Krasnoyarsk State Medical University Named After Professor V.F. Voino-Yasenetsky
Department of Pathological Anatomy Named After Professor P.G.Podzolkov

Lecture 5.

TUMORS

Contents:

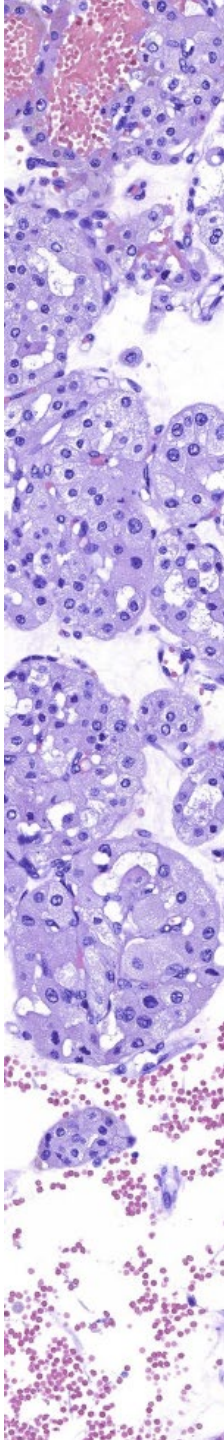
Part 1.

General information, risk factors for tumor development, classification principles.
Pathogenesis, progression, morphogenesis of tumors.
Metastasis.
Benign and malignant tumors.

Part 2.

Tumors of the squamous and the glandular epithelium.
The impact of the tumor on the body, complications, causes of death of cancer patients.
Morphological diagnosis of tumors.
Tumors from tissues - derivatives of mesenchyme, neuroectoderm, melanin-producing tissue.

Lector: Vladimir A. Khorzhevskii
Candidate of Medical Sciences (PhD),
Head of Pathology Department named after professor P.G. Podzolkov



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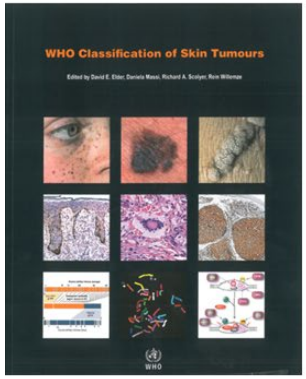
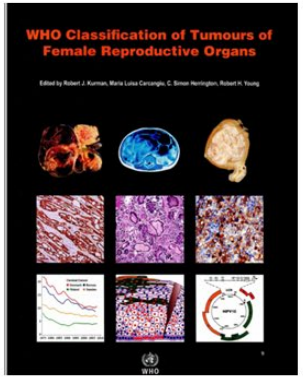
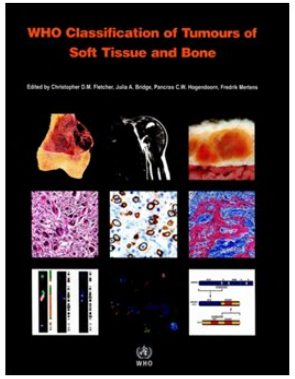
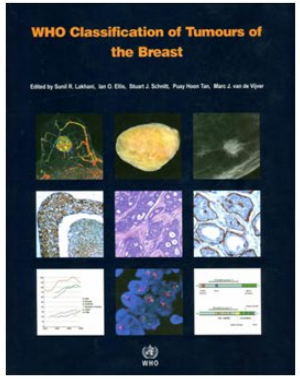
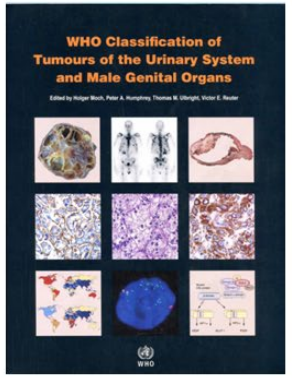
nomenclature of tumors

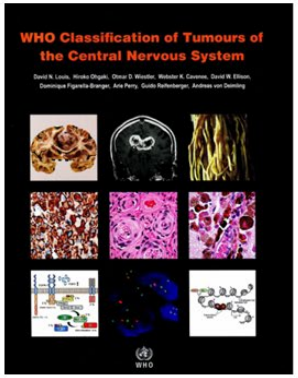






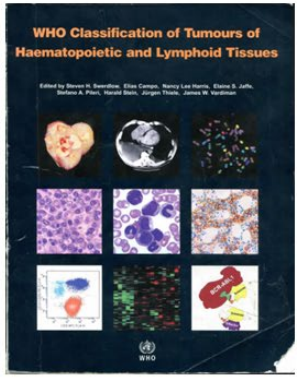


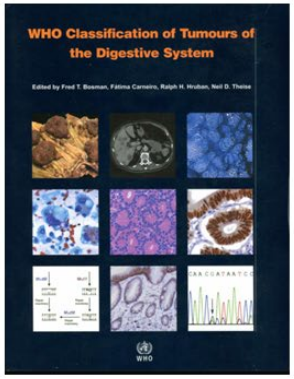


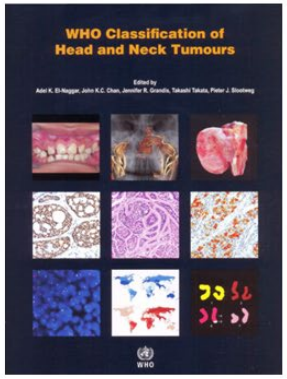


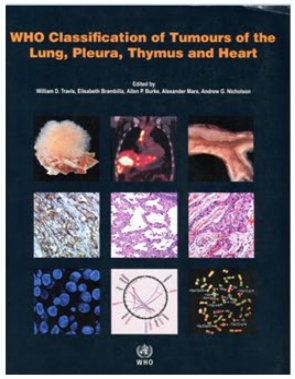
WHO OMS

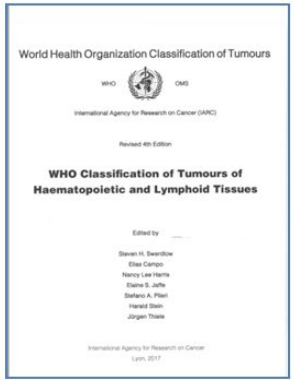
International Agency for Research on Cancer (IARC)

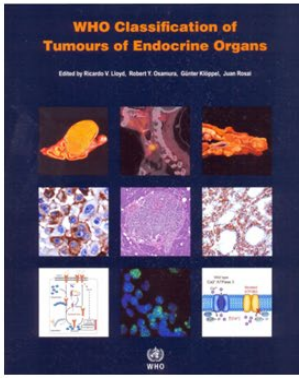








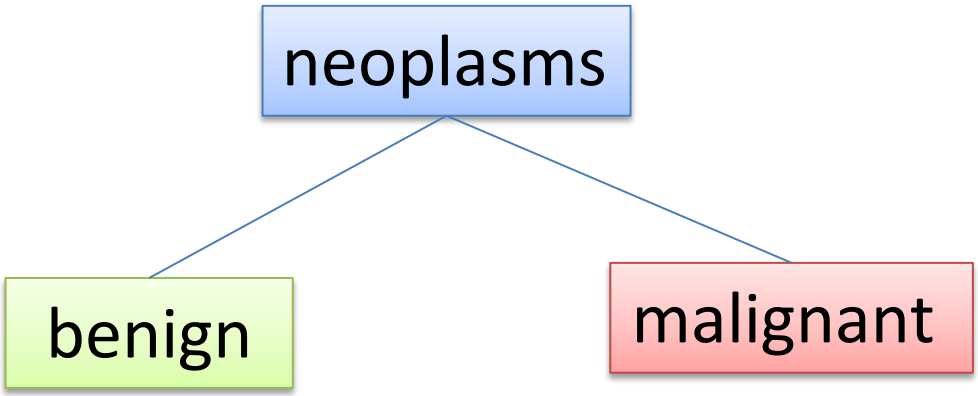




Tumor (*Neoplasia)*

- A tumor is tissue that grows in excess of and is not consistent with the growth of normal tissues and remains as excessive after the termination of the stimuli that caused this growth.

In common medical usage, a neoplasm often is referred to as a *tumor, and the study of tumors is called oncology* (from *oncos, "tumor," and logos, "study of").*



Benign tumor

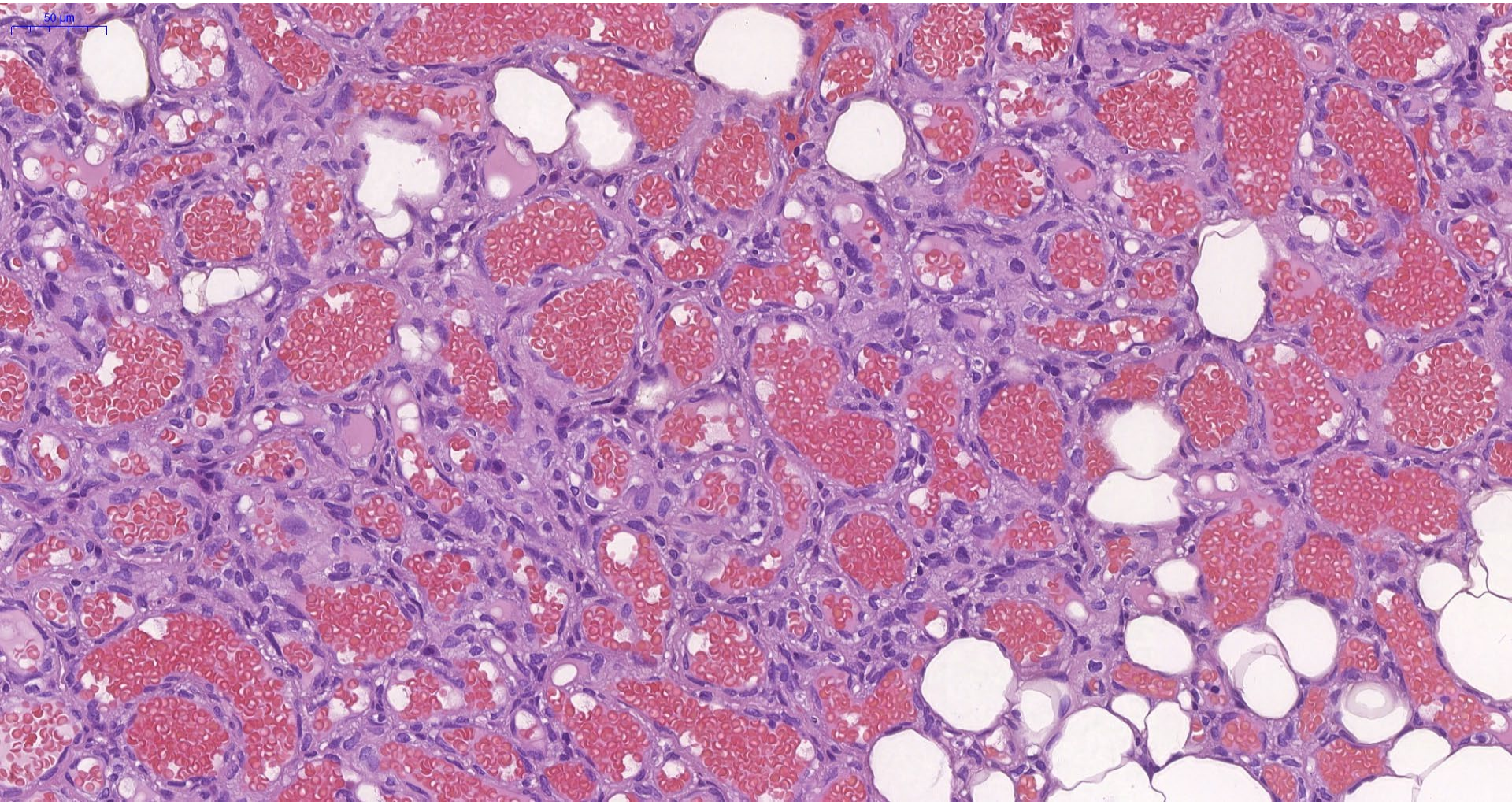
- **its microscopic and gross characteristics are considered to be relatively innocent, implying that it will remain localized and is amenable to local surgical removal.**
- **these are tumors do not have metastatic potential.**

benign tumors are designated by attaching the suffix *-oma to the cell type from which the tumor arises.*

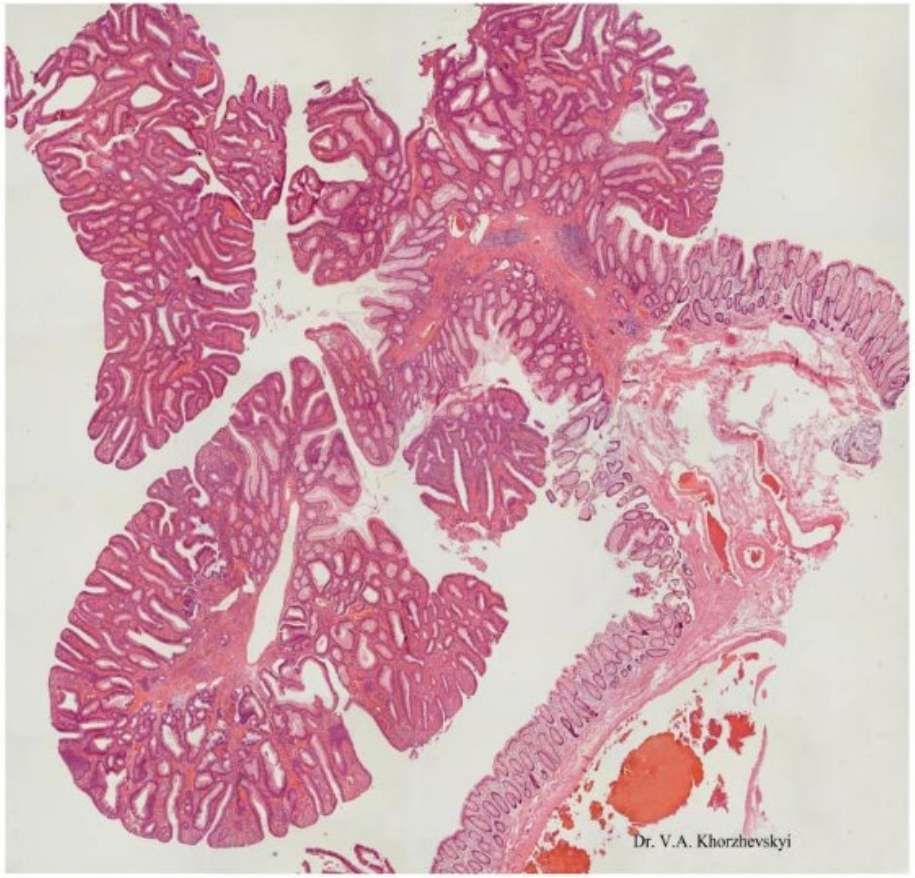
benign tumor arising in:
- fibrous tissue = *fibroma;*
- cartilaginous tumor = chondroma.

There are exceptions that can only be remembered (for example, mesothelioma is a malignant tumor from the mesothelium, lymphoma is a malignant tumor of lymphoid tissue, etc.).

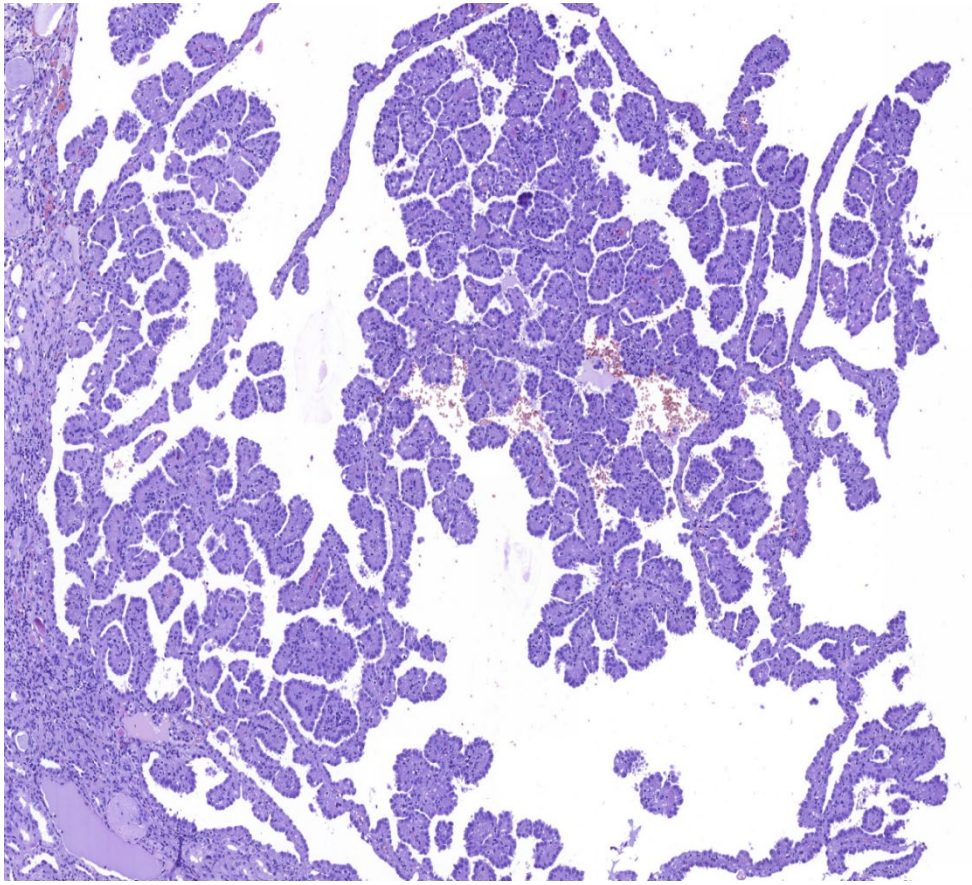
hemangioma



Intestinal adenoma



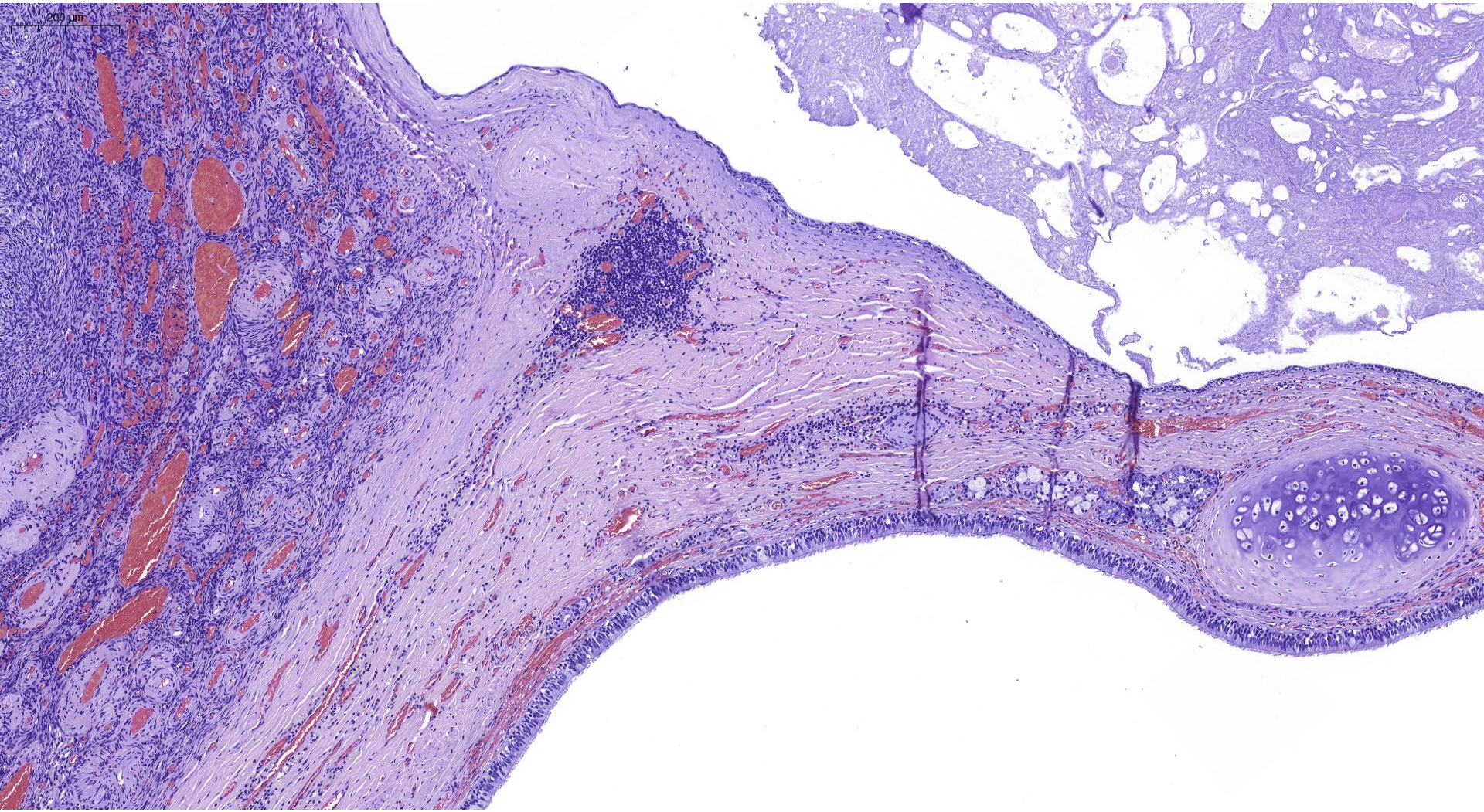
Papillary adenoma of the kidney



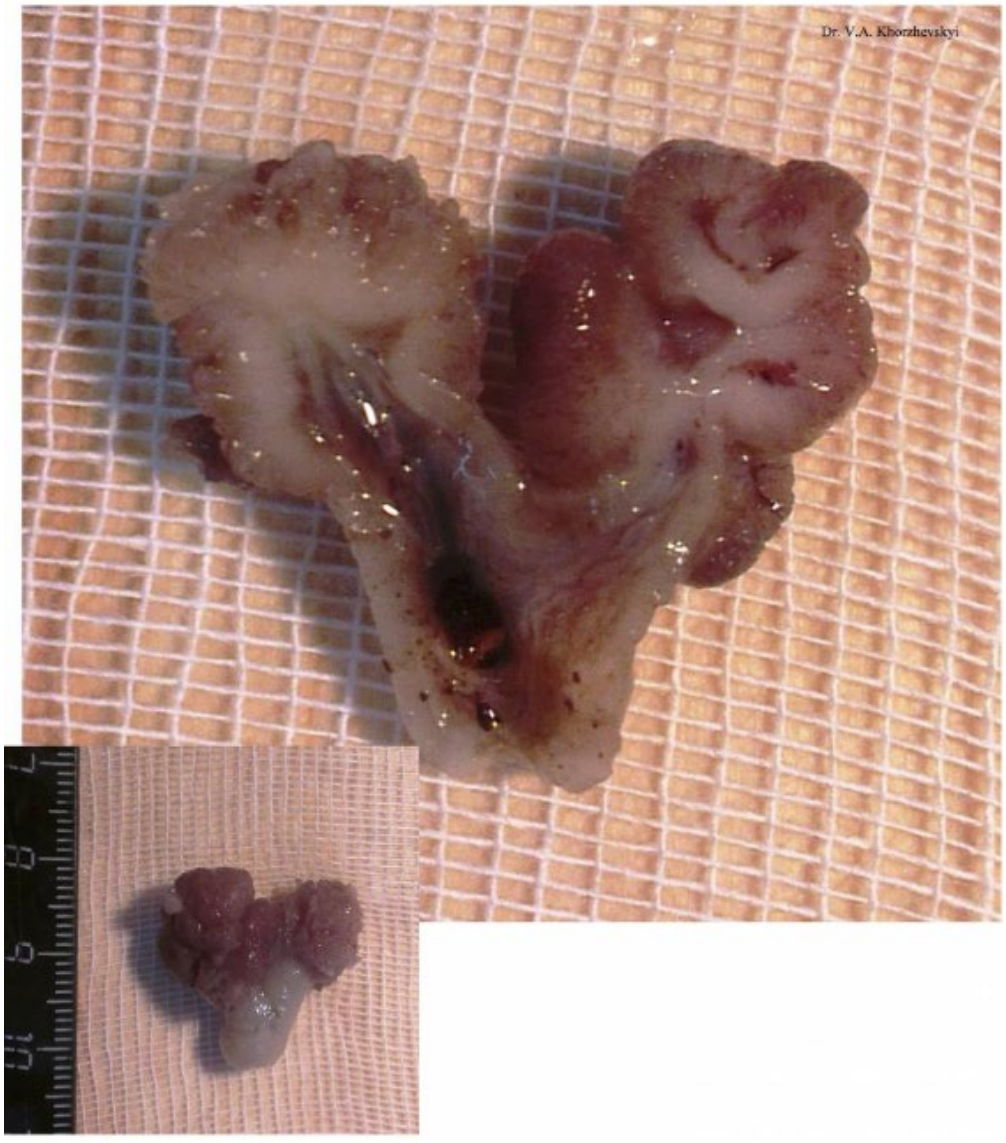
***More* varied** and complex **nomenclature** is applied **to benign epithelial tumors**.

The term ***adenoma*** *is generally applied* not only to benign epithelial neoplasms that produce glandlike structures, but also to benign epithelial neoplasms that are derived from glands but lack a glandular growth pattern.

Cystadenoma



Polyp



- is a macroscopic term that encompasses all tumors and tumor-like formations that rise above the surface of the mucous membrane or skin.

The prognosis of benign tumors is favorable

- compression of the surrounding structures (vessels, nerves)
- bleeding
- Relapses
- malignancy

Malignant Tumors

Malignant neoplasms arising in “solid” mesenchymal tissues or its derivatives

= **sarcomas**

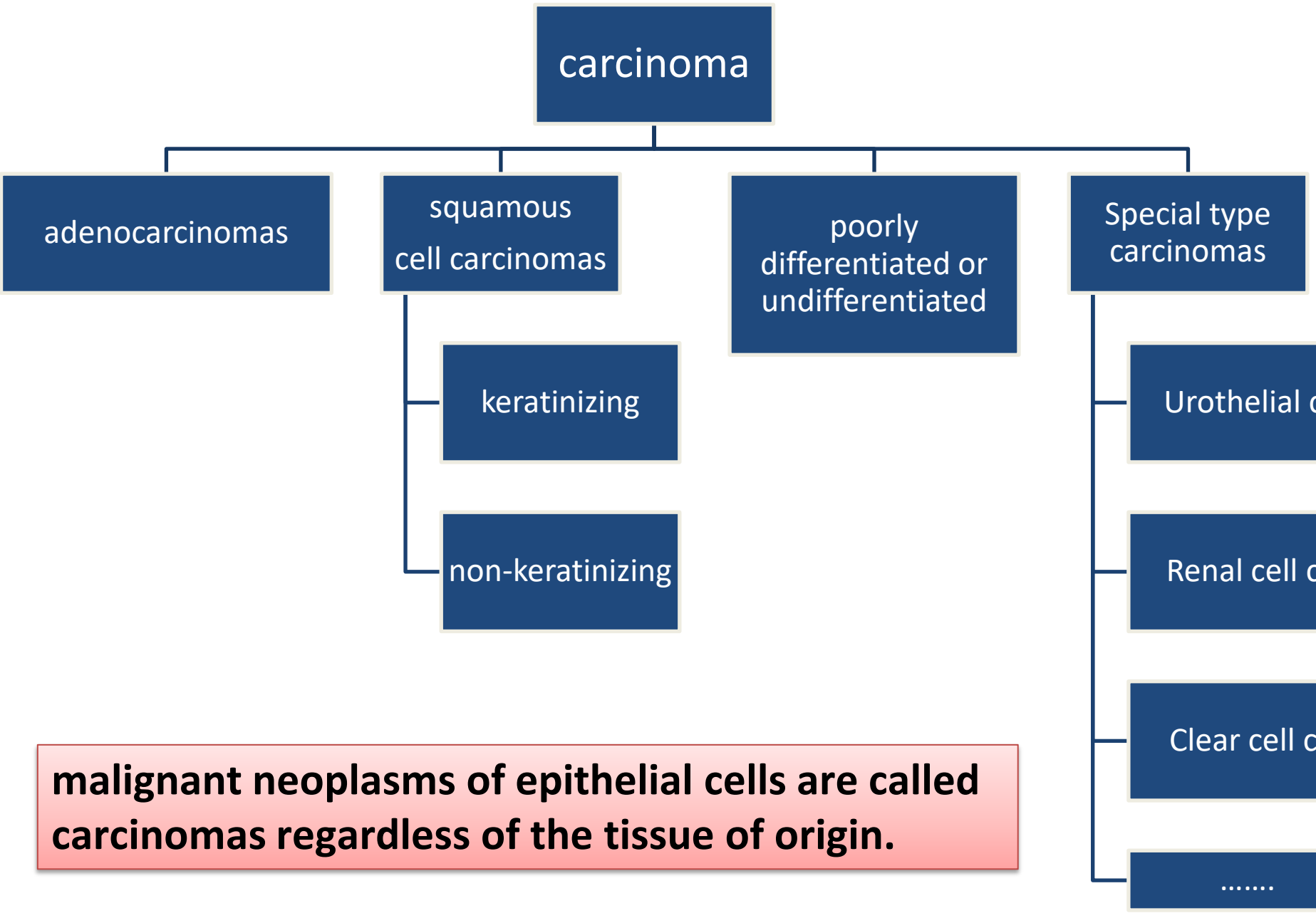
- chondrocyte = *chondrosarcoma*
- Fat cells = *liposarcoma*
- Vessels = angio*sarcoma*
- bone= osteo*sarcoma*

arising from the mesenchymal cells of the blood

= **leukemias or lymphomas**

Many malignant tumors from mesenchyme derivatives **have their own names**, for example: "glioblastoma" - malignant tumor from glial cells, "malignant tumor from peripheral nerves"

Malignant epithelial neoplasms



malignant neoplasms of epithelial cells are called carcinomas regardless of the tissue of origin.

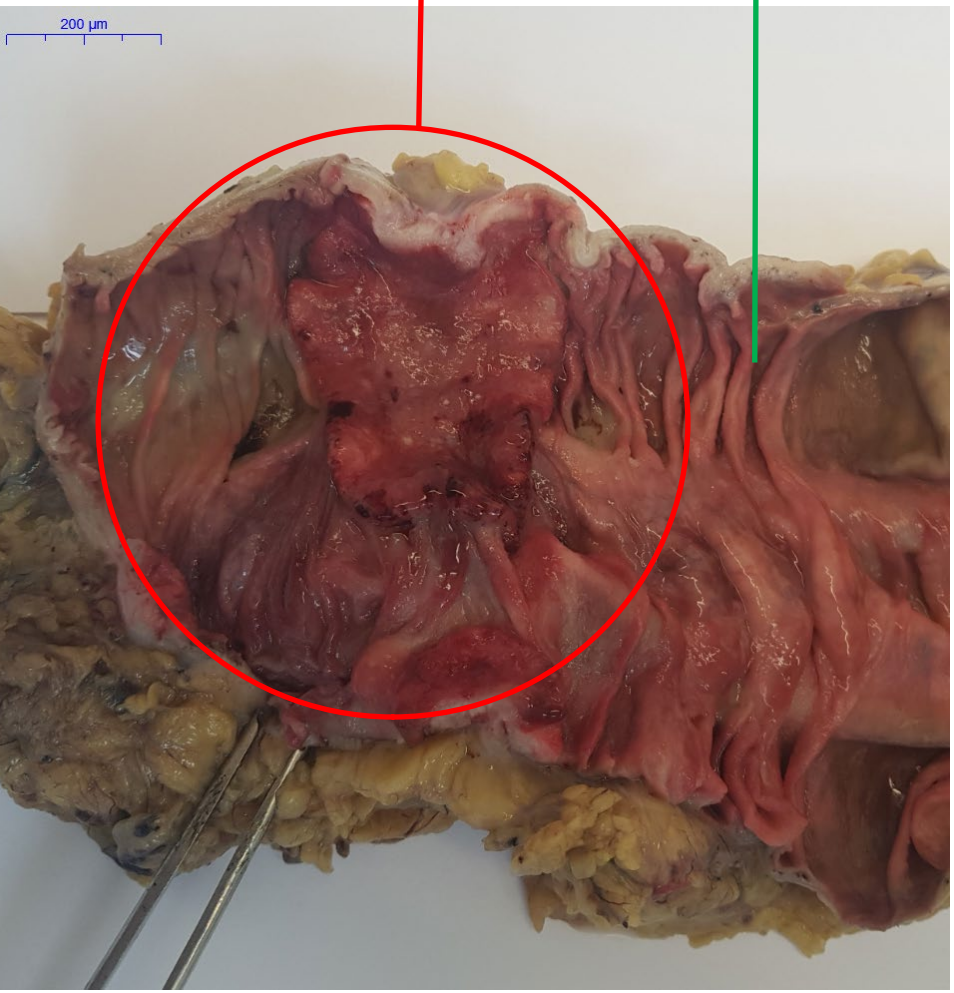
ICD-O" - International Code of Disease - Oncology

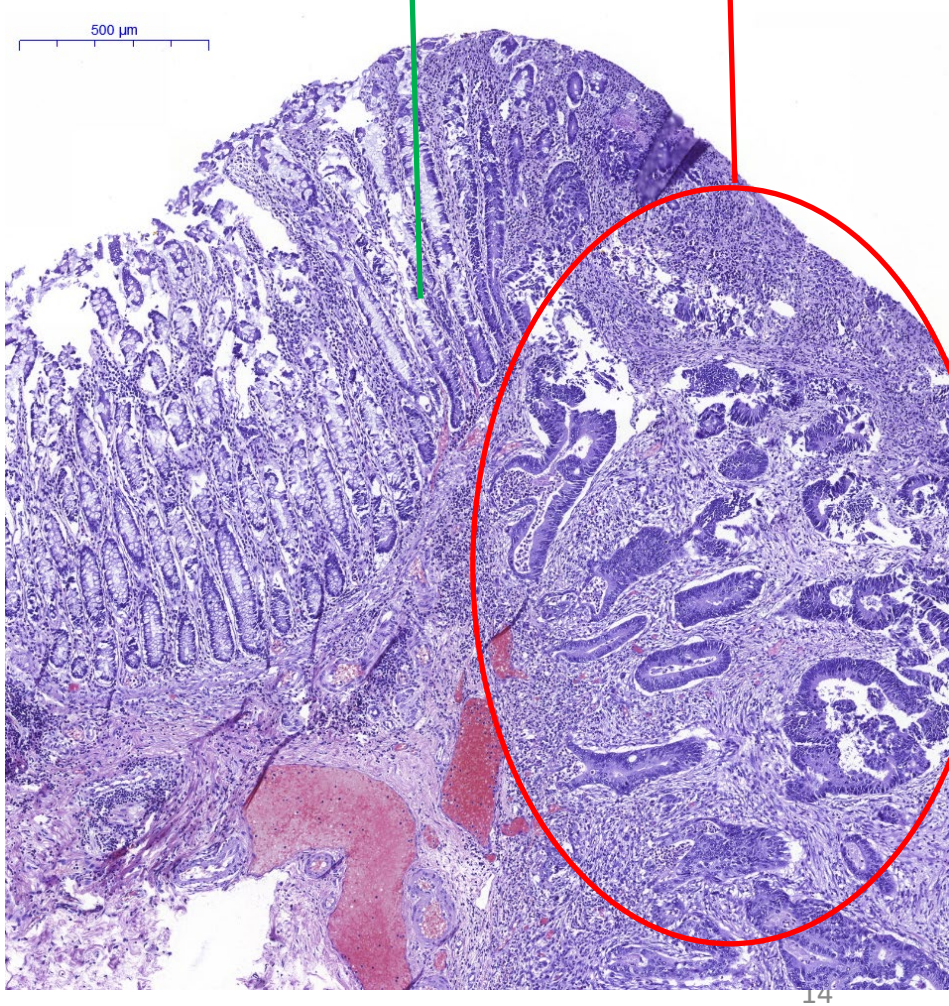
WHO classification of tumours of soft tissue[a,b]

ADIPOCYTIC TUMOURS	
Benign	
Lipoma	8850/0
Lipomatosis	
Lipomatosis of nerve	
Lipoblastoma	8881/0
Angiolipoma	8861/0
Myolipoma	8890/0
Chondroid lipoma	8862/0
Spindle cell/pleomorphic lipoma	8857/0
Hibernoma	8880/0
Intermediate (locally aggressive)	
Atypical lipomatous tumour	8850/1
Malignant	
Liposarcoma, not otherwise specified	8850/3
Dedifferentiated liposarcoma	8858/3
Myxoid liposarcoma	8852/3
Pleomorphic liposarcoma	8854/3
FIBROBLASTIC / MYOFIBROBLASTIC TUMOURS	
Benign	
Nodular fasciitis	8828/0*
Proliferative fasciitis	8828/0*
Proliferative myositis	8828/0*
Myositis ossificans	
Fibro-osseous pseudotumour of digits	
Ischaemic fasciitis	
Elastofibroma	8820/0
Fibrous hamartoma of infancy	
Fibromatosis colli	
Juvenile hyaline fibromatosis	
Inclusion body fibromatosis	
Fibroma of tendon sheath	8813/0
Desmoplastic fibroblastoma	8810/0
Inflammatory myofibroblastic tumour	8825/1
Low-grade myofibroblastic sarcoma	8825/3*
Myxoinflammatory fibroblastic sarcoma/ Atypical myxoinflammatory fibroblastic tumour	8811/1*
Infantile fibrosarcoma	8814/3
Malignant	
Adult fibrosarcoma	8810/3
Myxofibrosarcoma	8811/3
Low-grade fibromyxoid sarcoma	8840/3*
Sclerosing epithelioid fibrosarcoma	8840/3*
SO-CALLED FIBROHISTIOCYTIC TUMOURS	
Benign	
Tenosynovial giant cell tumour	
Localized type	9252/0
Diffuse type	9252/1*
Deep benign fibrous histiocytoma	8831/0
Intermediate (rarely metastasizing)	
Plexiform fibrohistiocytic tumour	8835/1
Giant cell tumour of soft tissues	9251/1
Malignant	
Malignant tenosynovial giant cell tumour	9252/3
SMOOTH MUSCLE TUMOURS	
Benign	
Deep leiomyoma	8890/0
Malignant	
Leiomyosarcoma (excluding skin)	8890/3
PERICYTIC (PERIVASCULAR) TUMOURS	
Glomus tumour (and variants)	8711/0
Glomangiomatosis	8711/1*
Malignant glomus tumour	8711/3

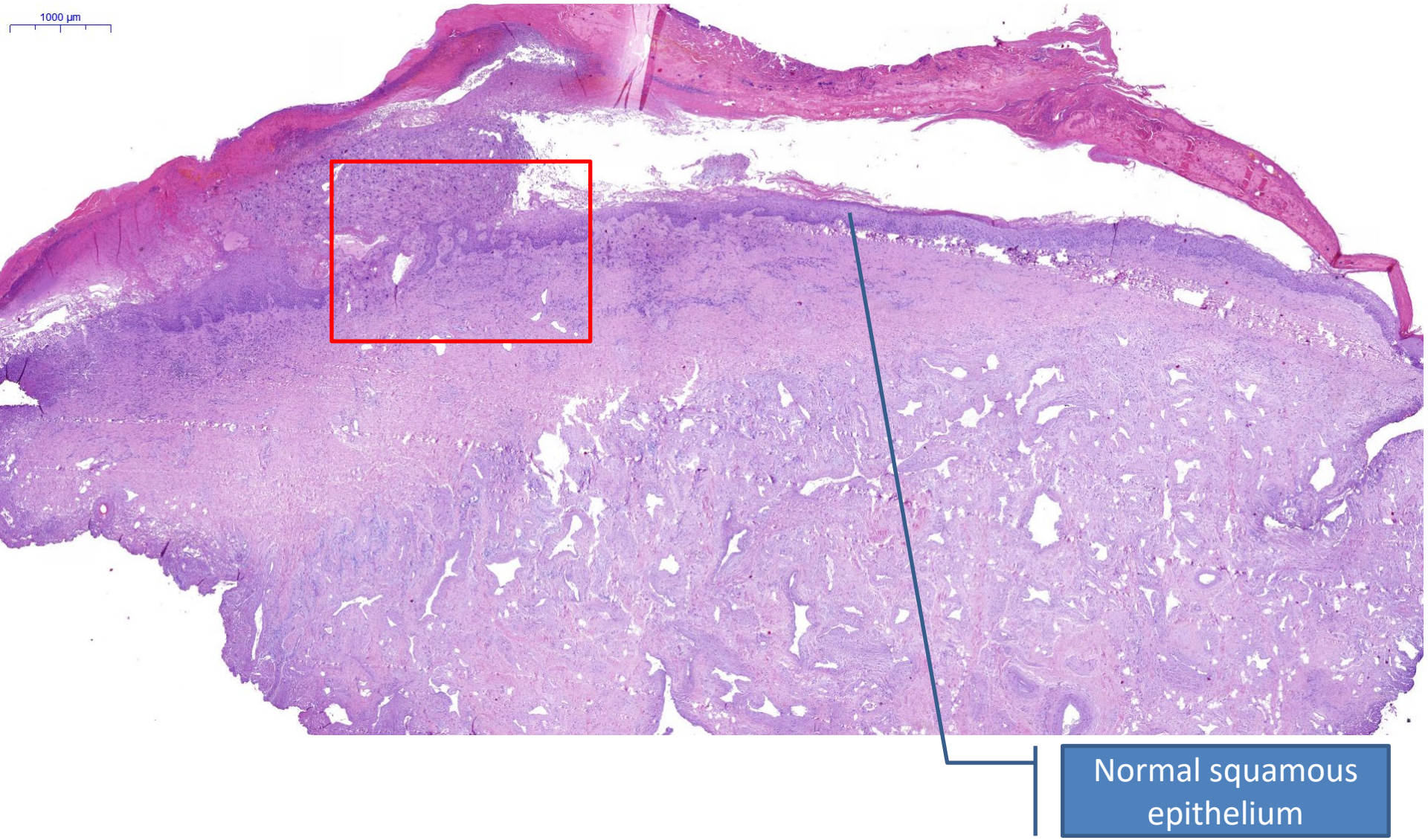
Adenocarcinomas

Normal colon

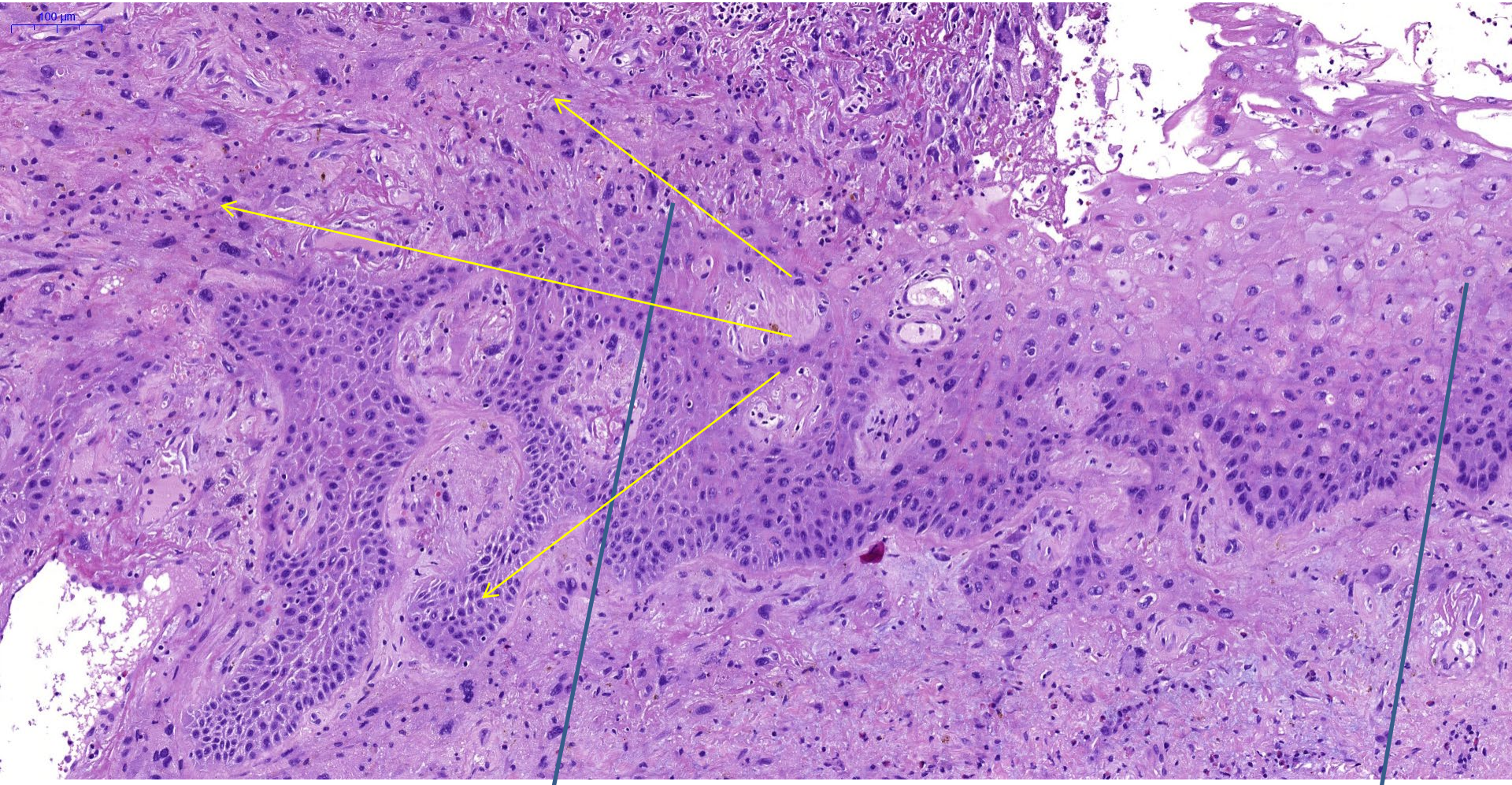




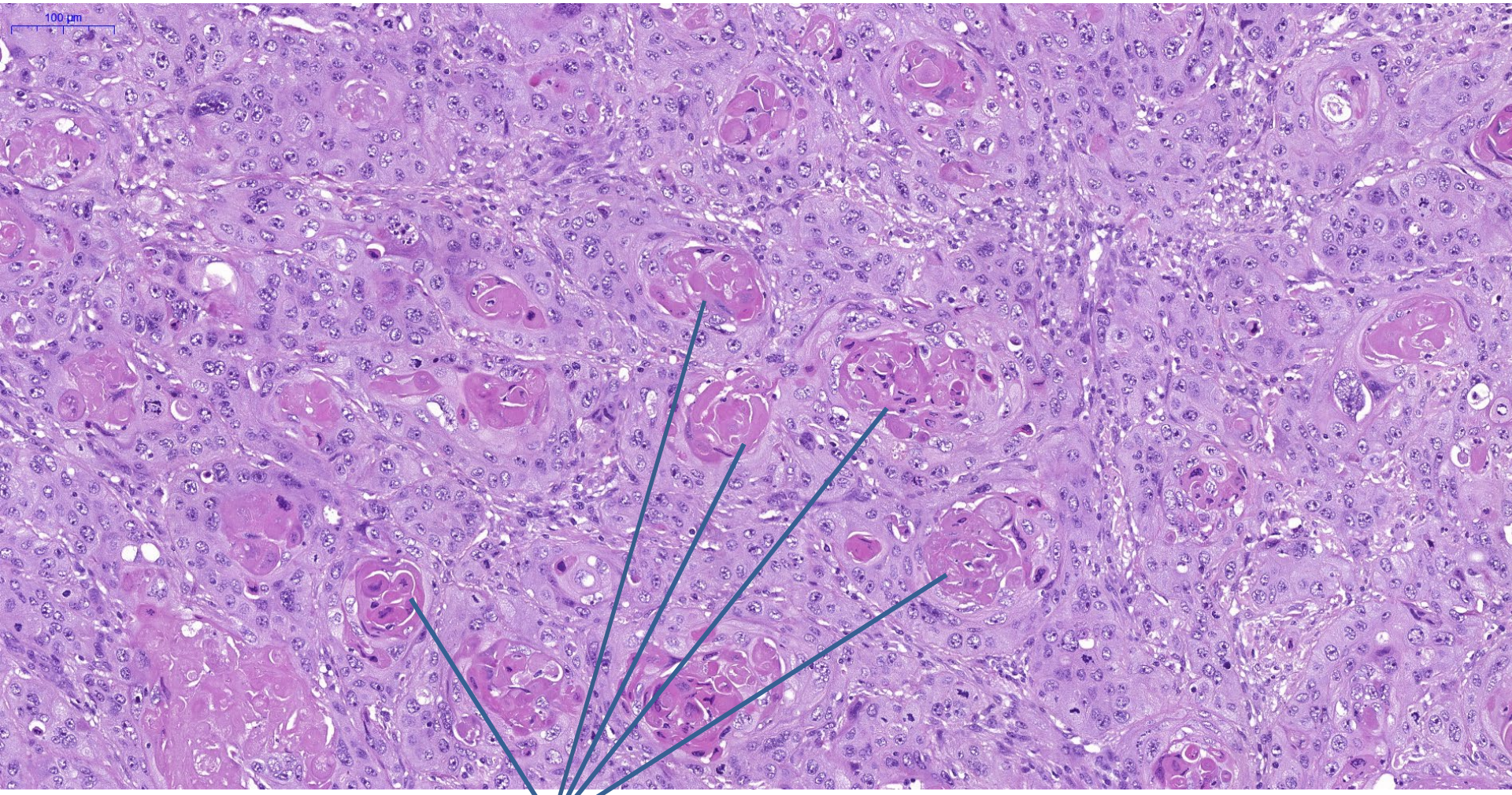
squamous cell non-keratinizing carcinoma



squamous cell non-keratinizing carcinoma



Lung
keratinizing squamous cell carcinomas



CHARACTERISTICS OF BENIGN AND MALIGNANT NEOPLASMS

Anaplasia

= lack of differentiation

Anaplasia
-malignant process
-result of dedifferentiation of a previously more differentiated cell
-tumor formation from stem cell that did not initially have signs of differentiation

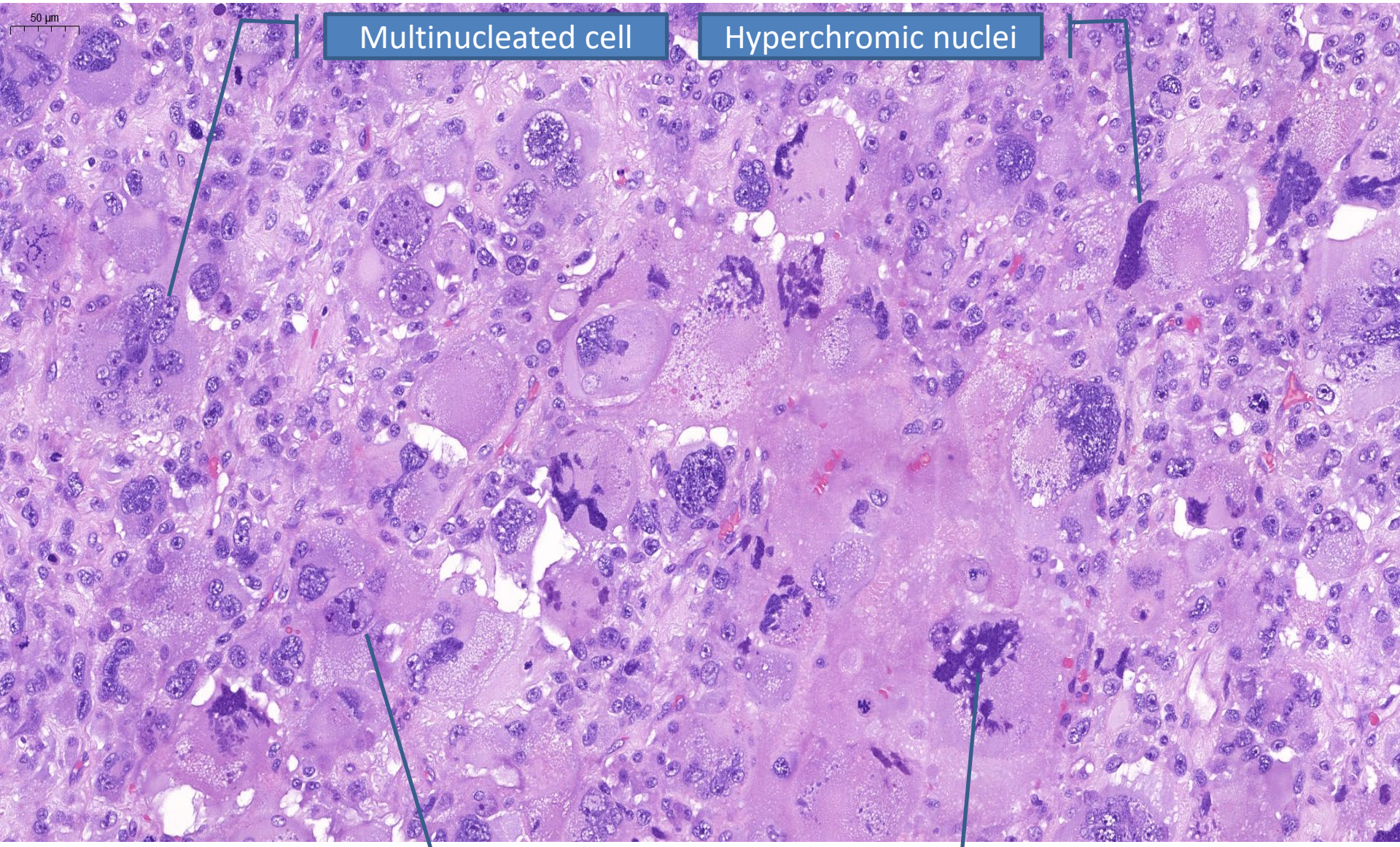
Tumors composed of undifferentiated cells are said to be *anaplastic, a feature that is a reliable indicator of* malignancy.

Anaplastic cells display the following morphologic features:

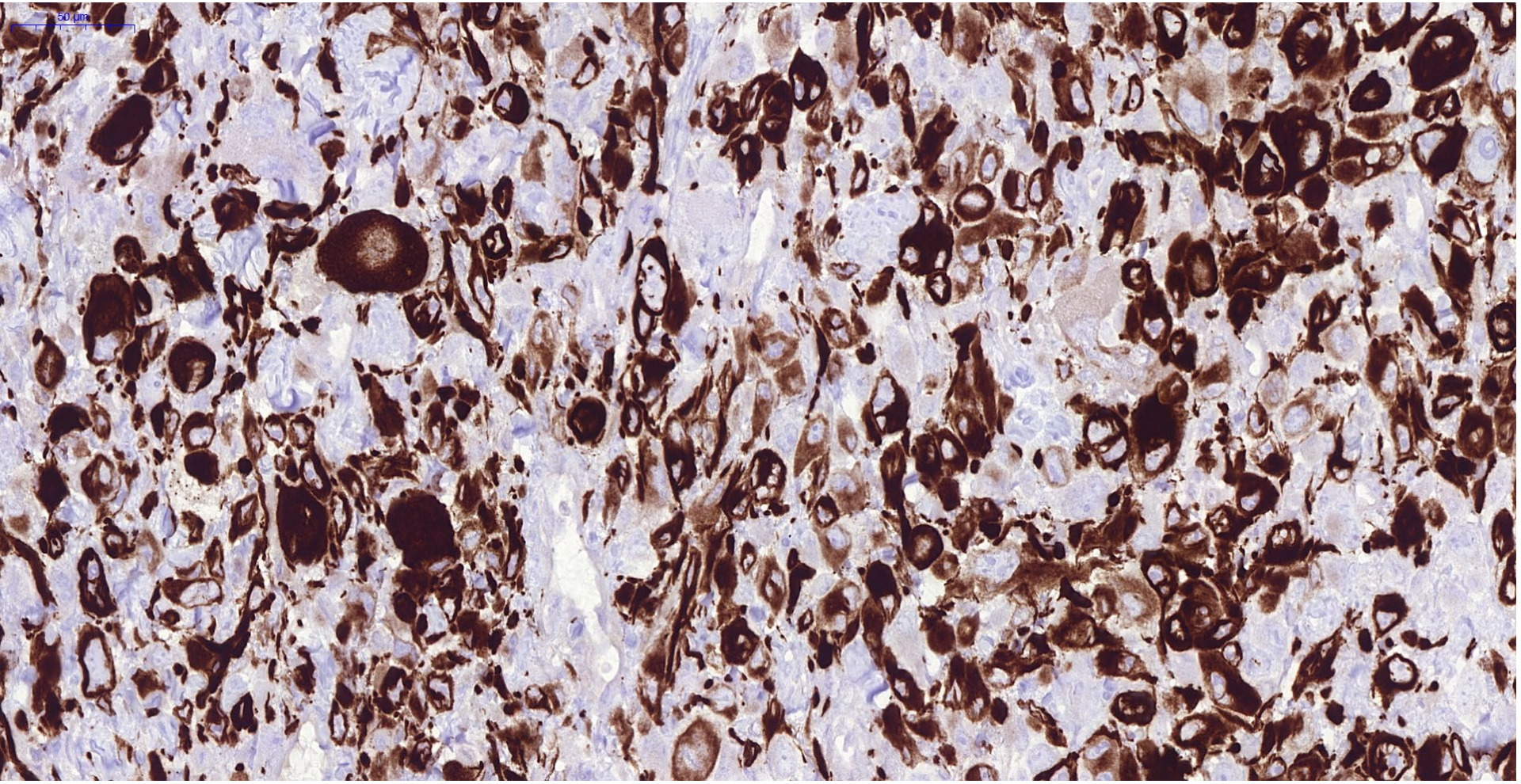
features
Pleomorphism (i.e., variation in size and shape)
Nuclear abnormalities (hyperchromatism, variation in nuclear size and shape, or unusually prominent single or multiple nucleoli)
Tumor giant cells may be formed
Atypical mitoses, which may be numerous
Loss of polarity

Well-differentiated tumor cells are likely to retain the functional capabilities of their normal counterparts, whereas **anaplastic tumor cells are much less likely to have specialized functional activities.**

Anaplastic rhabdomyosarcoma.

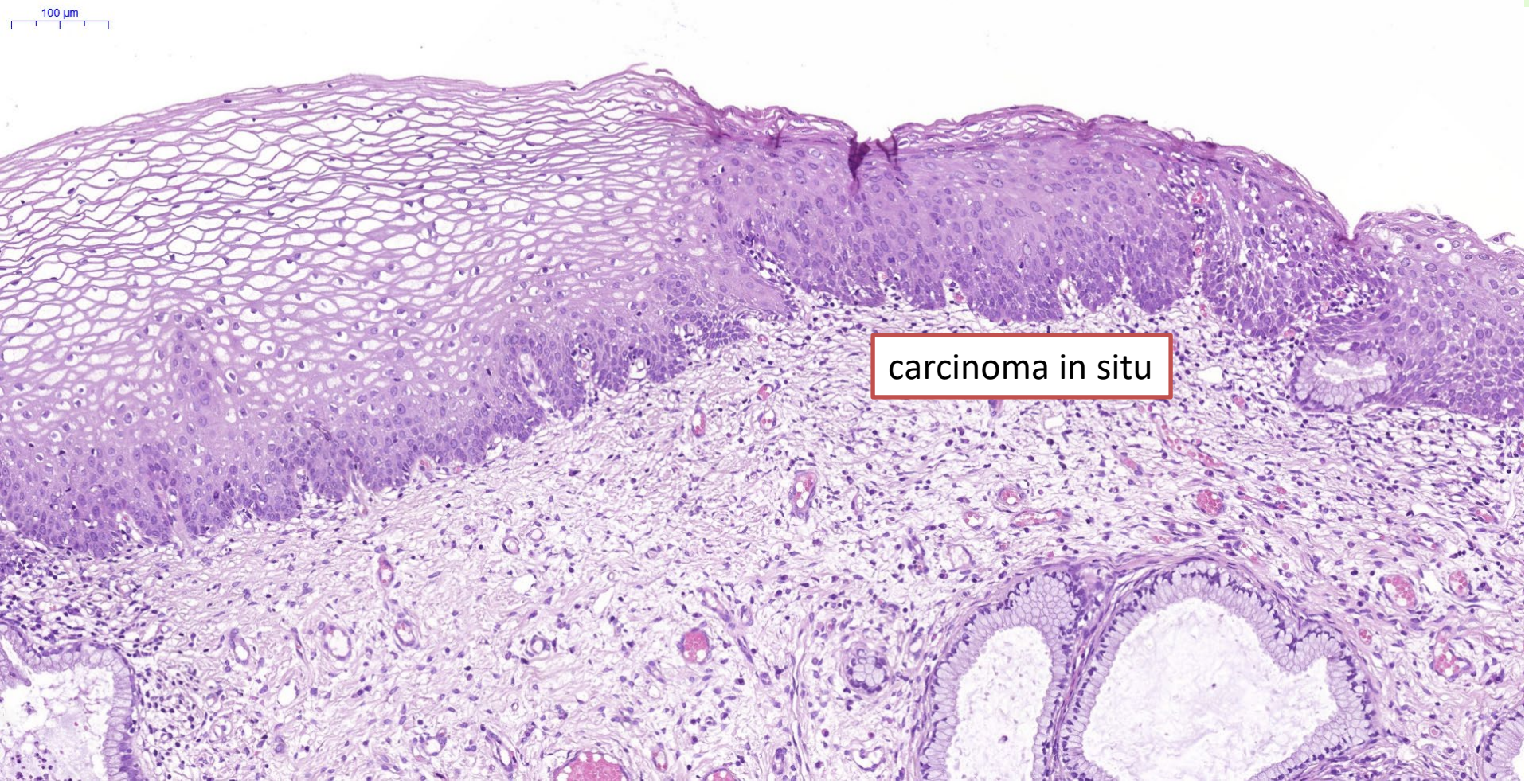


Desmin expression

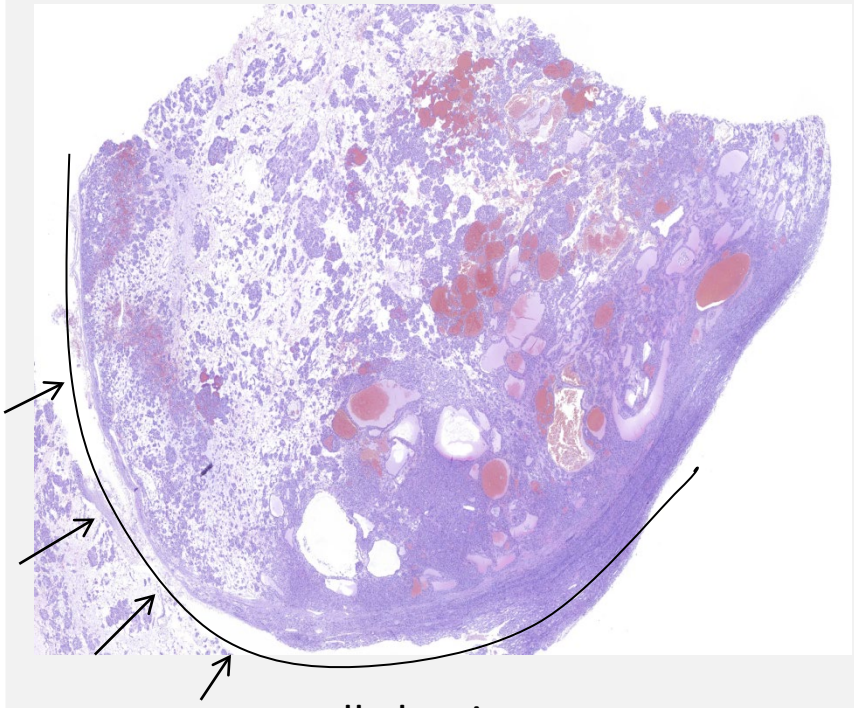
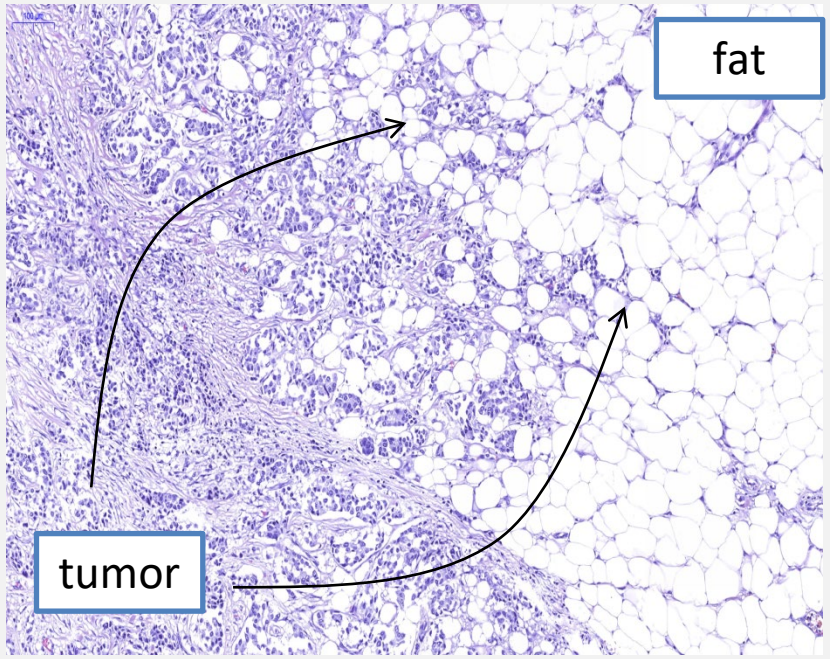


Dysplasia

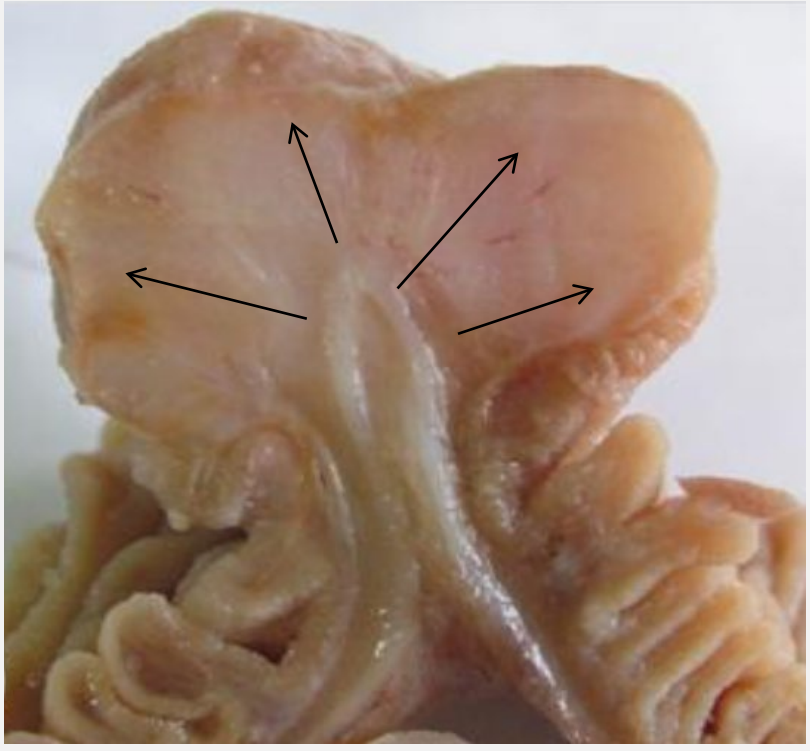
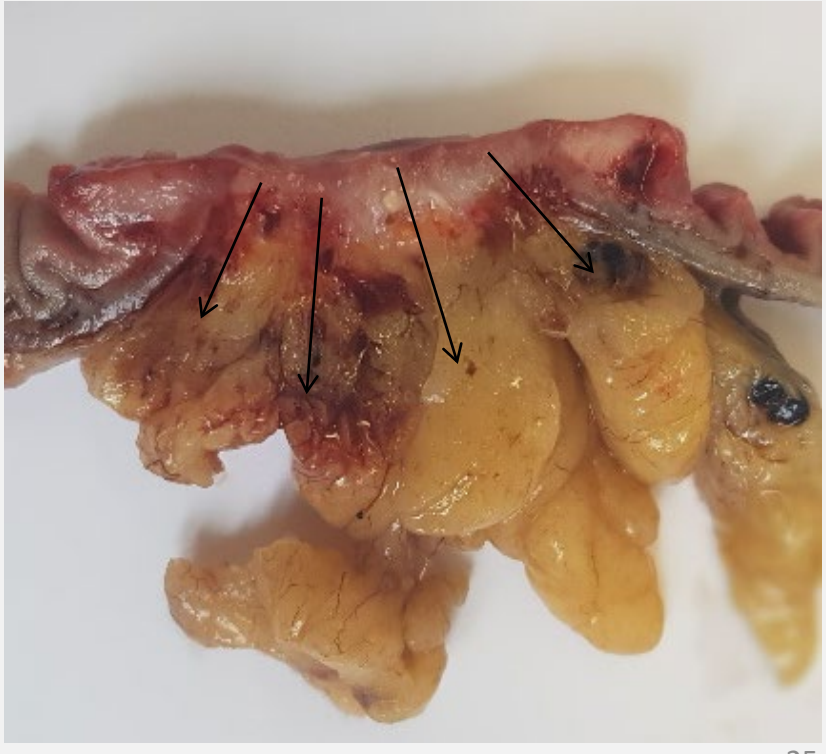
Dysplastic epithelium is recognized by a loss in the uniformity of individual cells and in their architectural orientation. Dysplastic cells exhibit considerable pleomorphism and often possess abnormally large, hyperchromatic nuclei. Mitotic figures are more abundant than usual and frequently appear in abnormal locations within the epithelium.



Types of growth and growth rate of tumors

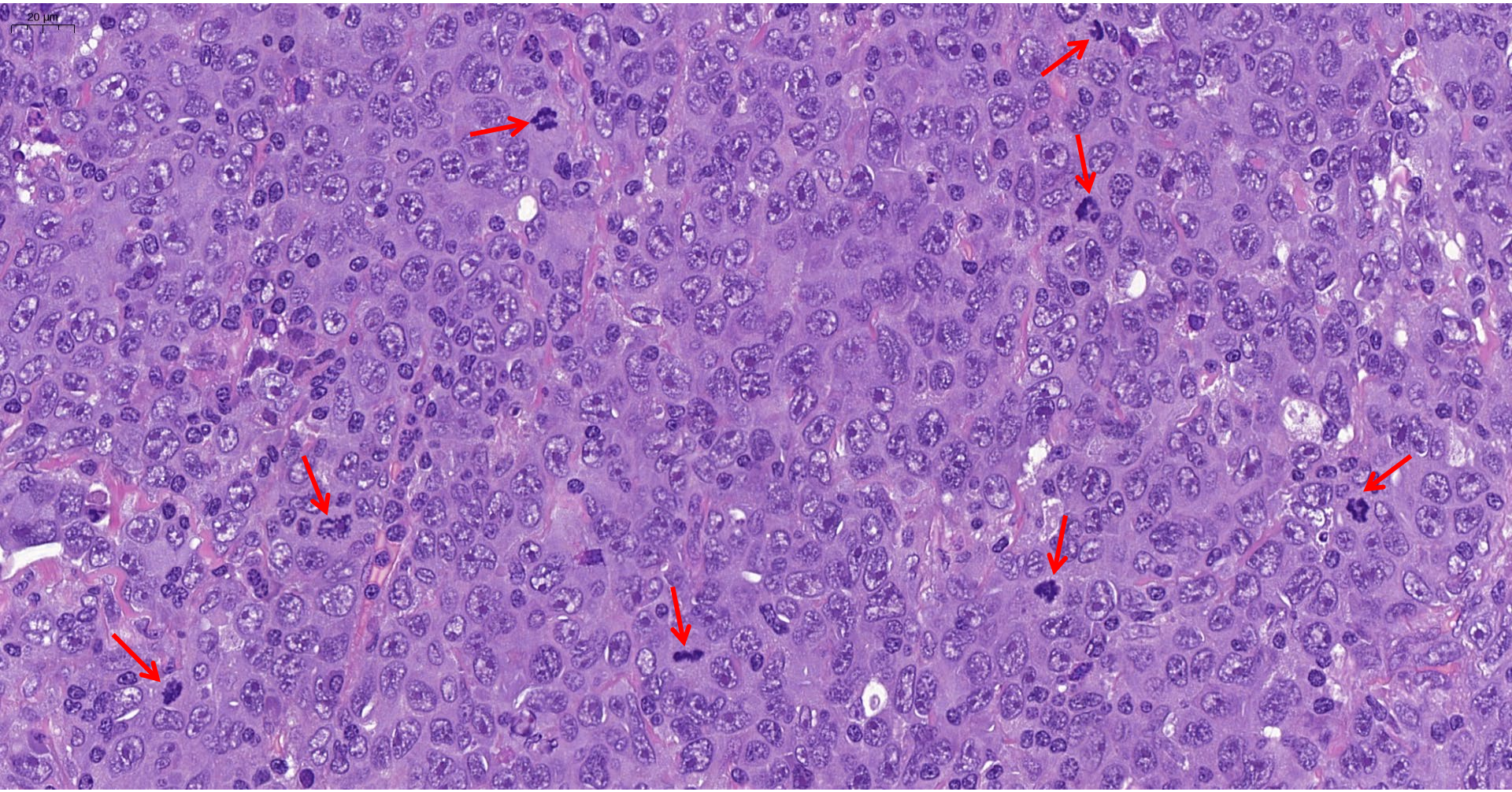
In relation to surrounding tissues	
Expansive	Infiltrating
Expansive growth involves the proliferation of tumor elements with the formation of a tumor node. Fibrous pseudocapsule is often formed.	Tumor growth in which proliferating tumor cells spread into the surrounding tissue, infiltrating it.
	
usually benign	usually malignant

Types of growth and growth rate of tumors

In relation to the lumen of an organ or the external environment	
Exophytic	Endophytic
"Outside growth" The tumor mass goes into the lumen of the organ.	The tumor mass extends into the interior of the organ wall
	
usually benign	usually malignant

Growth rate

malignant tumors are more prone to rapid growth than benign tumors



The morphological expression of high proliferative activity is the number of mitotic figures per unit area.

Metastasis

Metastasis is defined by the spread of a tumor to sites that are physically discontinuous with the primary tumor

Malignant neoplasms disseminate by one of three pathways:
(1) seeding within body cavities,
(2) lymphatic spread,
(3) hematogenous spread.

Spread by seeding

- occurs when neoplasms invade a natural body cavity. This mode of dissemination is particularly characteristic of cancers of the ovary and neoplasms of the central nervous system.

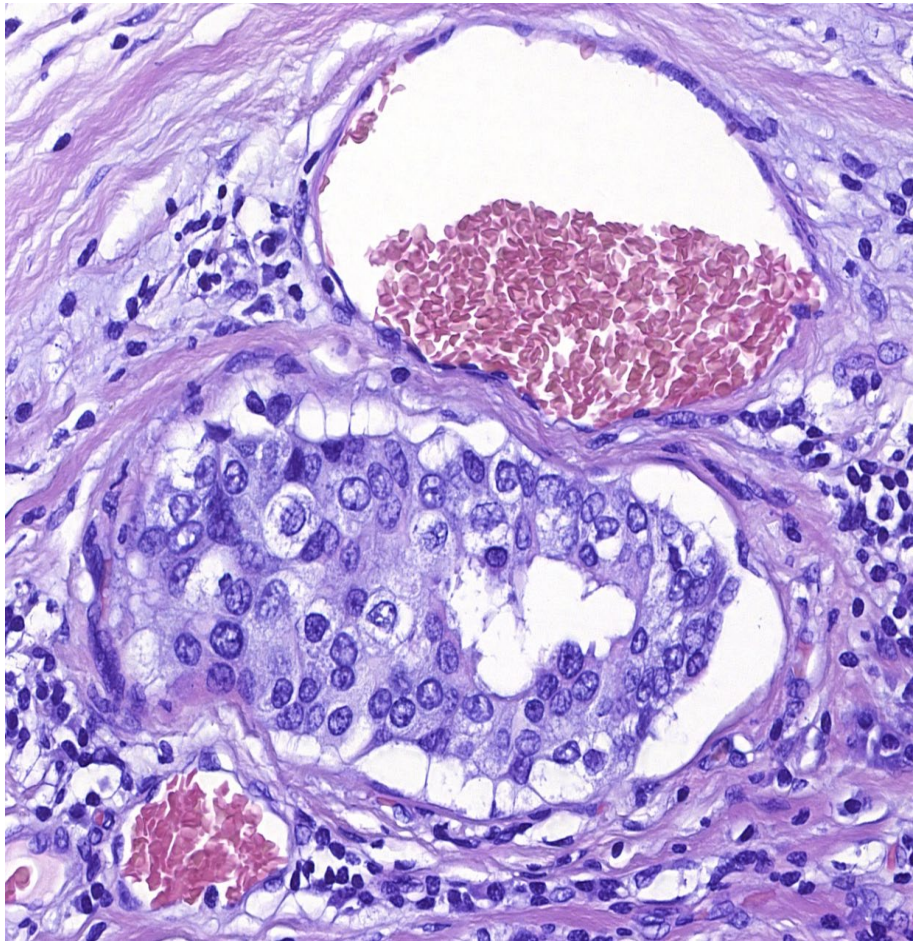
The lymphatic spread

-is typical for carcinomas. Tumor cells spread through the lymphatic vessels, causing primary damage to the regional lymph nodes.
-Breast carcinomas often lymphogenically metastasize.

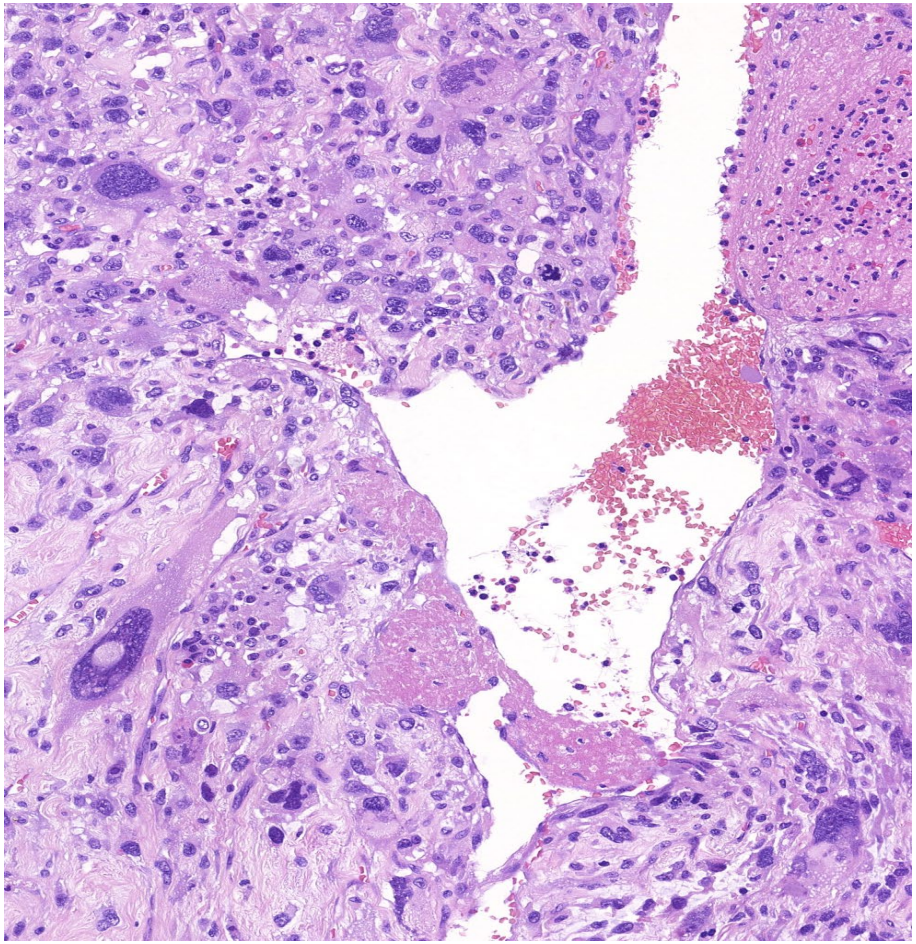
Hematogenous spread

-metastasis through the blood vessels. Typical way of sarcoma metastasis. Metastasis occurs when the tumor invades the veins with the detachment of tumor complexes.

The lymphatic spread



Hematogenous spread

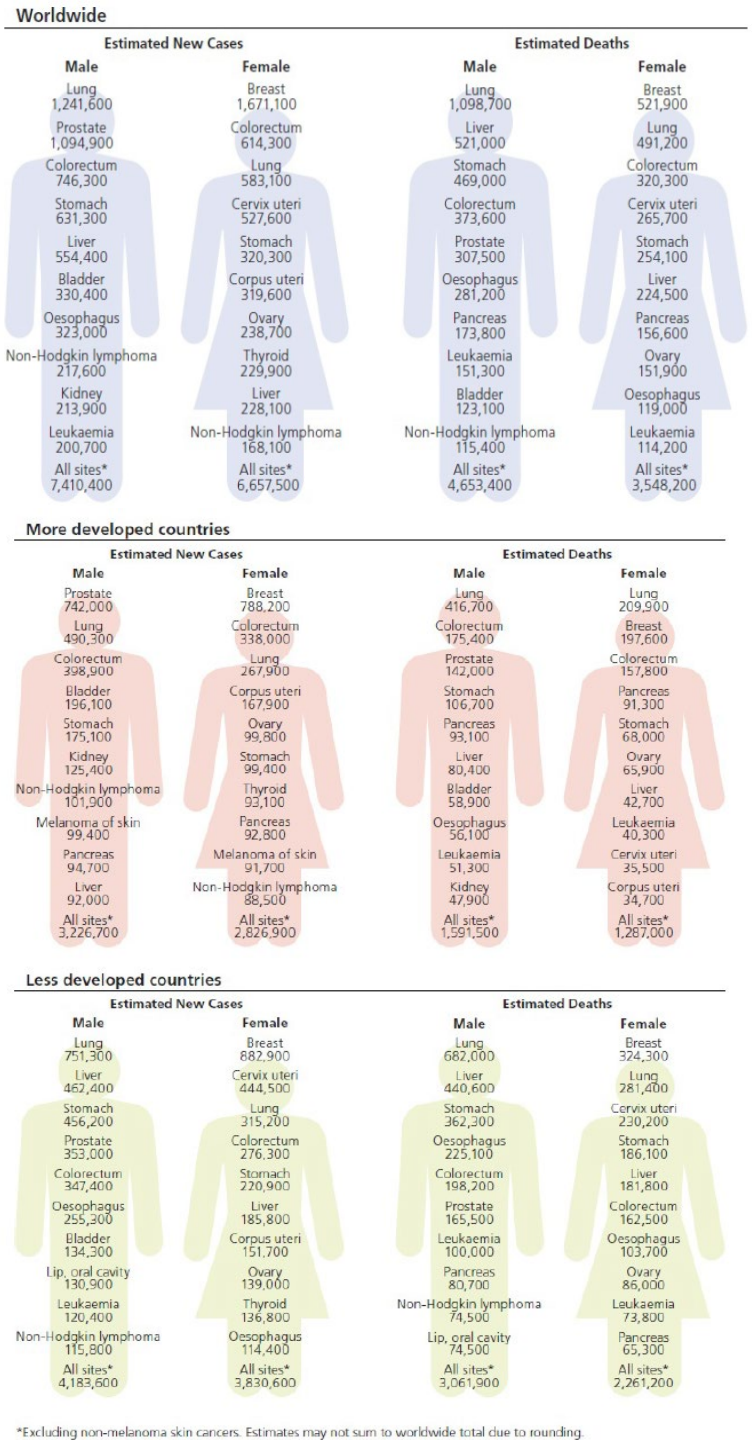


Resume

CHARACTERISTICS OF BENIGN AND MALIGNANT TUMORS

- Benign and malignant tumors can be distinguished from one another based on the degree of differentiation, rate of growth, local invasiveness, and distant spread.
- Benign tumors resemble the tissue of origin and are well differentiated; malignant tumors are poorly or completely undifferentiated (anaplastic).
- Benign tumors tend to be slow growing, whereas malignant tumors generally grow faster.
- Benign tumors are well circumscribed and have a capsule; malignant tumors are poorly circumscribed and invade the surrounding normal tissues.
- Benign tumors remain localized to the site of origin, whereas malignant tumors are locally invasive and metastasize to distant sites.

EPIDEMIOLOGY



*Excluding non-melanoma skin cancers. Estimates may not sum to worldwide total due to rounding.

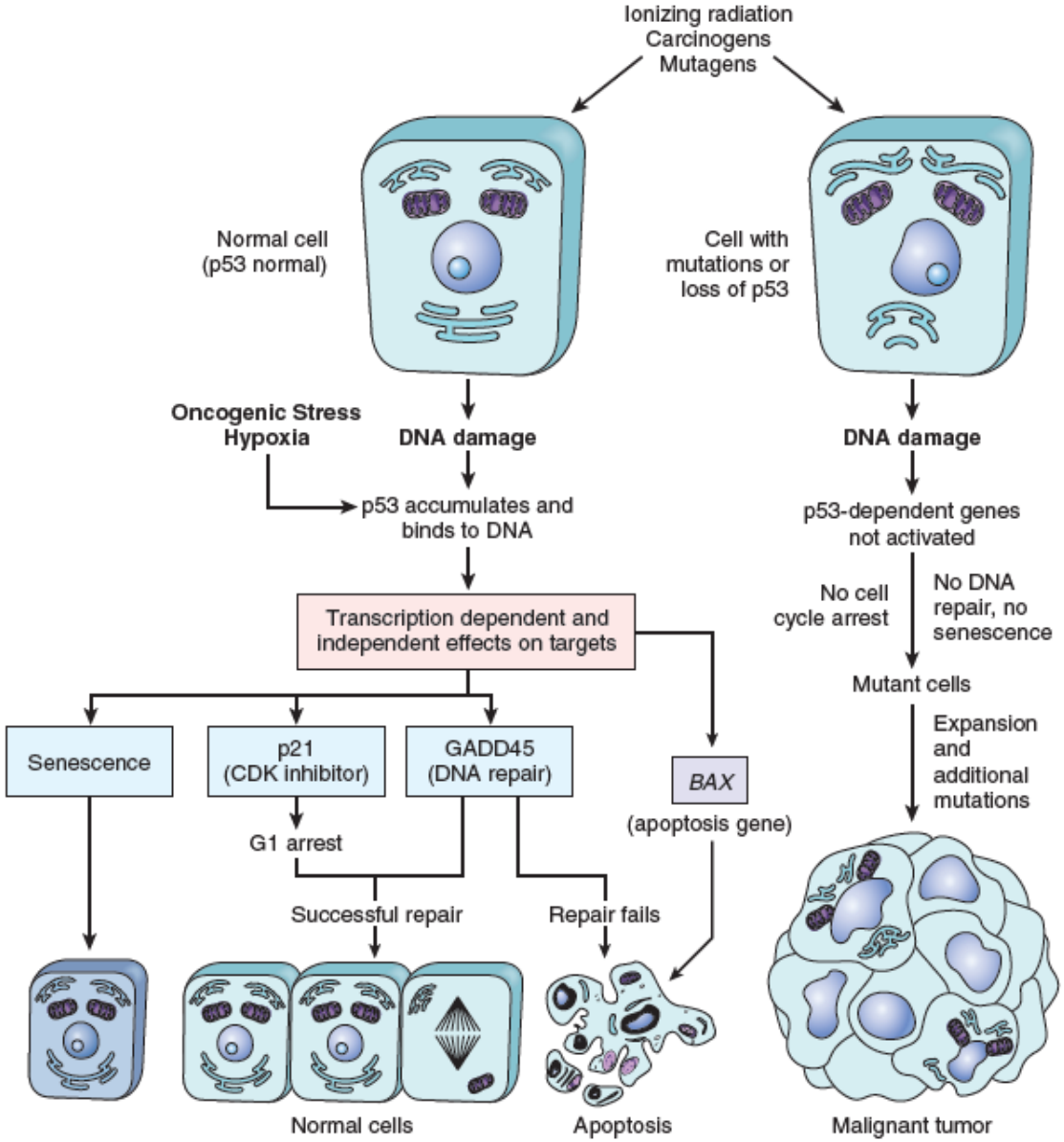
Из: DeVita, Hellman, and Rosenberg's cancer : principles & practice of oncology / [edited by] Vincent T. DeVita, Jr., Theodore S. Lawrence, Steven A. Rosenberg. Description: 11th edition. | Philadelphia : Wolters Kluwer, [2019] | Includes bibliographical references.

GENETIC LESIONS IN CANCER

The development of tumors is a genetically determined process associated with damage to genetic material, which does not lead to the appearance of lethal mutations.
There are 4 main groups of genes that become targets for the damaging effect:

1. Proto-oncogenes are growth promoters (their activation induces tumor growth);
2. Genes-suppressors of tumor growth (their inactivity leads to tumor growth);
3. Genes regulating apoptosis (their inactivation leads to tumor growth);
4. Genes for DNA repair (their inactivation leads to tumor growth).

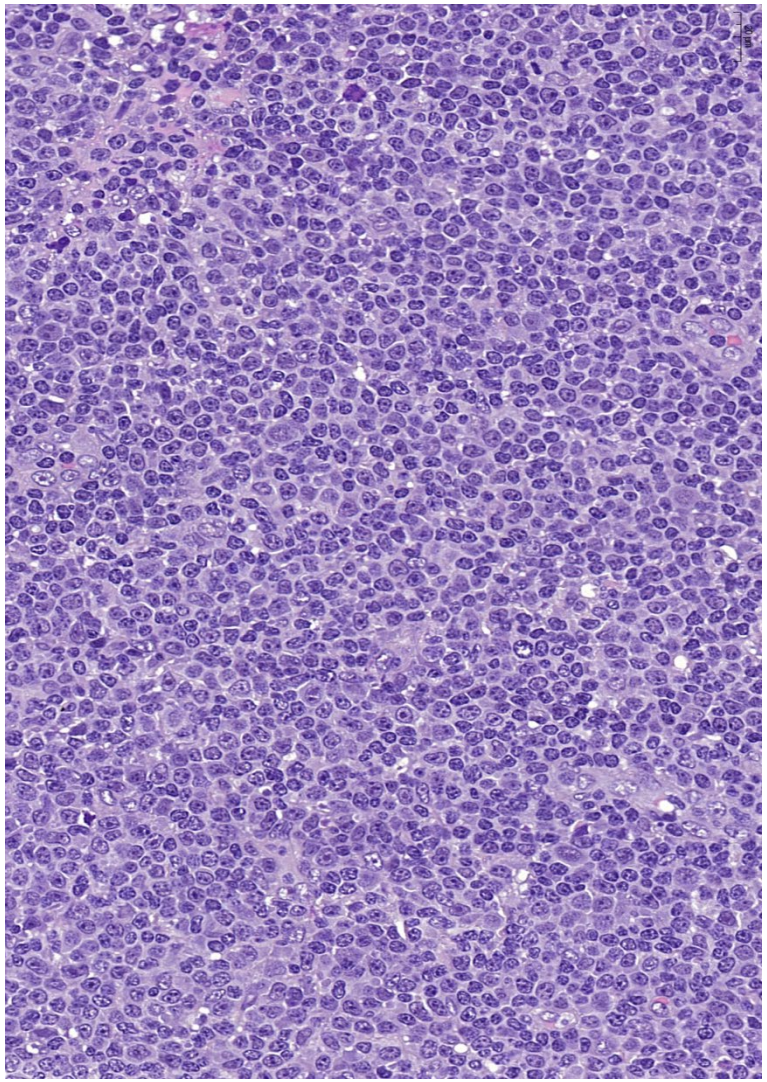
p53-encoding tumor suppressor gene

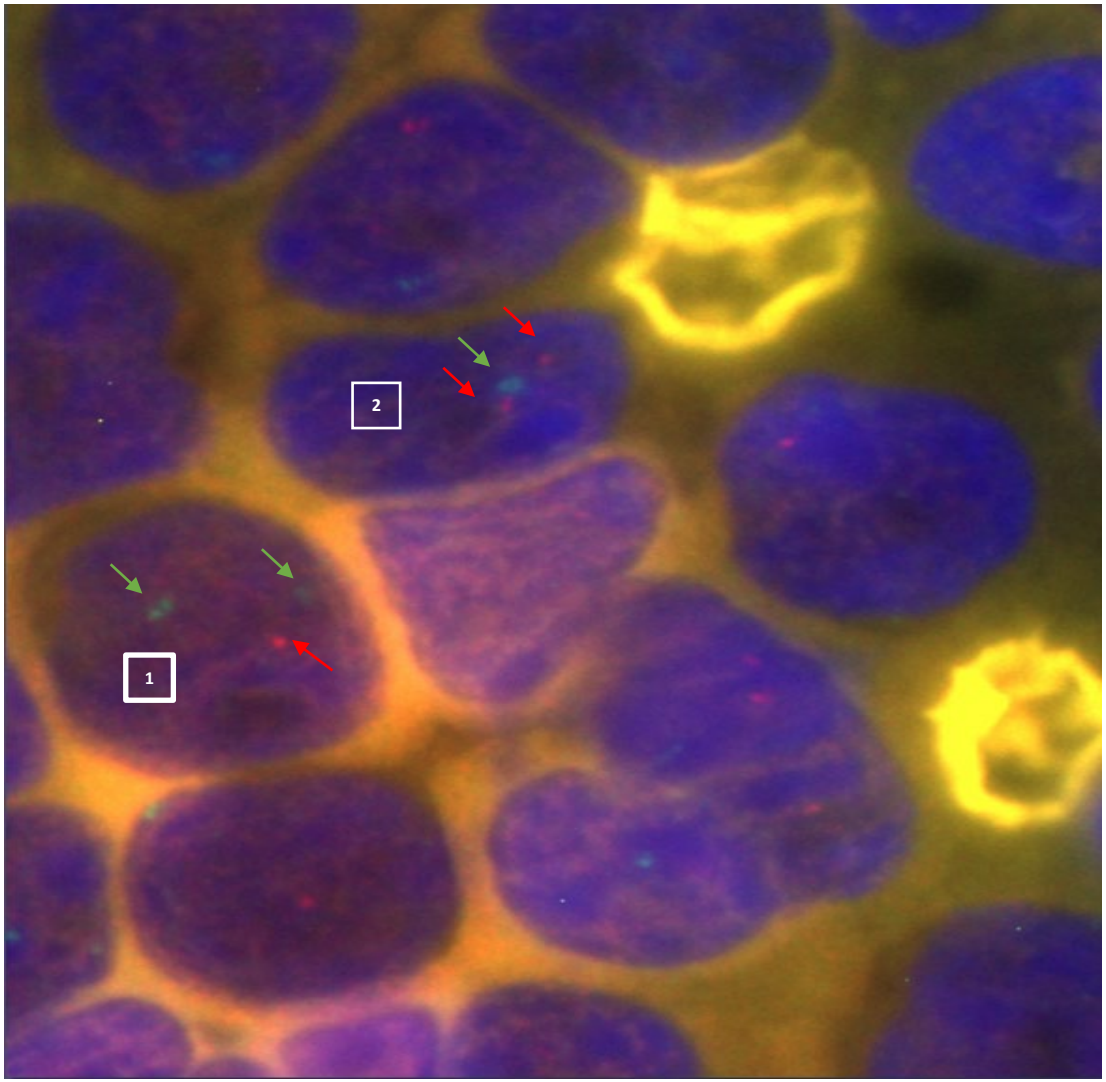


The role of p53 in maintaining the integrity of the genome. Activation of normal p53 by DNA-damaging agents or by hypoxia leads to cell cycle arrest in G1 and induction of DNA repair, by transcriptional upregulation of the cyclin-dependent kinase inhibitor CDKN1A (p21) and the *GADD45 genes.* Successful repair of DNA allows cells to proceed with the cell cycle; if DNA repair fails, p53 triggers either apoptosis or senescence. In cells with loss or mutations of TP53, DNA damage does not induce cell cycle arrest or DNA repair, and genetically damaged cells proliferate, giving rise eventually to malignant neoplasms.

*From: Kumar, V., Abbas, A. K., & Aster, J. C. (2017). Robbins Basic Pathology (10th ed.). Elsevier - Health Sciences Division.

p53-encoding tumor suppressor gene in case of CLL





Mutations in the genes of the mismatch checking system

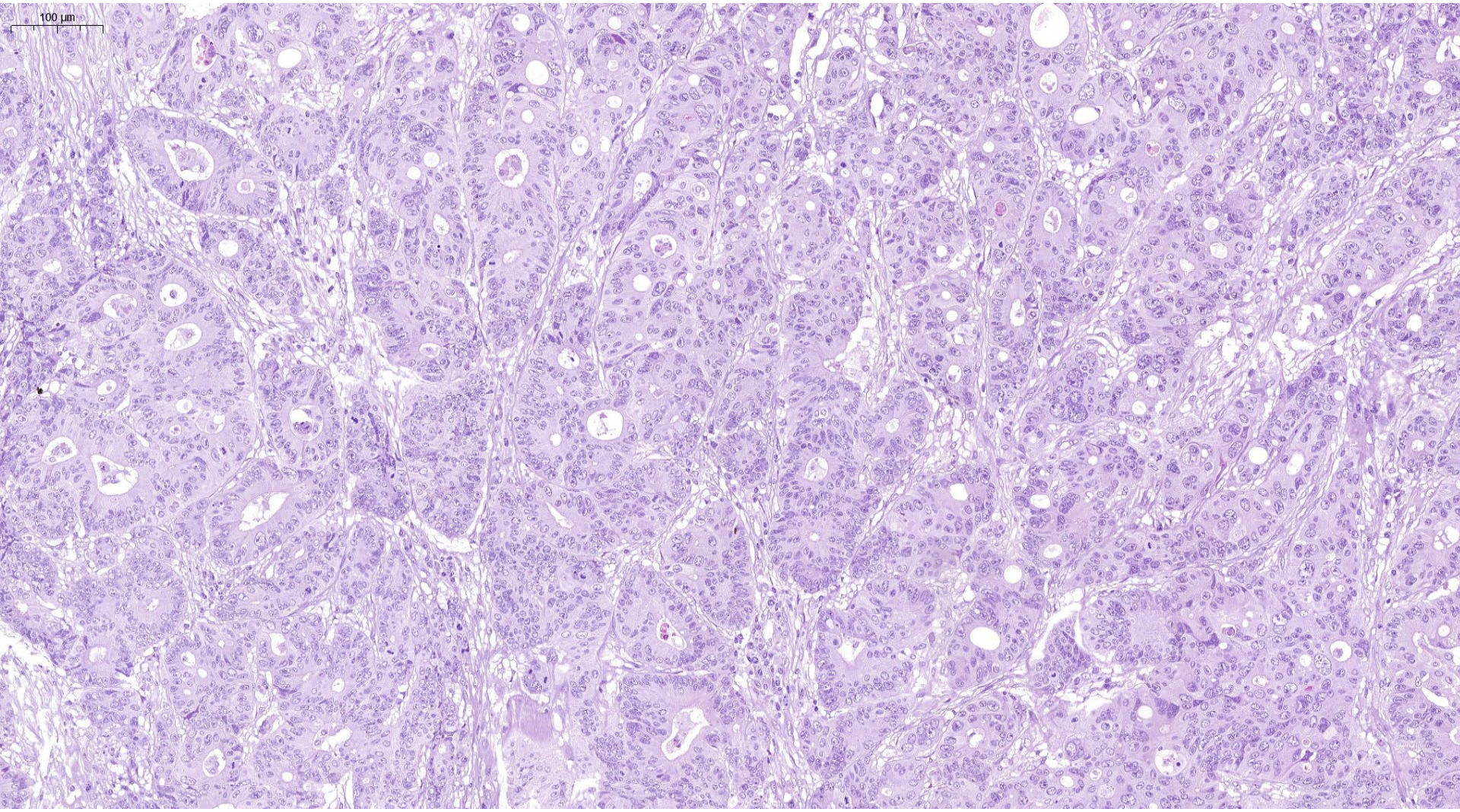
damage to simple repeating DNA sequences (microsatellites) occurs - a phenomenon called microsatellite instability (MSI).

The detection of such defective microsatellites = damage to genes responsible for correcting DNA assembly errors.

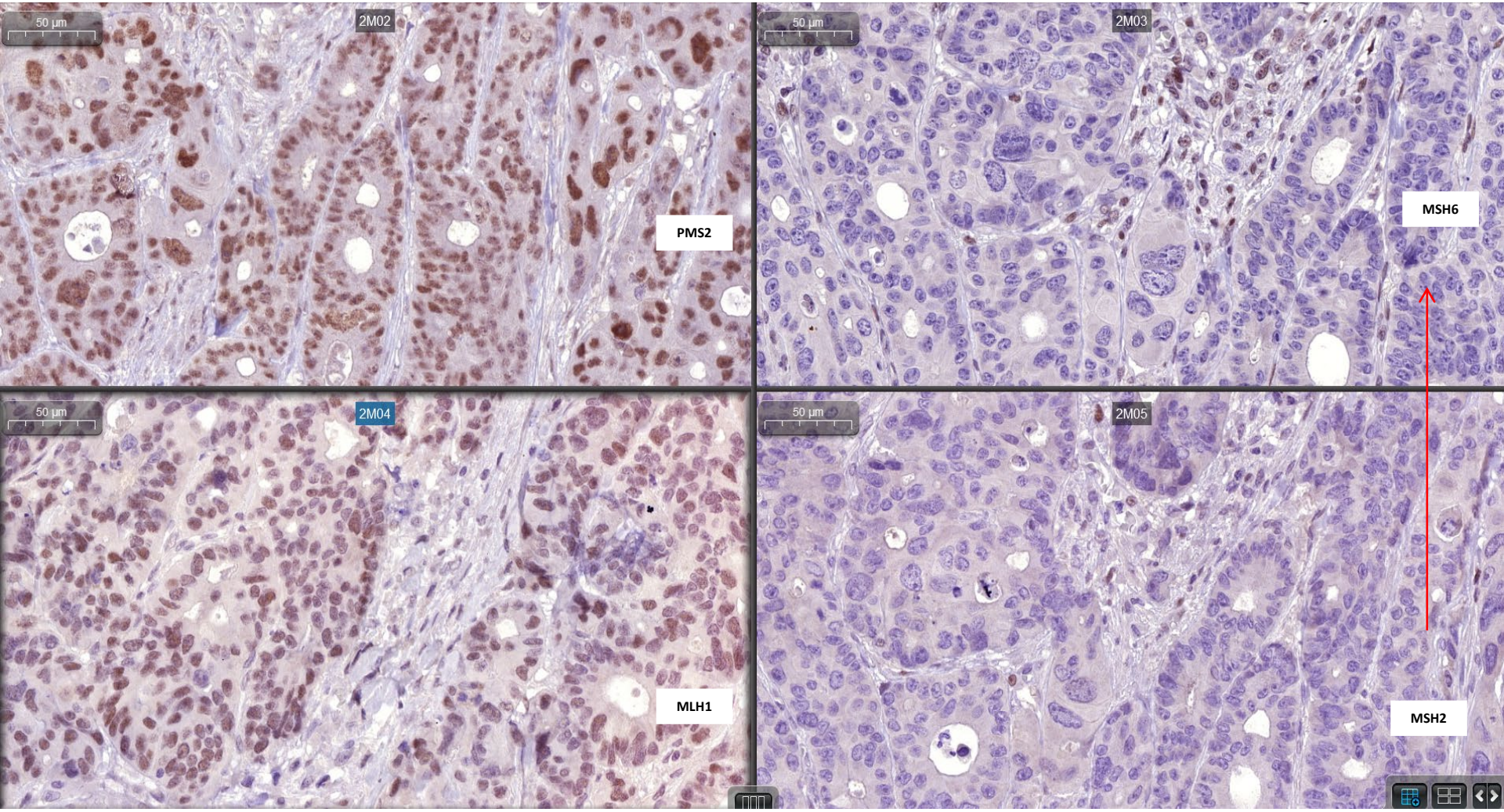
These genes include: MSH2, MSH6, MLH1, PMS2.Most of the microsatellite DNA sequences are located in the non-coding part of DNA.

Their damage leads to disruption of the cell cycle with constant cell proliferation and blocking of apoptosis.

hereditary non-polyposis adenocarcinoma of the colon



immunohistochemical study shows a loss of expression of MSH2 and MSH6



damage of MLH1 gene leads to damage to the PMS2 gene

damage of MSH2 gene leads to damage to the MSH6 gene

Etiology of malignant tumors: factors of carcinogenesis.

Direct-Acting Carcinogens

Alkylating Agents

β-Propiolactone
Dimethyl sulfate
Diepoxybutane
Anti-cancer drugs (cyclophosphamide, chlorambucil, nitrosoureas, and others)

Acylating Agents

1-Acetyl-imidazole
Dimethylcarbamyl chloride

Procarcinogens That Require Metabolic Activation

Polycyclic and Heterocyclic Aromatic Hydrocarbons

Benz(*a*)anthracene
Benzo(*a*)pyrene
Dibenz(*a,h*)anthracene
3-Methylcholanthrene
7, 12-Dimethylbenz(*a*)anthracene

Aromatic Amines, Amides, Azo Dyes

2-Naphthylamine (β-naphthylamine)
Benzidine
2-Acetylaminofluorene
Dimethylaminoazobenzene (butter yellow)

Natural Plant and Microbial Products

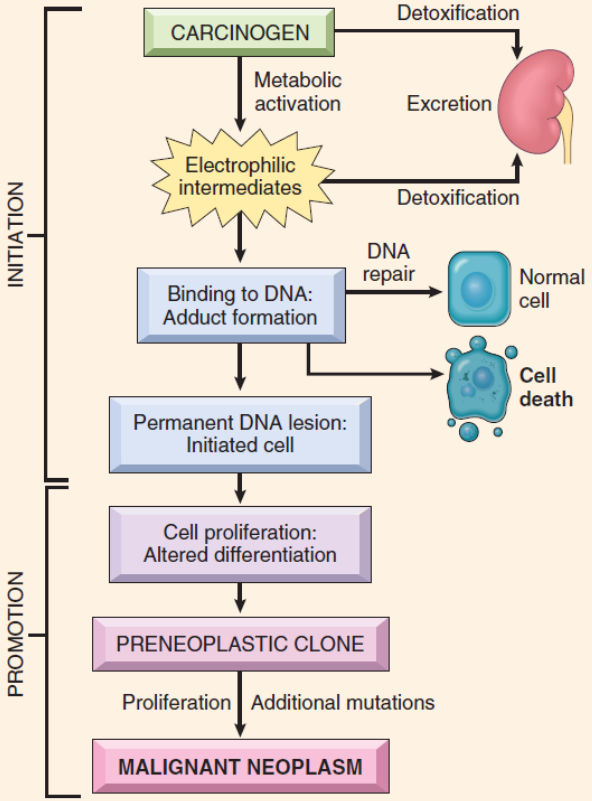
Aflatoxin B_1
Griseofulvin
Cycasin
Safrole
Betel nuts

Others

Nitrosamine and amides
Vinyl chloride, nickel, chromium
Insecticides, fungicides
Polychlorinated biphenyls

All carcinogenic factors can be divided into groups:

1. Chemical
2. Radiation
3. Microbial agents



Chemical carcinogens have highly reactive electrophile groups that directly damage DNA, leading to mutations and eventually cancer.

Radiation Carcinogenesis

- Ionizing radiation causes chromosome breakage, chromosome rearrangements, and, less frequently, point mutations, any of which may affect cancer genes and thereby drive carcinogenesis.

- UV rays in sunlight induce the formation of pyrimidine dimers within DNA, leading to mutations that can give rise to squamous cell carcinomas and melanomas of the skin.

Viral and Microbial Oncogenesis

Oncogenic RNA Viruses

- **HTLV-1 causes *adult T-cell leukemia/lymphoma* (ATLL),** a tumor that is endemic in certain parts of Japan, the Caribbean basin, South America, and Africa, and found sporadically elsewhere.

The HTLV-1 genome encodes a viral protein called *Tax, which* stimulates proliferation, enhances cell survival, and interferes with cell cycle controls.
Although this proliferation initially is polyclonal, the proliferating T cells are at increased risk for secondary mutations that may lead to the outgrowth of a monoclonal leukemia.

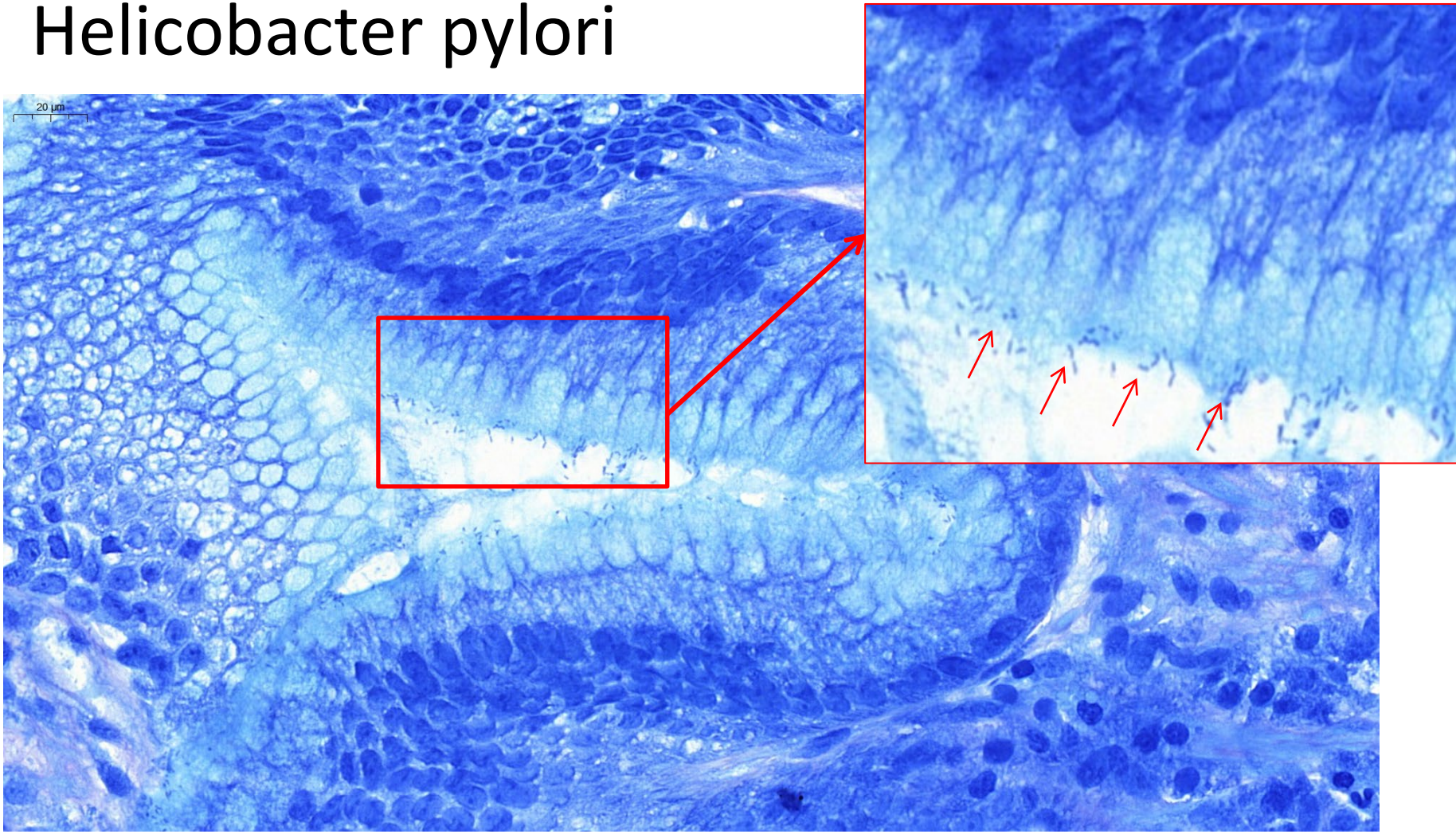
Leukemia develops in only 3% to 5% of the infected individuals, typically after a long latent period of 40 to 60 years.

oncogenic DNA viruses

- HPV,
- Epstein-Barr virus (EBV),
- Kaposi sarcoma herpesvirus (also called human herpesvirus-8),
- polyoma virus called Merkel cell virus,
- hepatitis B virus (HBV)

Virus	effect	Tumor
HPV type 16,18	expression of two viral oncoproteins, E6 and E7, which bind to the p53 and RB tumor suppressors.	Cervical squamous cell carcinoma
EBV	Certain EBV gene products contribute to oncogenesis by stimulating normal B-cell proliferation pathways	Burkitt lymphomas lymphomas in immunosuppressed patients, Hodgkin lymphoma, uncommon T-cell and NK-cell tumors, nasopharyngeal carcinoma, a subset of gastric carcinoma…
HHV8	KRAS and TP53 alterations have been reported in addition to aberrant expression of numerous genes related to neoangiogenesis and proliferation	Kaposi sarcoma
HBV and HCV	immunologically mediated chronic inflammation, with hepatocellular injury, stimulation of hepatocyte proliferation, and production of reactive oxygen species that can damage DNA	hepatocellular carcinomas

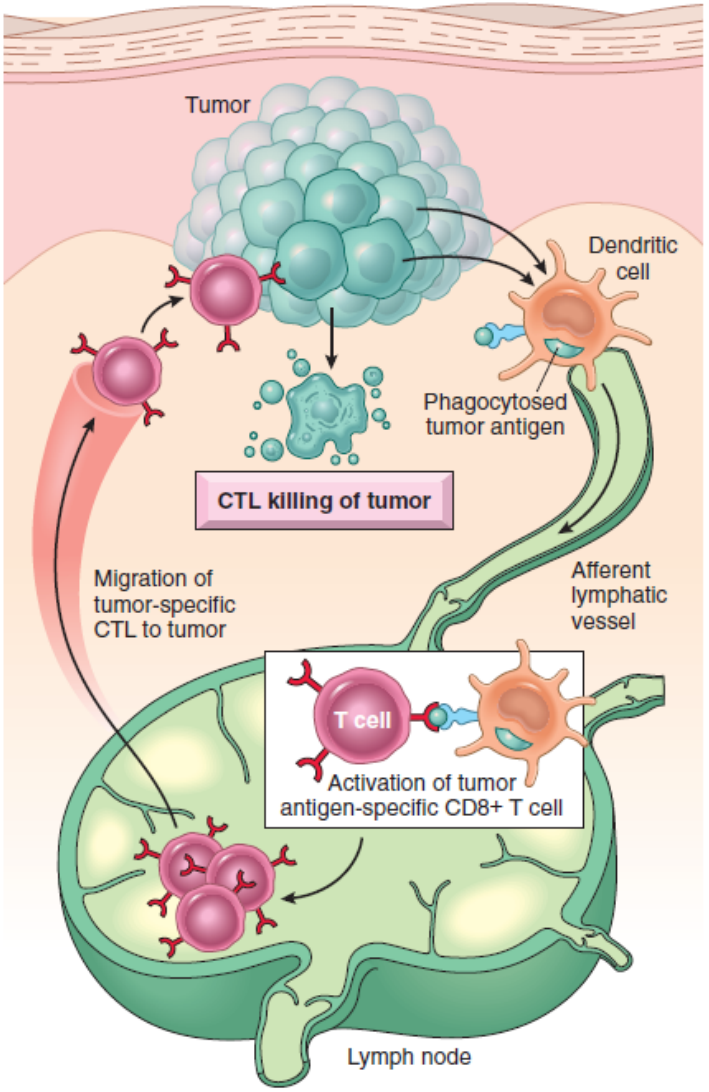
Helicobacter pylori



TUMOR AND IMMUNE REACTIONS

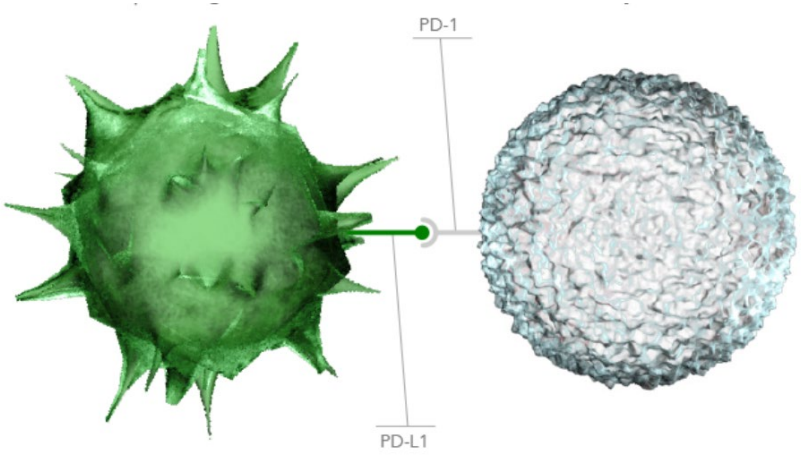
Antitumor activity is mediated by predominantly cell-mediated mechanisms. Tumor antigens are presented on the cell surface by MHC class I molecules and are recognized by CD8+ CTLs.

Tumors may avoid the immune system by several mechanisms, including selective outgrowth of antigen-negative variants, loss or reduced expression of histocompatibility molecules, and immunosuppression mediated by expression of certain factors

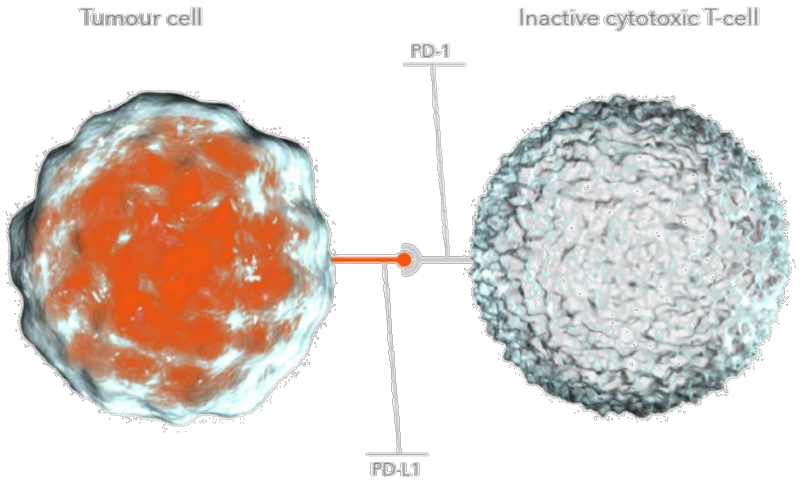


PD-L1

significant proportion of tumors of various localizations, despite the pronounced infiltration of cytotoxic T-cells, are not destroyed.



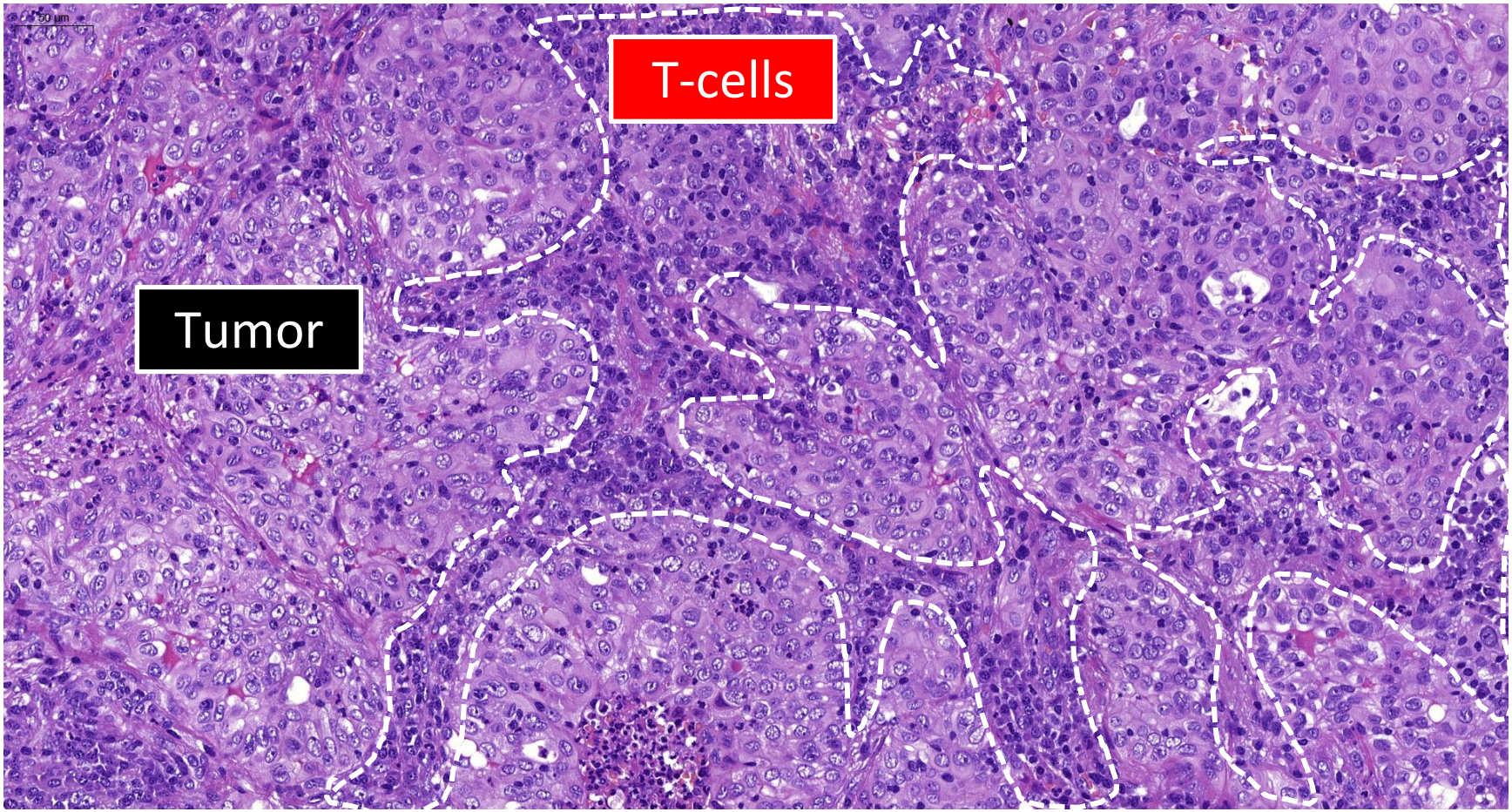
Inactivation of T-cells limits damage to normal tissues.



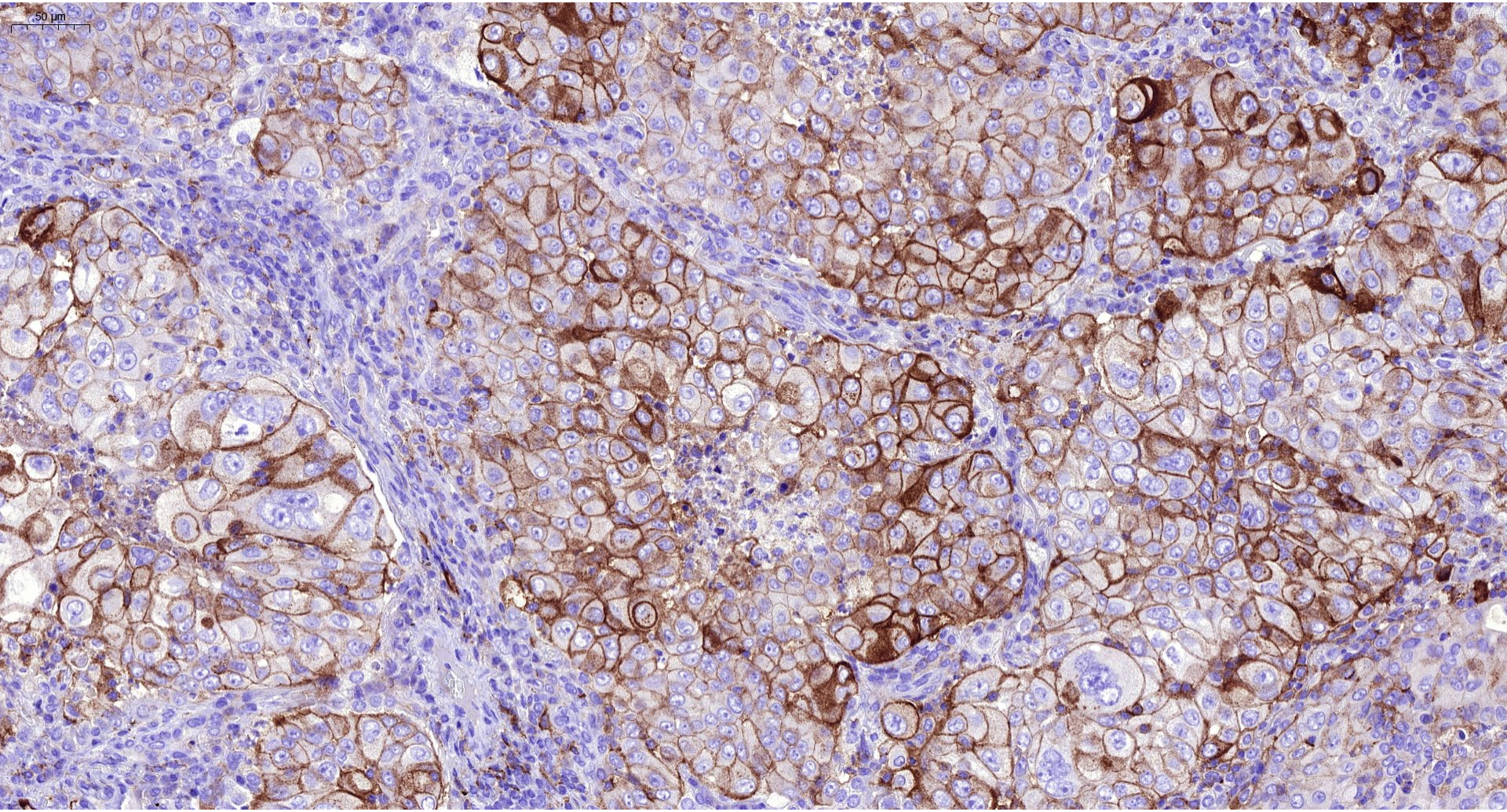
Upon binding to PD-1, PD-L1 delivers a suppressive signal to T-cells and an antiapoptotic signal to tumor cells, leading to T-cell dysfunction and tumor survival.

https://www.oncologica.com/immunofocus/

Lung adenocarcinoma



Lung adenocarcinoma
PD-L1 expression



All tumor cells express PD-L1

CLINICAL ASPECTS OF TUMOR GROWTH

Evaluation of malignant potential of tumors is based on a basic component – **"TNM-classification"** and other morphological, genetic, clinical, biochemical indicators

T - Tumor = indicates the size and extent of the primary tumor focus.
N - Node = involvement of regional lymph nodes in the metastatic process.
M - Metastasis = presence of distant metastases.

T – Primary tumour

TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Tis	Carcinoma in situ
Tis (DCIS)	Ductal carcinoma in situ
Tis (LCIS)	Lobular carcinoma in situ
Tis (Paget)	Paget disease of the nipple not associated with invasive carcinoma and/or carcinoma in situ (DCIS and/or LCIS) in the underlying breast parenchyma.

Note: Carcinomas in the breast parenchyma associated with Paget disease are categorized based on the size and characteristics of the parenchymal disease, although the presence of Paget disease should still be noted.

T1	Tumour 2 cm or less in greatest dimension
T1mi	Microinvasion 0.1 cm or less in greatest dimension*

Note: *Microinvasion is the extension of cancer cells beyond the basement membrane into the adjacent tissues with no focus more than 0.1 cm in greatest dimension. When there are multiple foci of microinvasion, the size of only the largest focus is used to classify the microinvasion. (Do not use the sum of all individual foci.) The presence of multiple foci of microinvasion should be noted, as it is with multiple larger invasive carcinomas.

T1a	More than 0.1 cm but not more than 0.5 cm in greatest dimension
T1b	More than 0.5 cm but not more than 1 cm in greatest dimension
T1c	More than 1 cm but not more than 2 cm in greatest dimension
T2	Tumour more than 2 cm but not more than 5 cm in greatest dimension
T3	Tumour more than 5 cm in greatest dimension
T4	Tumour of any size with direct extension to chest wall and/or to skin (ulceration or skin nodules)

Note: Invasion of the dermis alone does not qualify as T4. Chest wall includes ribs, intercostal muscles, and serratus anterior muscle but not pectoral muscle.

T4a	Extension to chest wall (does not include pectoralis muscle invasion only)
T4b	Ulceration, ipsilateral satellite skin nodules, or skin oedema (including peau d'orange)
T4c	Both 4a and 4b, above
T4d	Inflammatory carcinoma

N – Regional lymph nodes

NX	Regional lymph nodes cannot be assessed (e.g. previously removed)
N0	No regional lymph-node metastasis
N1	Metastasis in movable ipsilateral level I, II axillary lymph node(s)
N2	Metastasis in ipsilateral level I, II axillary lymph node(s) that are clinically fixed or matted; or in clinically detected* ipsilateral internal mammary lymph node(s) in the absence of clinically evident axillary lymph-node metastasis
N2a	Metastasis in axillary lymph node(s) fixed to one another (matted) or to other structures
N2b	Metastasis only in clinically detected* internal mammary lymph node(s) and in the absence of clinically detected axillary lymph-node metastasis
N3	Metastasis in ipsilateral infraclavicular (level III axillary) lymph node(s) with or without level I, II axillary lymph-node involvement; or in clinically detected* ipsilateral internal mammary lymph node(s) with clinically evident level I, II axillary lymph-node metastasis; or metastasis in ipsilateral supraclavicular lymph node(s) with or without axillary or internal mammary lymph node involvement
N3a	Metastasis in infraclavicular lymph node(s)
N3b	Metastasis in internal mammary and axillary lymph nodes
N3c	Metastasis in supraclavicular lymph node(s)

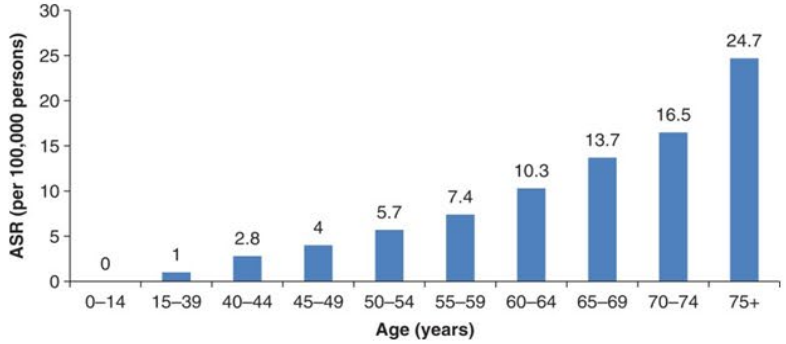
Note: * "Clinically detected" is defined as detected by clinical examination or by imaging studies (excluding lymphoscintigraphy) and having characteristics highly suspicious for malignancy or a presumed pathological macrometastasis based on fine-needle aspiration biopsy with cytological examination. Confirmation of clinically detected metastatic disease by fine-needle aspiration without excision biopsy is designated with an (f) suffix, e.g., cN3a(f).
Excisional biopsy of a lymph node or biopsy of a sentinel node, in the absence of assignment of a pT, is classified as a clinical N, e.g., cN1. Pathological classification (pN) is used for excision or sentinel lymph node biopsy only in conjunction with a pathological T assignment.

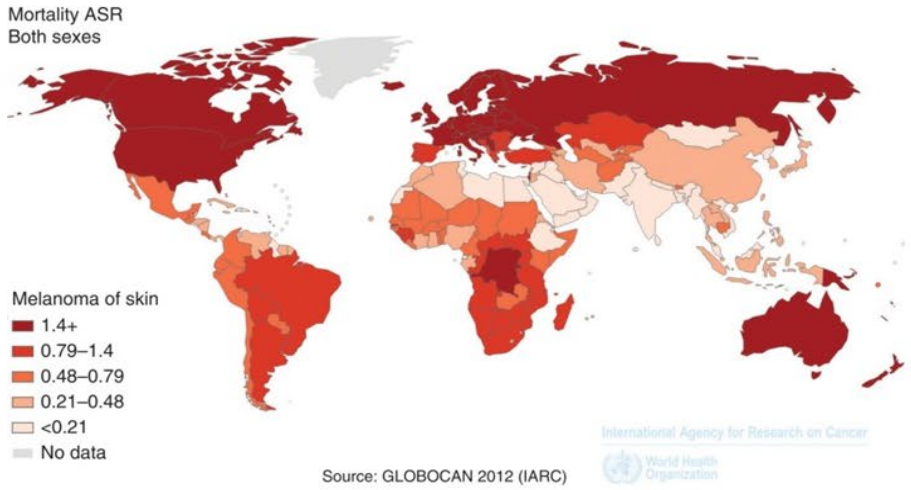
M – Distant metastasis

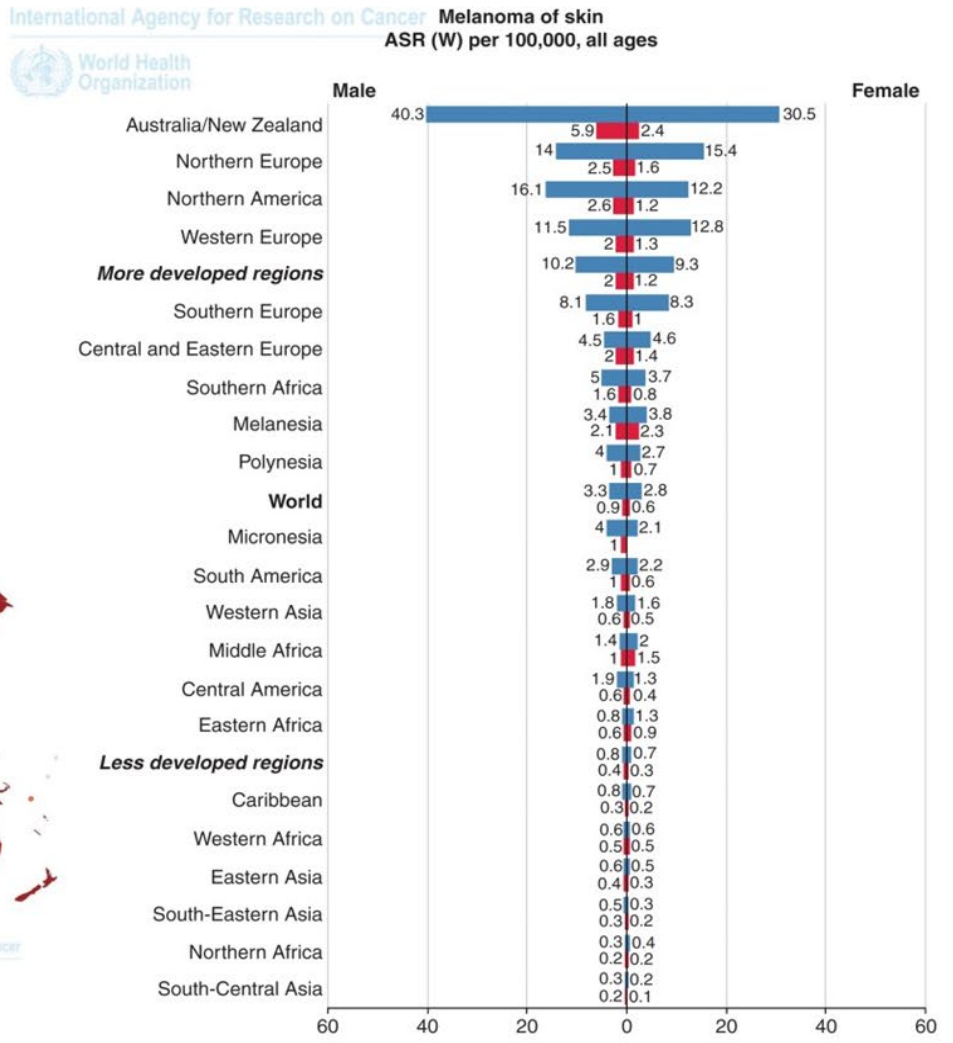
M0	No distant metastasis
M1	Distant metastasis

Tumors from tissues - derivatives of mesenchyme, neuroectoderm, melanin-producing tissue.

Epidemiology







Risk factors

- As with other cutaneous malignancies, melanoma is mainly caused by UV light–induced DNA damage that leads to the stepwise acquisition of driver mutations.

1. family history of melanoma,
2. large number of nevi,
3. fair skin,
4. exposure to ultraviolet light,
5. age over 50 years,
6. genodermatosis (xeroderma, etc.).

Macroscopic signs of melanoma:

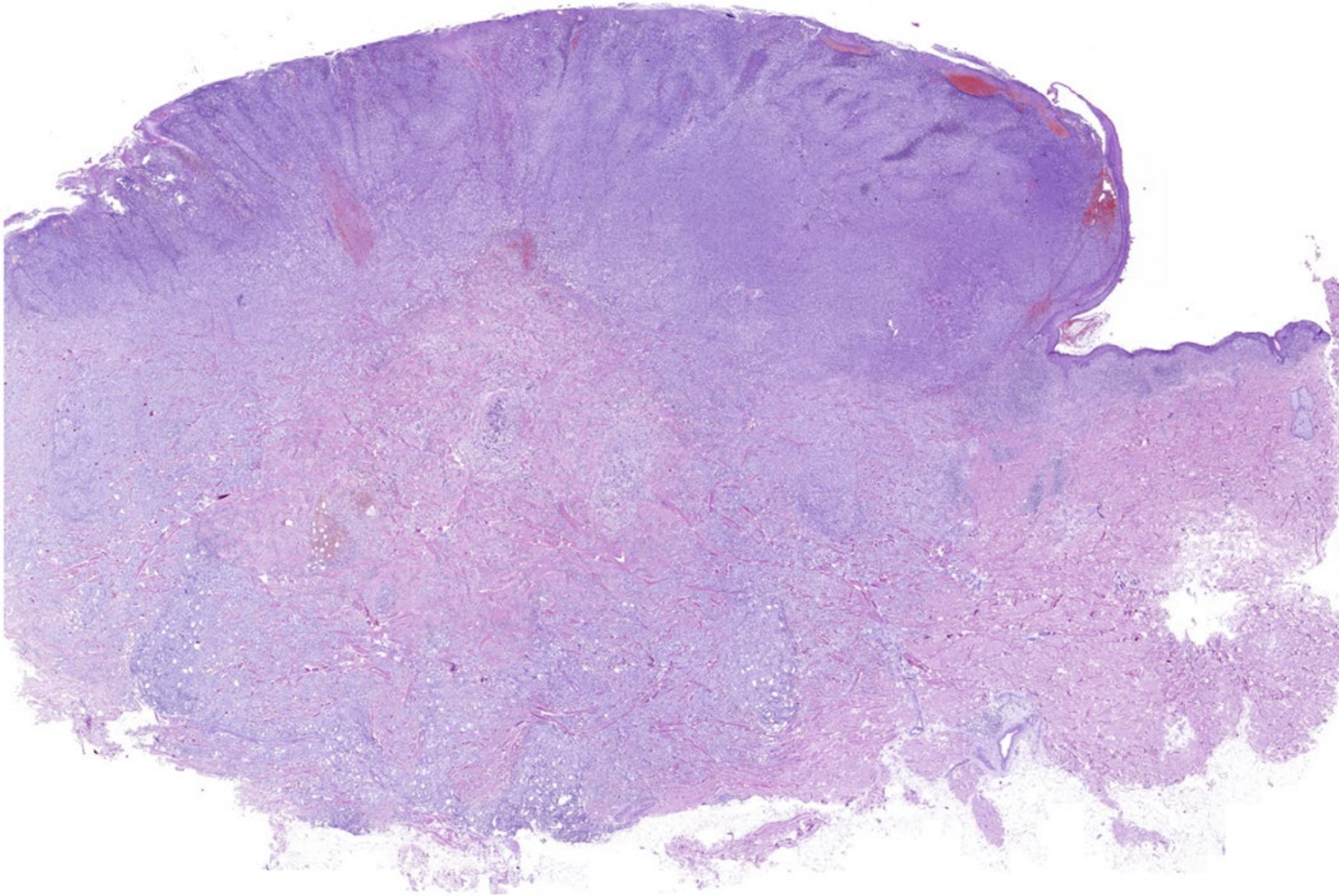
1. Asymmetry of pigmentation
2. Irregular contours of pigment distribution
3. Variations in the intensity of pigmentation in the redistribution of one focus
4. The diameter of the focus is more than mm5.
5. Dynamic change of focus color.

Morphological criteria for the diagnosis of melanoma

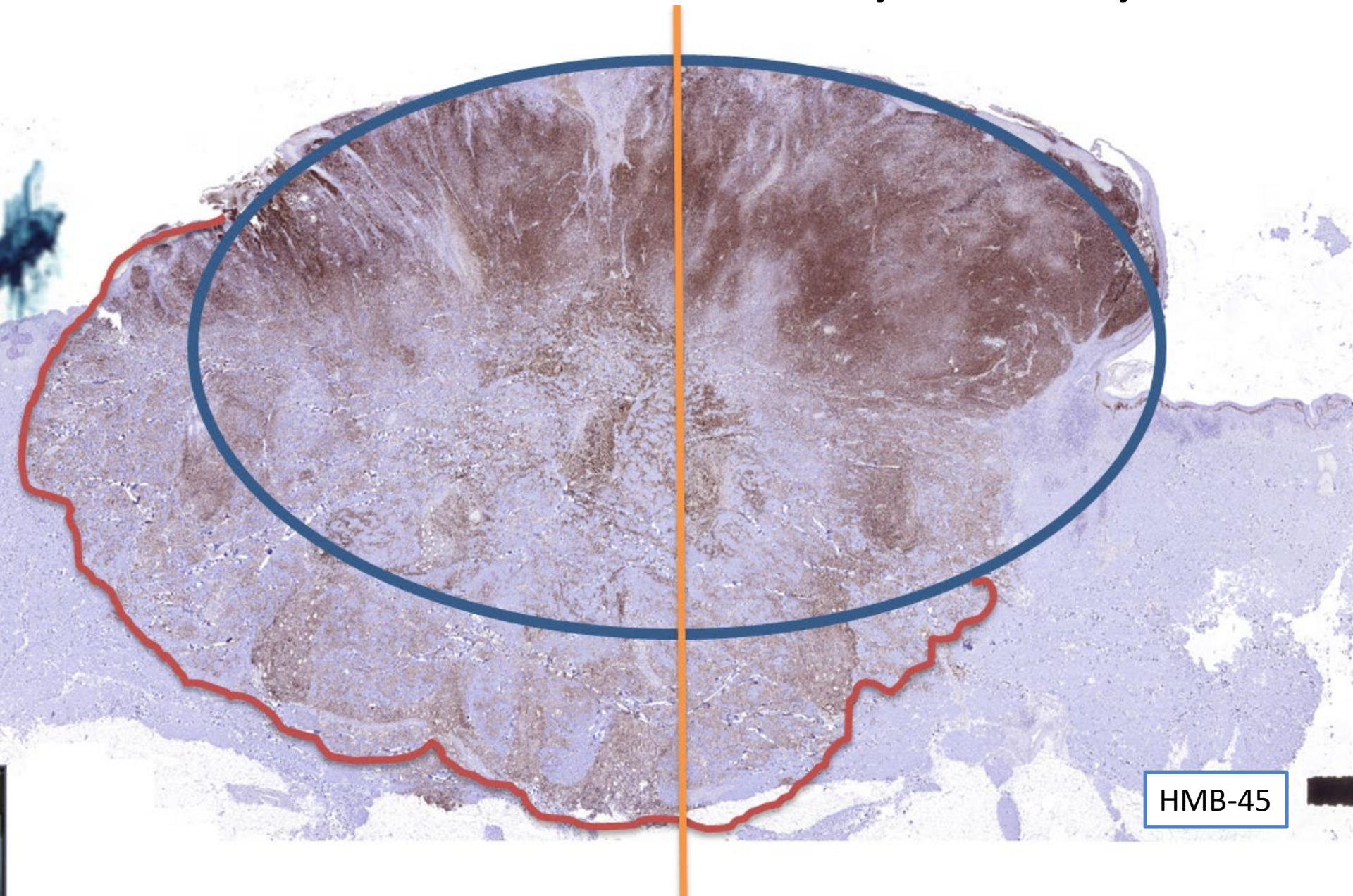
Morphological diagnosis of melanoma consists of a comprehensive analysis of the criteria:

- 1. Architectural;
- 2. Cytological;
- 3. environment.

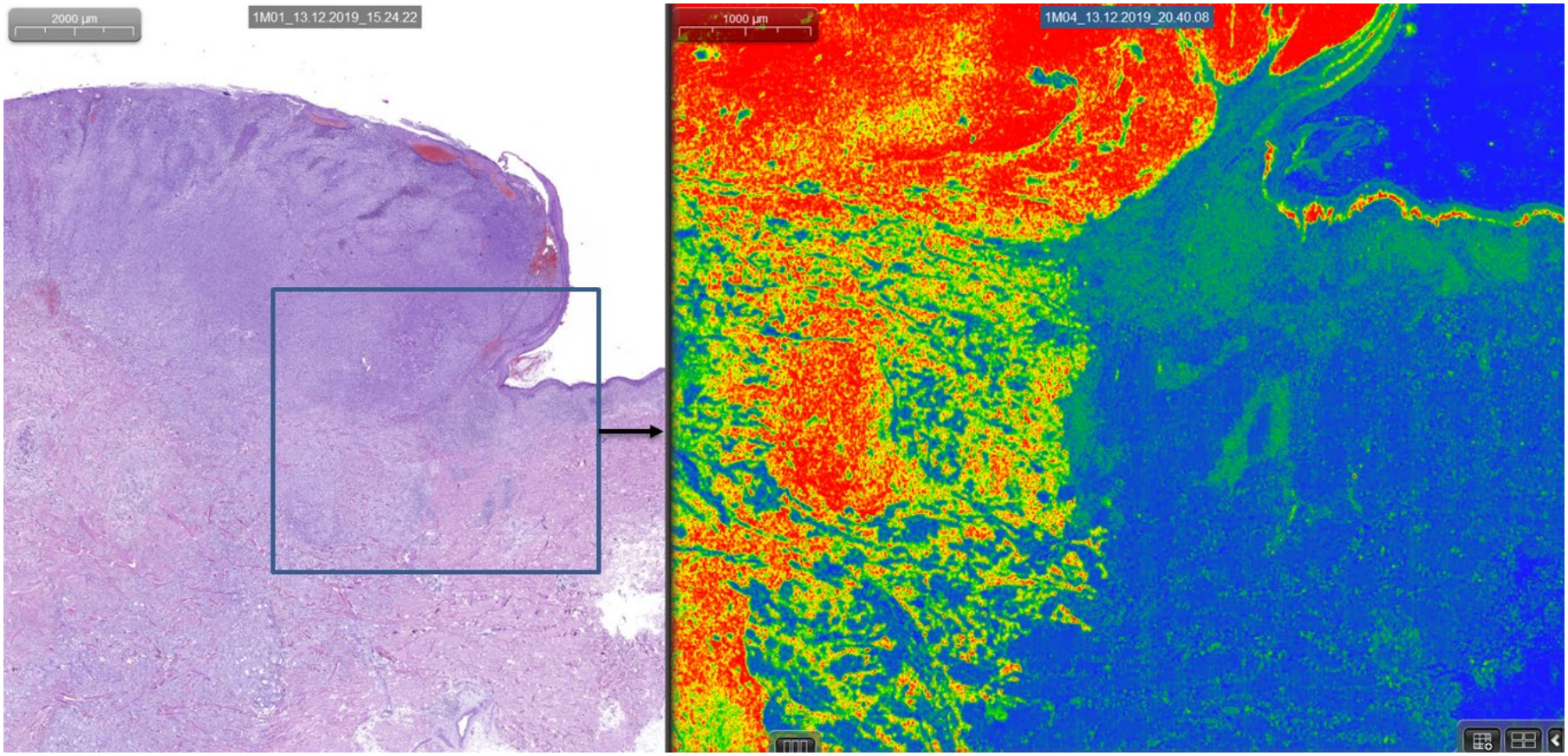
Vertical and lateral asymmetry



Vertical and lateral asymmetry

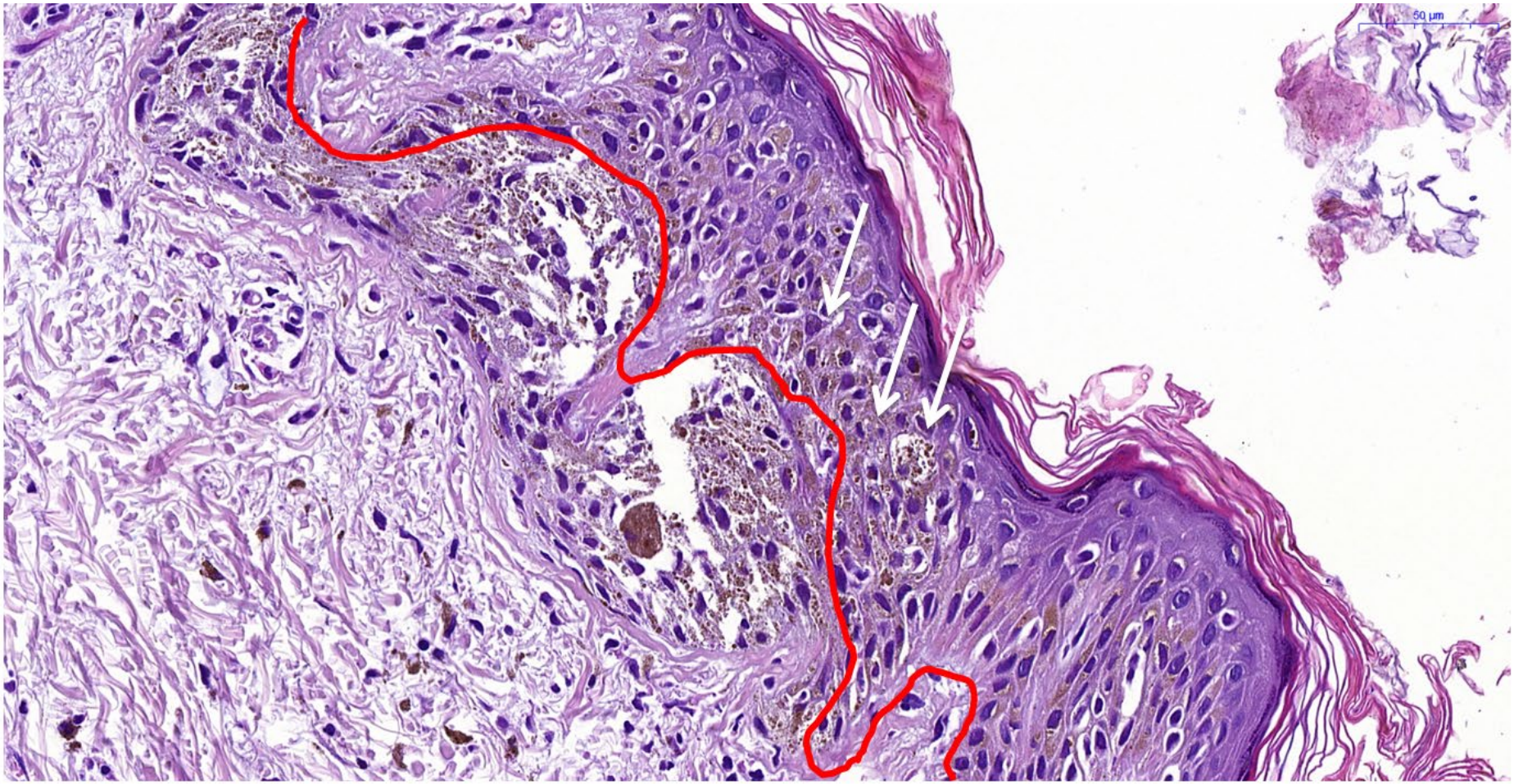


Poor detachment

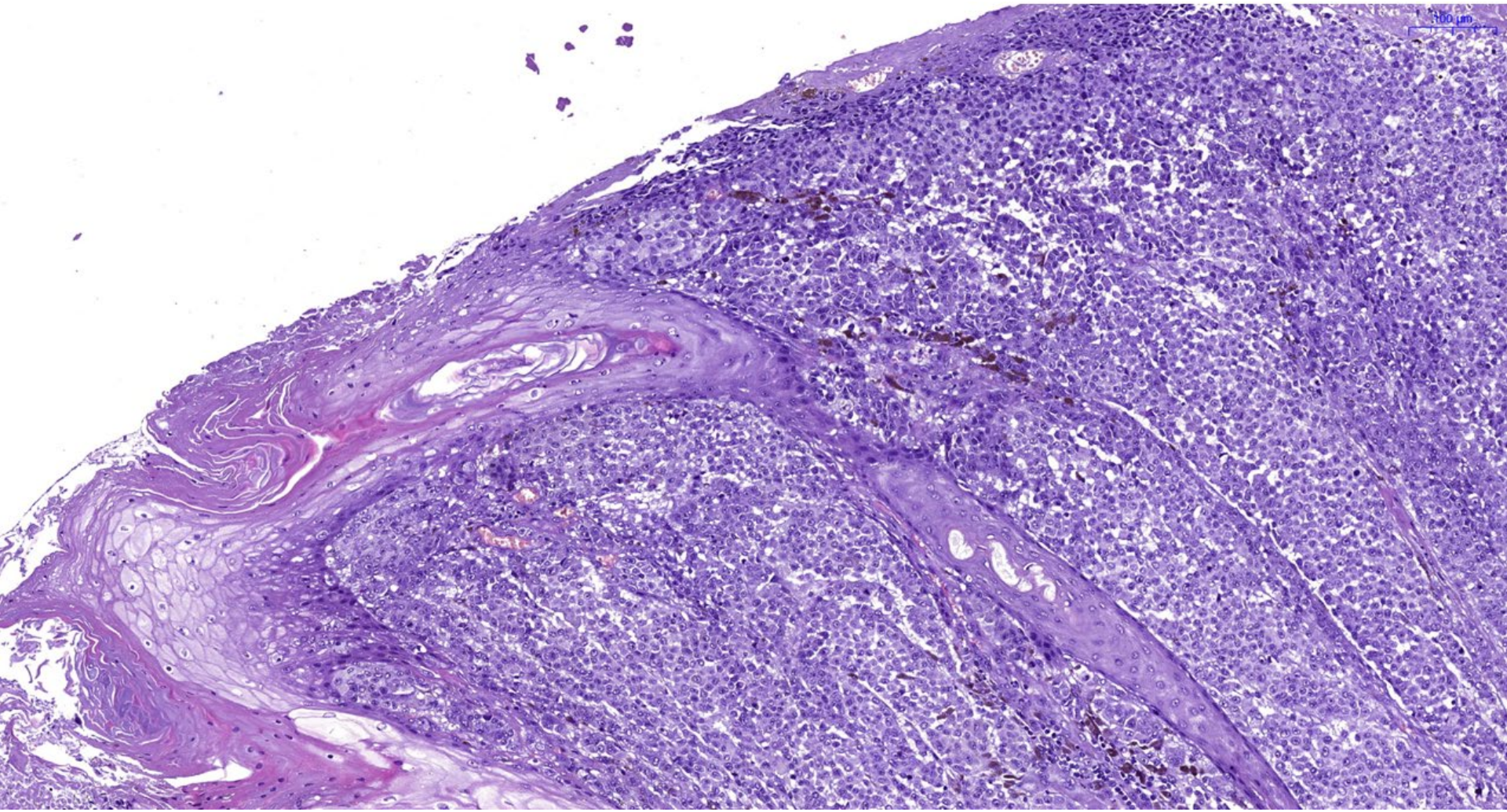


Tumor proliferation in melanoma has irregular boundaries, which is a manifestation of the infiltrative type of growth characteristic of many malignant tumors.

The spread of tumor cells into the surface layers of the epidermis



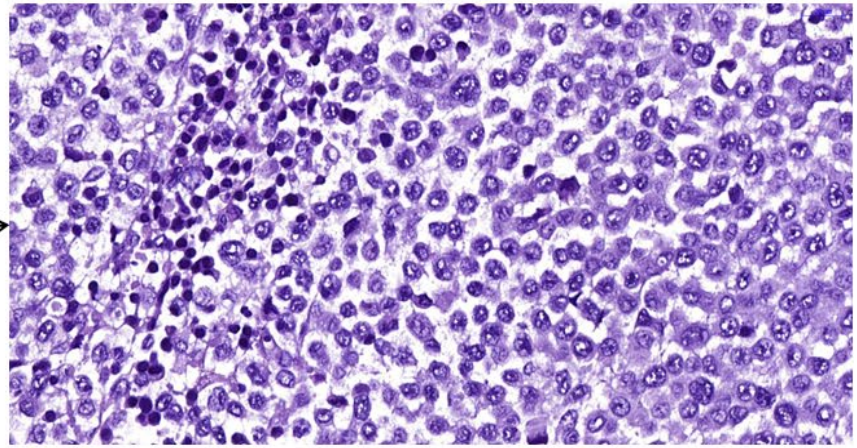
Replacement of the epidermis

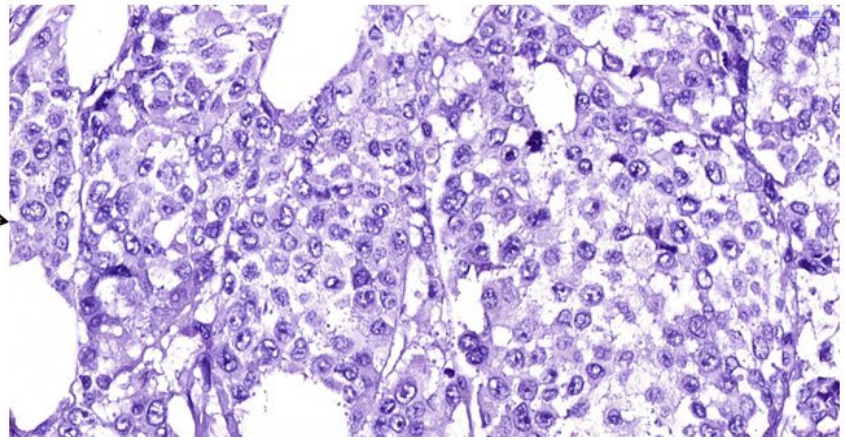


Expressed colonization of the epidermal layer by tumor cells leads to its destruction

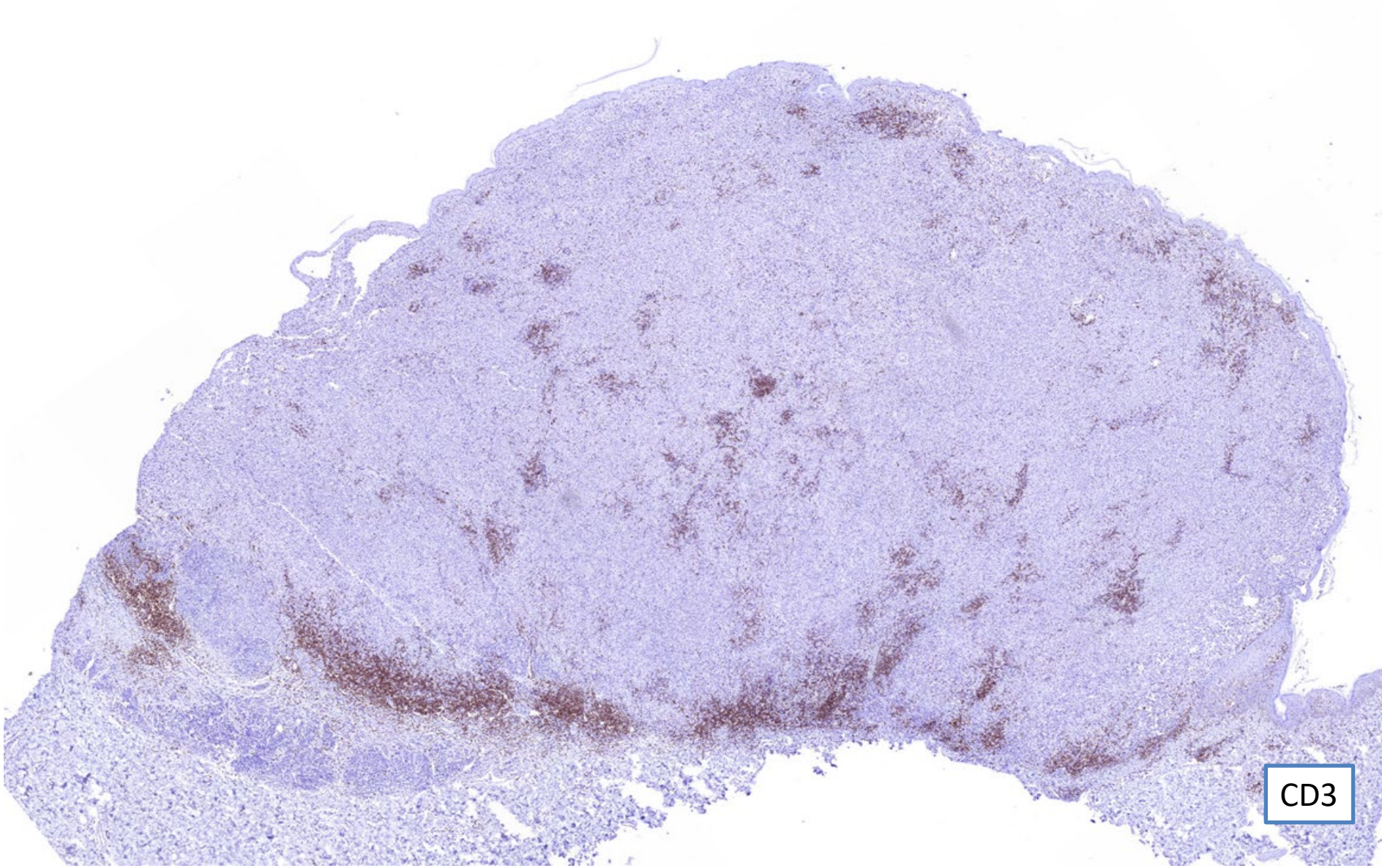
Lack of vertical "maturation"

Tumor cells not only retain their size,
but also often increase in size.

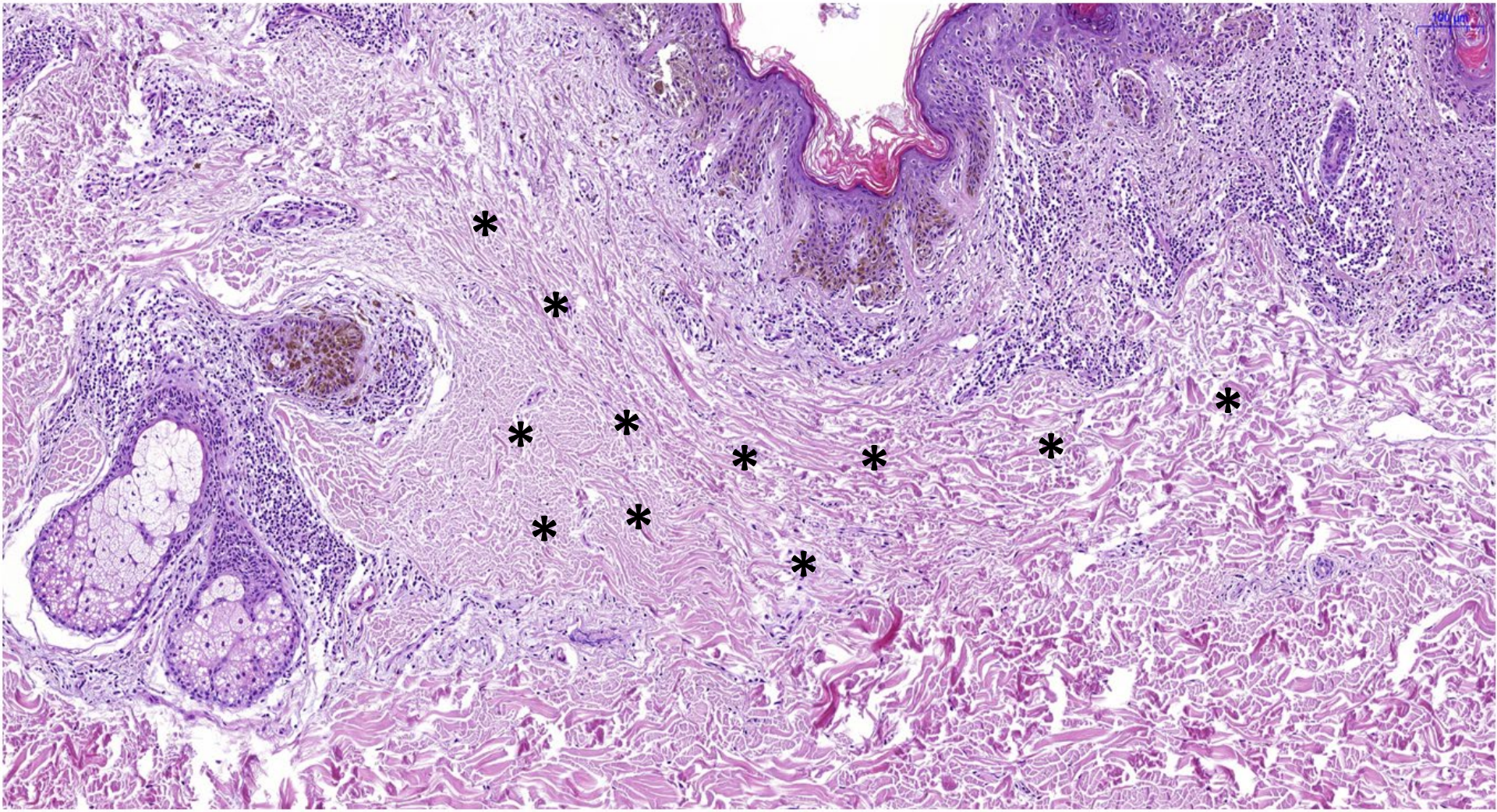




Demarcation inflammatory infiltrate

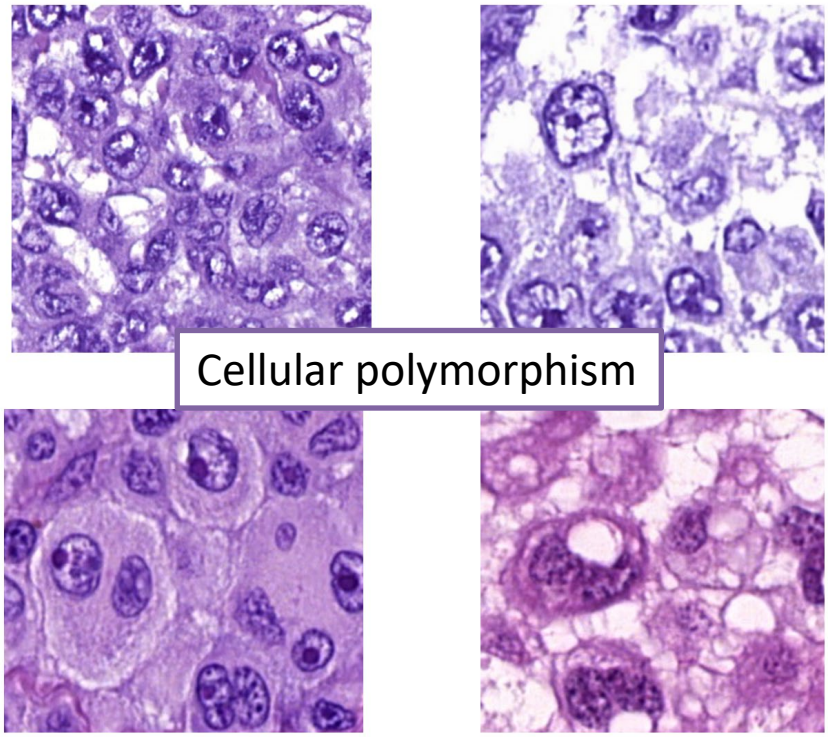


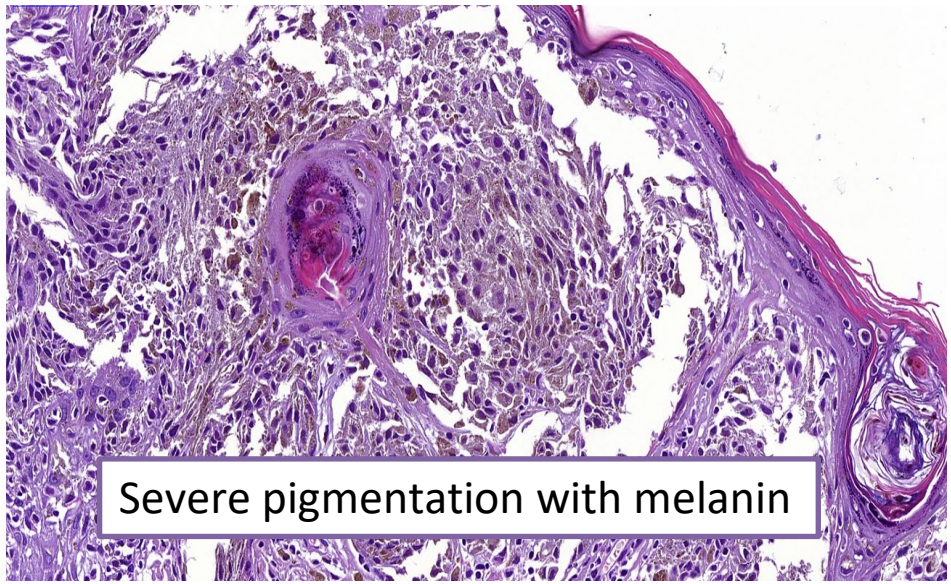
Desmoplastic reaction

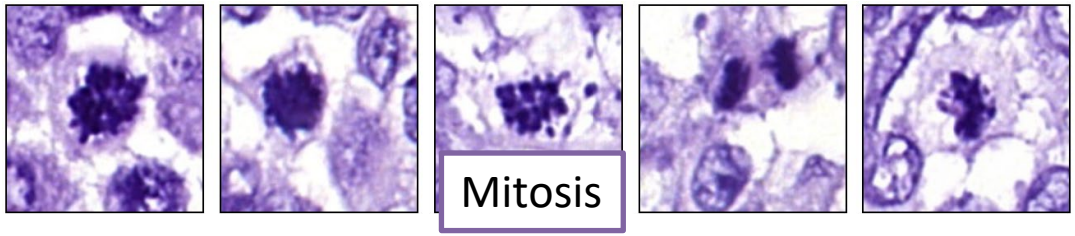


Cytological criteria

- Cellular polymorphism
- Mitosis
- Severe pigmentation with melanin

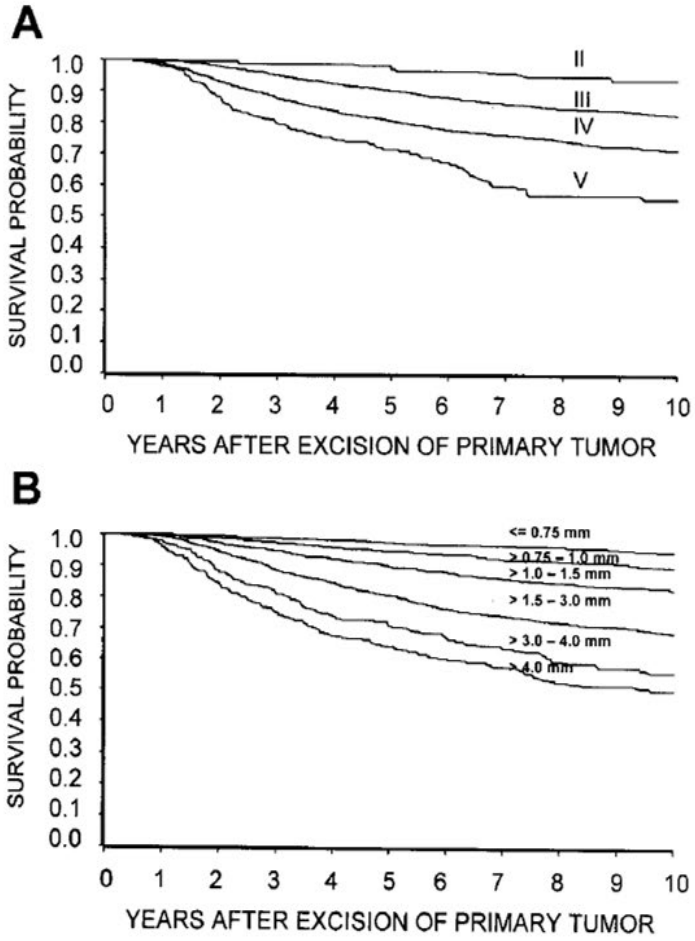


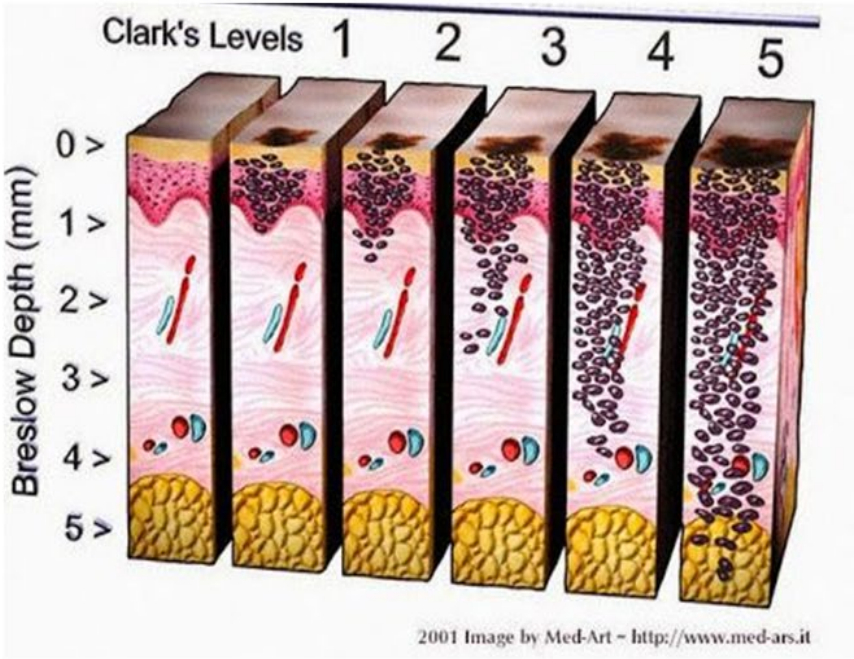




prognosis in melanoma

(Clark's level of invasion and Breslow's depth of invasion)





CANCER March 1, 2001 / Volume 91 / Number 5

CNS TUMORS

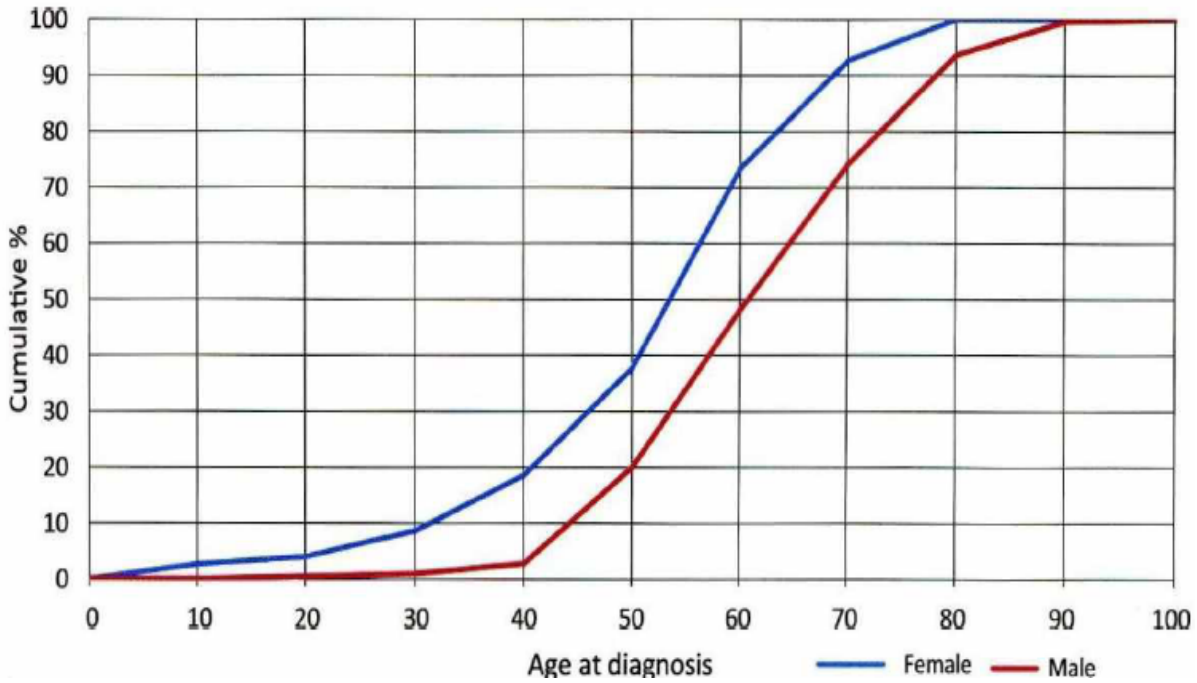
FEATURES OF CNS TUMORS

- lead to compression of the brain or spinal cord;
- grow into the brain tissue, causing its destruction;
- often accompanied by the development of hemorrhages and foci of necrosis;
- lead to impaired cerebrospinal fluid flow, followed by hydrocephalus;
- often cause cerebral edema;
- astrocytic tumors metastasize within the brain and spinal cord (except in cases of brain bypass surgery).

TUMORS FROM ASTROCYTES

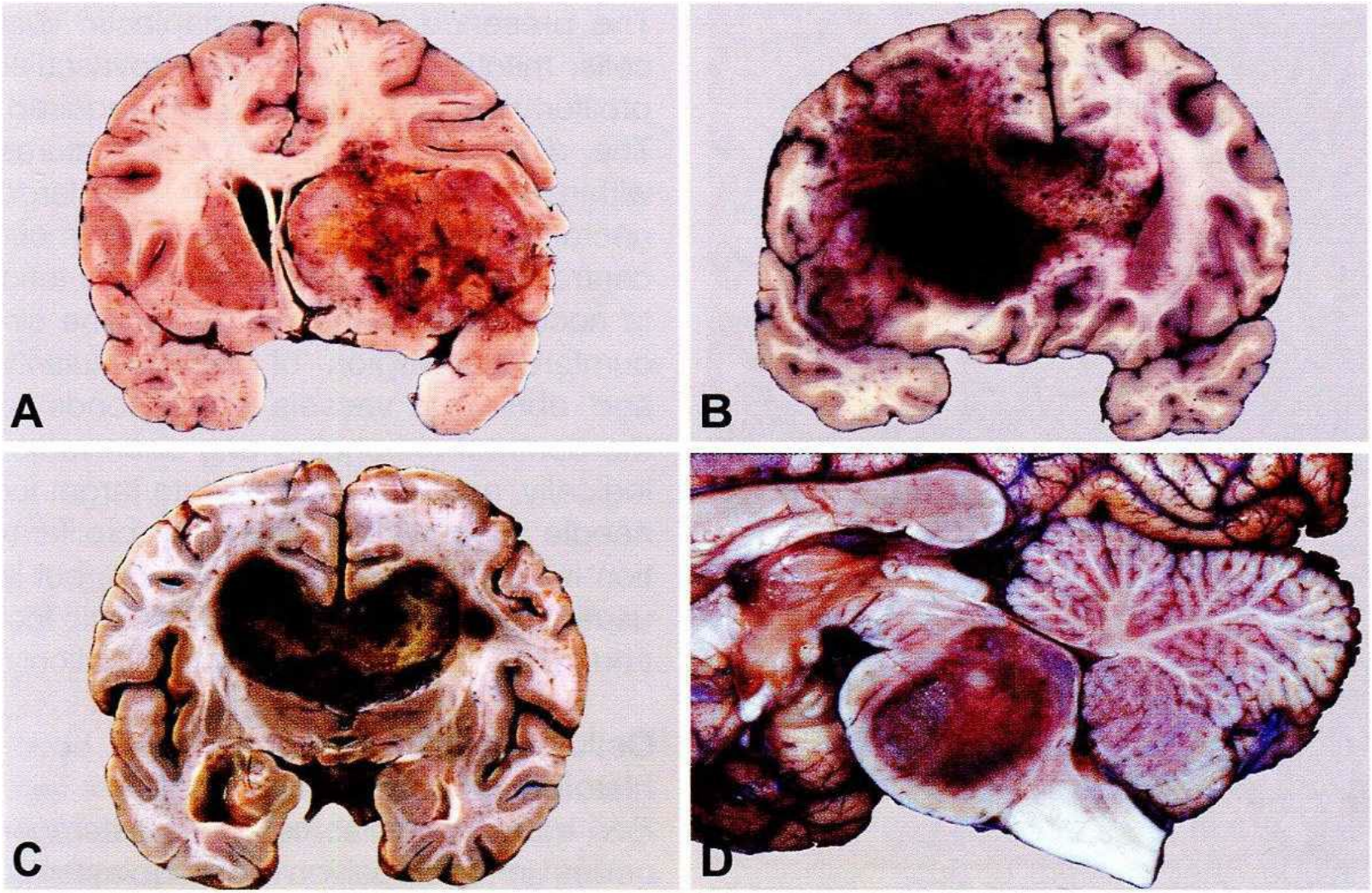
4 large subgroups can be distinguished, based on the progenitor cell.

1. Astrocytomas
2. Oligoastrocytomas
3. Oligodendrogliomas
4. Glioblastoma



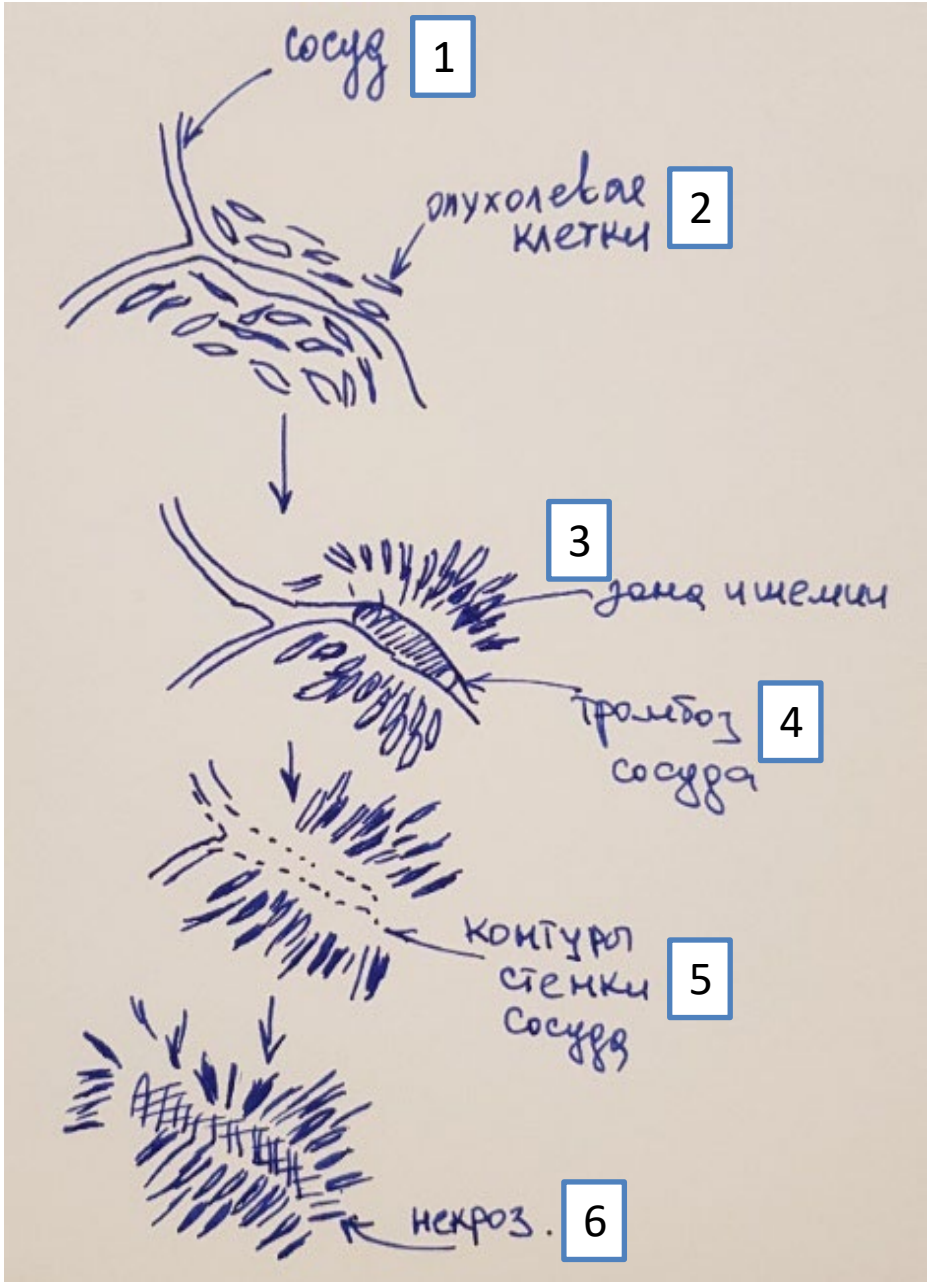
David N. Louis, Hiroko Ohgaki, Otmar D. Wiestler, Webster K. Cavenee (Eds): WHO Classification of Tumours of the Central Nervous System (Revised 4th edition). IARC: Lyon 2016.

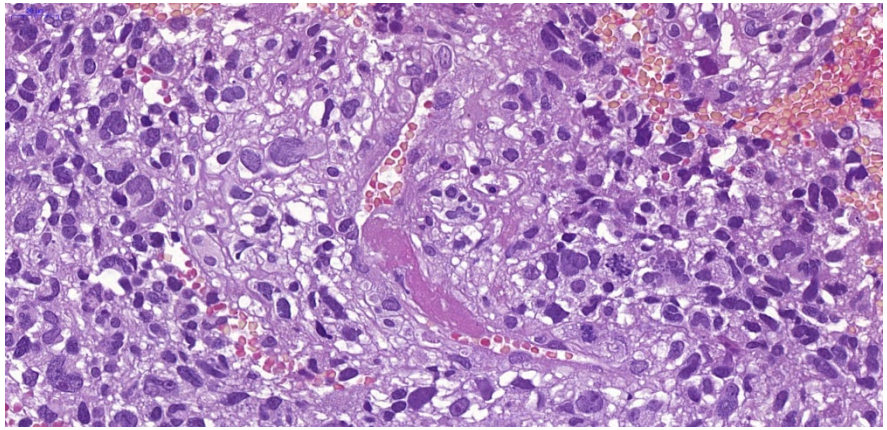
macroscopic picture

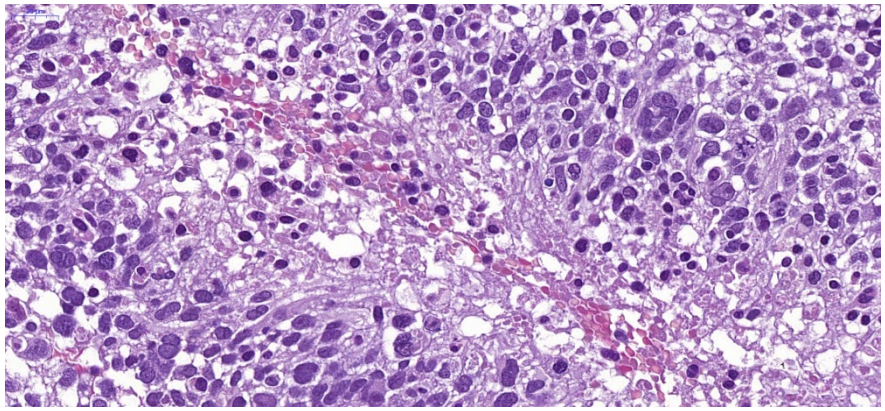


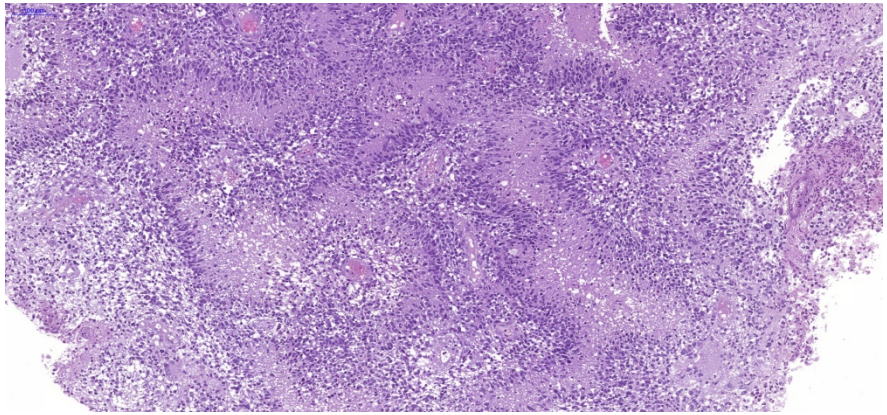
David N. Louis, Hiroko Ohgaki, Otmar D. Wiestler, Webster K. Cavenee (Eds): WHO Classification of Tumours of the Central Nervous System (Revised 4th edition). IARC: Lyon 2016.

morphogenesis of glioblastoma

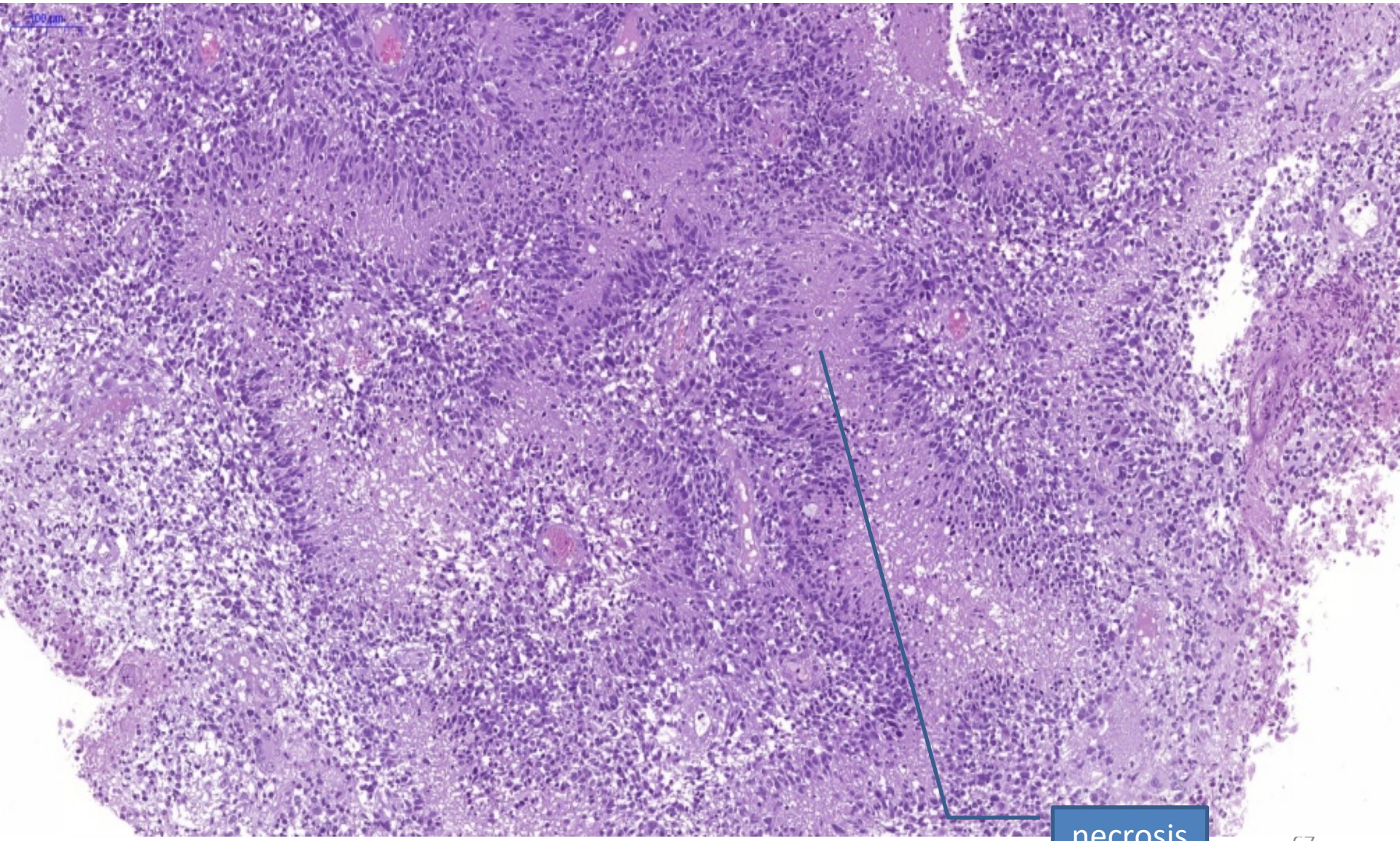




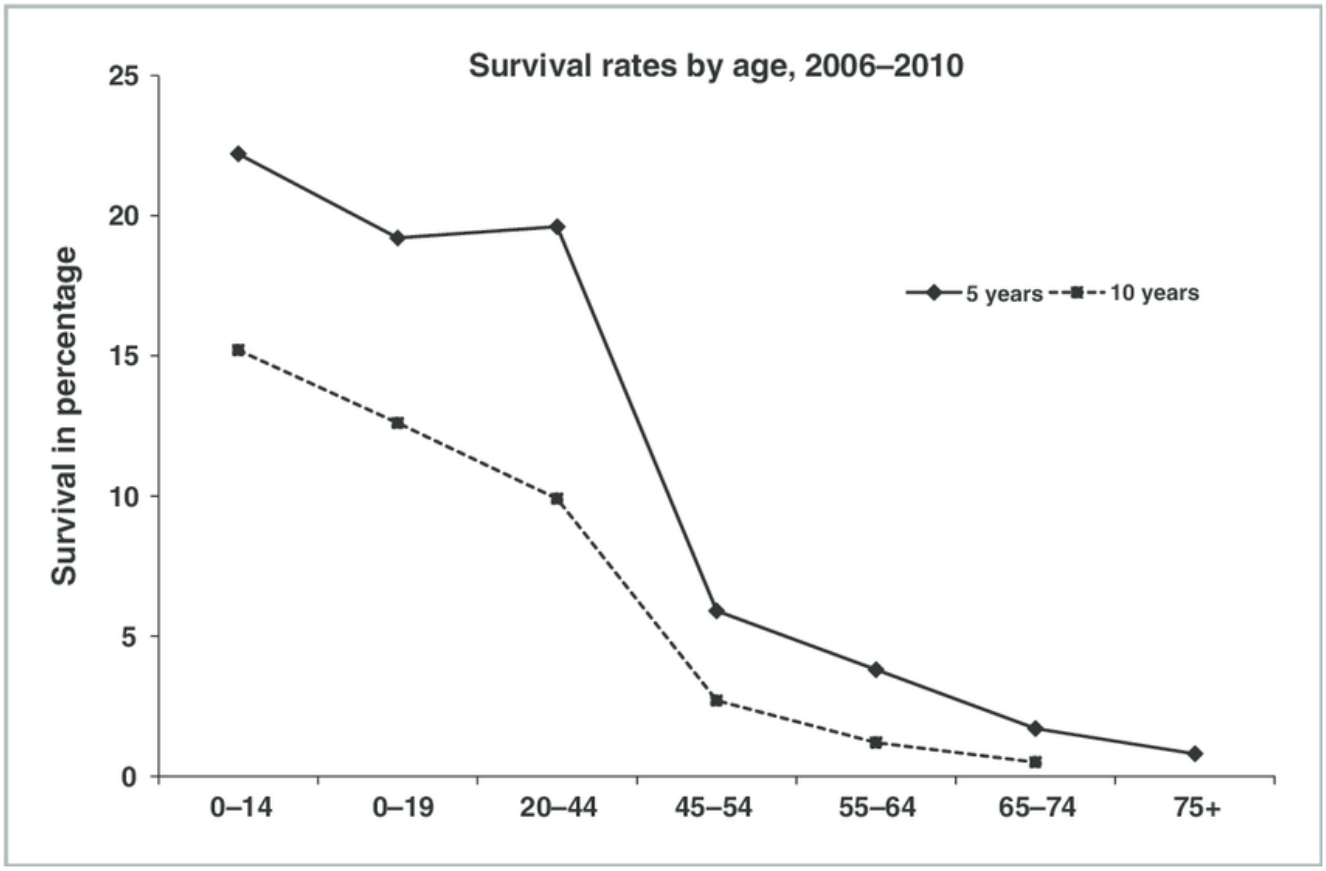




Glioblastoma with typical necrosis



Prognosis



Five-and 10-year relative survival rates for glioblastoma by age, SEER 18 registries, 1995 to 2010. X-axis, age groups; Y-axis, survival in percentage. Rates are in percentage (%). Estimated by CBTRUS using SEER Program (www.seer.cancer.gov) SEER Ã Stat Database: Incidence—SEER 18 Regs Research Data þ Hurricane Katrina Impacted Louisiana Cases, Nov 2011 Sub (1973–2009 varying)—linked to county attributes—total U.S., 1969–2010 counties, National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2012, based on the November 2011 submission (2).

Thanks for attention!