

$$X = dX - dt$$

The SI unit of exposure dose power is amperes per kilogram ( $1C/kg\cdot s = 1 A/kg$ ). In non-SI units, the power of the exposure dose has been measured in x-rays per hour [R/h] or milliRöntgen per hour [mR/h].

### POWER OF THE ABSORBED DOSE

is the incremental absorbed dose **dD** over time

$$dt: D = dD - dt$$

The SI unit of absorbed dose power is the gray per second [Gy-s<sup>-1</sup>].

### DOSE EQUIVALENT POWER

Similar to the power of the exposure and absorbed dose, one can determine the power of the dose equivalent **H**. This is the ratio of the increase in the dose equivalent **dH** over time

$$dt: H = dH - dt$$

The SI unit of dose equivalent power is sievert per second [Sv-s<sup>-1</sup>]. The off-system unit was rem per hour [rem-h<sup>-1</sup>].

WIELKOŚĆ	JEDNOSTKI		Związek pomiędzy jednostkami
	używane obecnie	używane dawniej	
Aktywność	Bq [s <sup>-1</sup> ]	Ci	1 Ci = 3,7 · 10 <sup>10</sup> Bq 1 Bq = 27 pCi
Dawka ekspozycyjna	C/kg	R	1 R = 2,58 · 10 <sup>-4</sup> C/kg
Moc dawki ekspozycyjnej	A/kg pA/kg	R/h mR/h	1 R/h = 7,17 · 10 <sup>-8</sup> A/kg 1 A/kg = 1,4 · 10 <sup>7</sup> R/h 1 pA/kg = 0,014 mR/h
Dawka pochłonięta	Gy [J/kg] cGy	rad	1 rad = 0,01 Gy 1 rad = 1 cGy
Moc dawki pochłoniętej	Gy/s [W/kg] cGy/h μGy/h	rad/h mrad/h	1 rad/h = 2,78 · 10 <sup>-6</sup> Gy/s 1 rad/h = 1 cGy/h 1 mrad/h = 10 μGy/h
Równoważnik dawki	Sv mSv	rem mrem	1 rem = 0.01 Sv 1 mSv = 100 mrem
Moc równoważnika dawki	Sv/s Sv/h mSv/h	rem/h mrem/h	1 Sv/s = 36 · 10 <sup>4</sup> rem/h 1 Sv/h = 100 rem/h 1 mSv/h = 100 mrem/h

### RADIATION PROTECTION DOSE MONITORING

- effective loading dose equivalent
- other derivatives indicators determining risk      ionizing radiation:

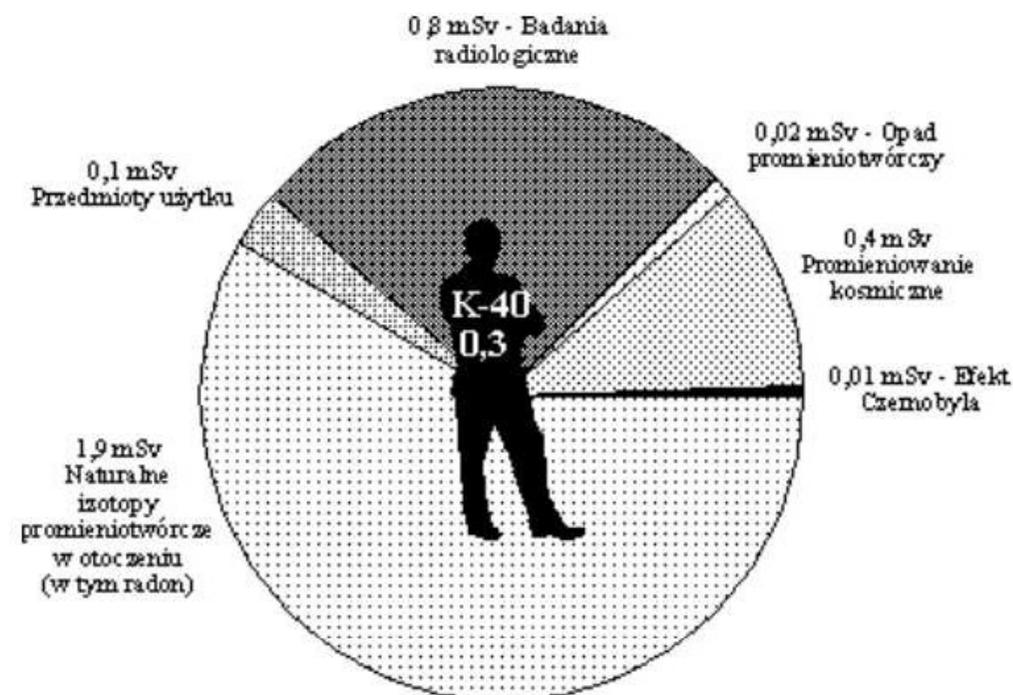
**ALI** (Annual Limit on Intake) annual intake of a radioactive substance by ingestion or inhalation (in Bq)

**DAC** (Derived Air Concentration) concentration of radioactive substances in the air (in Bq/m<sup>3</sup>)

## DOSES IN X-RAY AND POLLUTION

Lp	Rodzaj badania rtg	Średnia dawka powierzchniowa na zdjęcie [mSv]		Redukcja rocznego kolektywnego równoważnika dawki [man · Sv]
		1984	1995	
1	kręgosłup lędźwiowy AP	9,6	6,1	284
2	kręgosłup lędźwiowy LAT	23,2	16,0	121
3	staw lędźwiowo-krzyżowy LAT	39,7	29,0	45
4	klatka piersiowa PA	0,23	0,16	56
5	jama brzuszna AP	8,2	5,6	399
6	miednica AP	6,5	4,4	382
7	czaszka AP	4,9	3,0	38
8	czaszka LAT	2,6	1,5	19
9	kręgosłup piersiowy AP	6,2	4,7	58
10	kręgosłup piersiowy LAT	14,0	13,0	15
		wartość średniego iloczynu [Gy x cm <sup>2</sup> ] na badanie		
11	przewód pokarmowy (z barytem)	18,8	13,0	652
12	wlew doodbytniczy	41,0	25,8	1285
13	dożylna urografia	30,9	13,4	1373
Całkowita redukcja rocznego kolektywnego równoważnika dawki o				4727

\*- dla wielkości wyrażonej iloczynem [Gy x cm<sup>2</sup>] brak jest polskiego odpowiednika angielskiego określenia „area exposure products”.



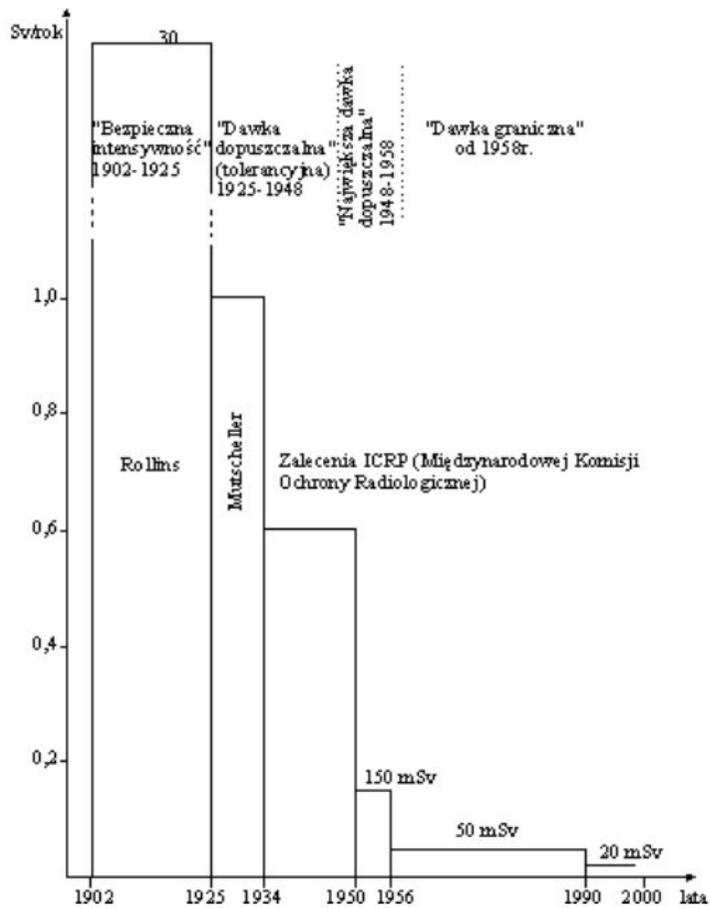
Rys. 8.1. Orientacyjne dawki roczne w [mSv] od różnych źródeł promieniowania. Źródła naturalne - 2,6 mSv, źródła sztuczne - 0,9 mSv

## RADIATION PROTECTION - SENSITIVITY OF ORGANS

Narząd lub tkanka	Współczynnik ryzyka ( $10^{-2} \text{ Sv}^{-1}$ )	
	Cała populacja	Pracownicy
Pęcherz	0,29	0,24
Szypik kostny	1,04	0,83
Powierzchnia kości	0,07	0,06
Piersi	0,36	0,29
Okreźnica	1,03	0,82
Wątroba	0,16	0,13
Płuca	0,80	0,64
Przetyk	0,24	0,19
Jajniki	0,15	0,12
Skóra	0,04	0,03
Żołądek	1,00	0,80
Tarczycyca	0,15	0,12
Pozostałe	0,59	0,47
Razem	5,92	4,74
<b>Ryzyko ciężkich uszkodzeń genetycznych</b>		
Gonady	1,33	0,80
Razem - skutki stochastyczne	7,3	5,6

Dawka pochłonięta GV	EFEKTY
O-021	No effects nyMavalnxzli kiiuiczuie
0.25 - 1.0	Niezxiaezzie przejćiov:e reduction in the number of iimfocxu and leukocj9ón- oño;et- nocflonnx cf. Rzaólró uw stepujująca objawy choroł' poo-o'iujące nieztoł- now do dzia³: o oby e cowace sa ca general zÓo do wydania norizhr.-cb tasks
1.0 - ? 0	Nausea and sensation from ięczechcia, wunx oty- mog-a ow'strpic after dan-ce higher o-d. 1.25 M' among 20 - 23 ^i napromienionvef. Zmnie {'--mni'' zbv limfocx'- <b>tów i obojętnych granulocytów z opóźnionym powrotem do wartości prawidłowych</b>
2.0 - 3.0	Udno i i u=rmioty w -iagi pier'n-zeeo day. Obrec uta;en z earlynsvh objawóo- 'in tveoÓu or even dłużsm-. After the Period of utajeoi poj zvrizj a xie objanw o umizi'kOH53E¥In nzxilenie: loss of lzbrzeze and og'ilx.e sense of sickness, bói <b>gUdil</b> 'Dlżdość oo"-lo¥, uwbroc m,ux-, biegóóku, mcxii:zziac niezziac meeo slozraia. R'vzdooUnie praufiopodobnie o- rotate 3 mie e- m-. ;if you ujamoi not wphm- zleeo xtanu zdro-n that before npromienioiva- <b>ni-r</b> or oie wwxtpia help <b>ari a o-</b> ow' u zz)s aženiz :ub umzów
3.0 - 6,0	Bored*.ci. wnnnion and bieaxtnY a s: "iaau piariv:m-c b )ii1Yu _-hours. Period uta eniz *cz owwznvch objau-'io- even o Mugo i l tvgo'inia. ti ata > 'O,m iS !"MDRRIR QAA3¿AA" ? "UC 0 "ÓI * @OI@CTLD R' CIZ SO rveoozii . f³órnex towzm:-nn: kr awienia. base brn-ołoczca. ww-broc mo'. jamv uxmej ignition and gaidzieh. miewa and claim in ti on n'eo- day. Pojaow5a no - rmpanks z_-onóo- w obre e 2 -6 tveodni, possible G?" deaths among eY sponcrwwveh. 1 t'irzn' received a dose of mo:o 4. 3 Gv and dm p-ozostarcz b cza relion-but sceccli o¥olo lx poow thatł fi mieuem-.
6,0 i więcej	Nausea. Owniioth- i run nkz in ciae u pieiw:zy cJs several eodztñ. Short period of urajeniz i o- some ch pwpa'i acb brnY ;zsno obre onvef obj obj awóo- in the bodyu pien - co n-eoóniz. Biegunkz ¥rn-otoYi. fi aza )im oiocma {oz-. .me ow Óocmvv). Zap aleuie throat and yamv mtce,; rorach at the end of the first:ze co tu-goóniz. Smbbie destruction and erc o- diveim h godniu of the familiar ozekszfñ i. until l0b'e nlrspoaoñ wx-ch people.

COURSE- HOROBY ASKJIN TJI O OB. xToer nzižxxs-Nlżst'cm . "o:ioVZ W HIROSZIMIE I NAGASAKI				
D.1HN4	Pierwszy T'day	Second Tvdzien	Third week	Lic zba Zq^DDÓw
4,5 - 6 €i;-	4l'ileć i wych' Goraczk m zoatiz Drgan¥. biepuóY a U*zkmfñnie brtnni <b>Leukopenia</b>	G-orachli a Leukooenia Anemia V'vp*dznie T.'!o-		100% w pierwszym i drugim tygodniu
3-4.* %'	Mdłóści i wymioty Bmlbloieoia louzerie	Erac A a Leukoc'enia /oemia	BraL 1 GoiiczYa . B iR- guiAa wlosów, <b>Leukopenia</b>  Anemia	5 (i "i from the third tygodniae o
2-3 Gy	The post Zolgdłom-o-elite. Mdłóści i wymioty Brak łaknienia. Biegunka	<b>Leukopenia</b>	Brak łaknienia, Goiqcsła. Bie- s-m a, spzóa e mb:ón-. Leufop ,  Wewnętrzne krwawienia	%isa than 10'z e':zó:im goómu or later
1 - from €ii-	Post: zoladşon'o- jelitowa	<b>Leukopenia</b>	Gorączka, wypadanie wlo-  <b>Leukopenia, Uszkodzenie krtni R' melrzae krwawienia</b>	Nie wystąpiły



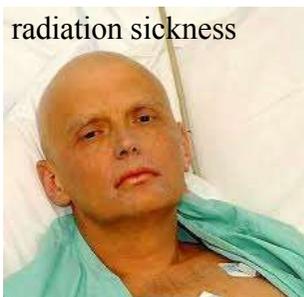
leukemia,  
anaplastic ischemia - radiation sickness



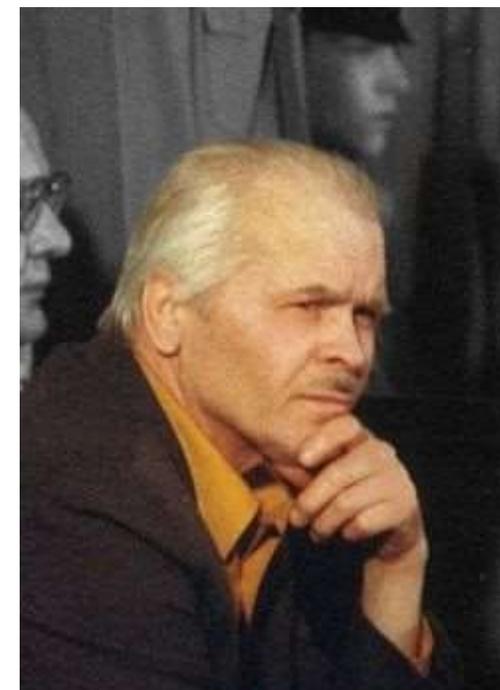
Tsutomu Yamaguchi



leukemia



radiation sickness



Anatoly Stepanovich Diatlov

**TASK... 1 pt to pass !!!**

# DOSIMETERS



RADIATION MONITORING SYSTEM



RADIATION MONITOR



GAMMA AND X-RAY DOSIMETERS.



RADIATION MONITORS



MOBILE RADIATION SCANNING SYSTEM



DOSIMETER-RADIOMETER OF ALPHA, BETA, X-RAY RADIATION, GAMMA AND NEUTRON

<https://www.tals.eu/dosimeters>

# PERSONAL DOSIMETERS

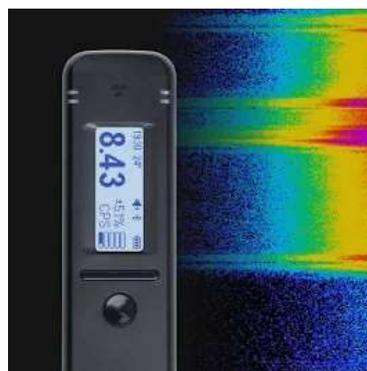
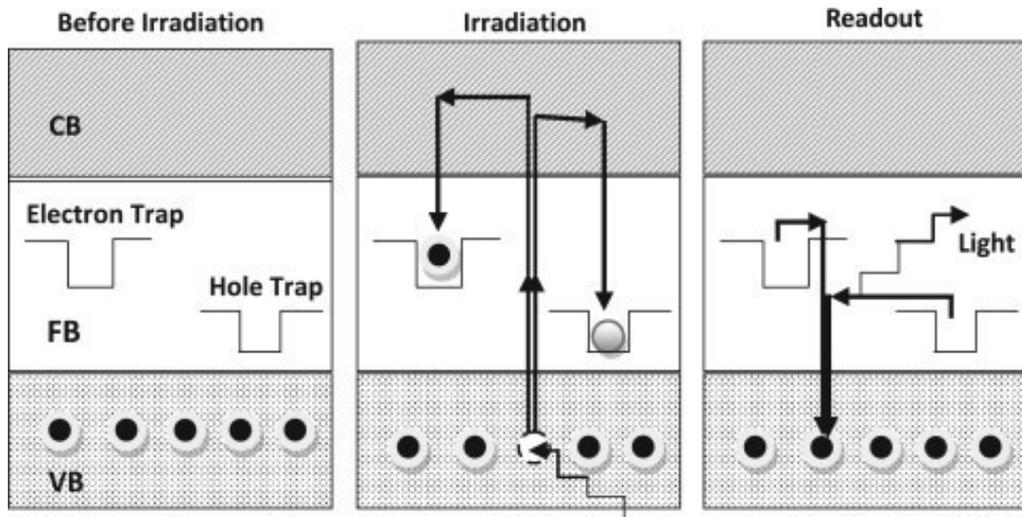


1. BETA RADIATION INDEX
2. SHIELDING FRAME
3. DIRECTIONZKAZ
4. METAL FILTER
5. FASTENER
6. FILM INSPECTION HOLE
7. NAMEPLATE IMPRINT
8. HALF-SHADOW FILTER
9. PLASTIC FILTER
10. TRANSPARENT FRONT (COVER)
11. ATTACHMENT CLIP \* STAGGERED ATTACHMENT TO THE FRONT (LID) AND BACK (SHELL).



ADDITIONAL ITEM FOR THE EXAMINATION: PRINCIPLE OF OPERATION - FIRST 3 PEOPLE

# PERSONAL THERMOLUMINESCENT DOSIMETERS



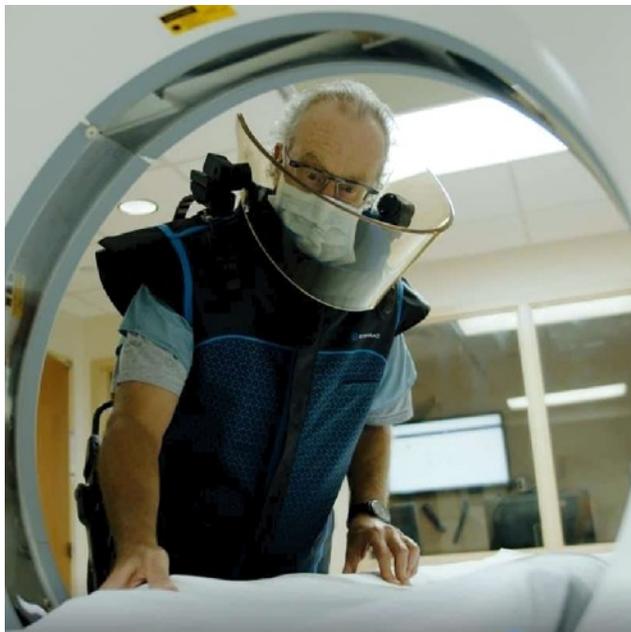
SERIAL NUMBER      HAND AND FINGER  
 NAME                      RING SIZE  
 DATE OF INSERTION      EMPLOYEE REGISTRATION NUMBER      SERIAL NUMBER



# INDIVIDUAL SHIELDS - PATIENT







Keeping Physician Radiation Dose As Low As Reasonably Achievable (ALARA)



Maxilizing Protection while Negating the Weight



**VISOR** 0.30 mm lead eq. (91.5% attenuation at 100 kVp)

**INTEGRATED THYROID COLLAR** 0.50 mm lead eq. (97.2% attenuation at 100 kVp)

**EXPANDED PROTECTION** 0.50 mm lead eq. on both front and sides. 1.0 mm lead eq. in front overlap area

**PROPRIETARY EXOSKELETON** Removes up to 100% of the weight from the user's body

