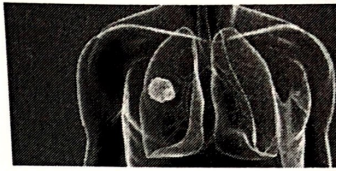

# Anticancer drugs

Pharmacology 3

Dr. Rawan Abudalo

Department of Clinical Pharmacy and Pharmacy Practice

Faculty of Pharmaceutical Sciences

Hashemite University

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* حكت الدكتوره الـ Backgroud slide اعتبرو قراءه

# Background

* لما نحكي malignancy فهاد يعني إنه خبيث و بمعنى إنه شرس و بالـ further screening منقدر نحدد إذا كان malignant أو benign tumor.

- It is the **second most common cause of death in the developed nations**
- **One in three people** will be diagnosed with cancer during their lifetime
- There are approximately 200 types of cancer, each with different causes, symptoms and treatments
- The terms *cancer*, *malignant neoplasm* and *malignant tumour* are synonymous. ← أسماء أخرى
- cancerous cell is uncontrollably growing and has the potential for invading local tissue and spreading to other parts of the body, a process called metastases.

* لما الـ Cancer ينتشر ويروح للـ neighboring tissue و الـ metastases هي من الـ classification بأنه السرطان يعتبر malignant.

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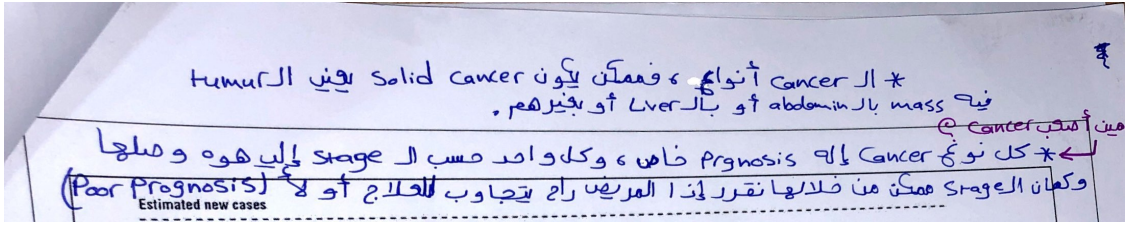


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* يجب لما يجي المريض عالدكتور منشوف هو بأي Stage
فهل هو كان بالـ metastases stages مثلاً . والأصعب هو
لما يكون المريض asymptomatic لكن أحياناً بشعر بـ bone Pain
وما نقدر لهيك ويس يفحصوه بلاقو إنه السرطان عنده منتشر عالعظم ،
وهاد بأثر عالـ Prognosis وعلى العلاج .
فكل ما المريض إجى بوقت أبكر من بداية السرطان فهون بكون عنده
good prognosis للمريض .

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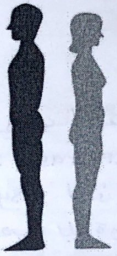
Estimated new cases

| | | | Males | Females | | | |
|---|---|---|---|---|---|---|---|
| Prostate | 174,650 | 20% |  |  | Breast | 268,600 | 30% |
| Lung & bronchus | 116,920 | 13% | | | Lung & bronchus | 111,710 | 13% |
| Colon & rectum | 78,500 | 9% | | | Colon & rectum | 67,100 | 8% |
| Urinary bladder | 61,700 | 7% | | | Uterine corpus | 61,880 | 7% |
| Melanoma of the skin | 57,220 | 7% | | | Melanoma of the skin | 39,260 | 4% |
| Kidney & renal pelvis | 44,120 | 5% | | | Thyroid | 37,810 | 4% |
| Non-Hodgkin lymphoma | 41,090 | 5% | | | Non-Hodgkin lymphoma | 33,110 | 3% |
| Oral cavity & pharynx | 38,140 | 4% | | | Kidney & renal pelvis | 29,700 | 3% |
| Leukemia | 35,920 | 4% | | | Pancreas | 26,830 | 3% |
| Pancreas | 29,940 | 3% | | | Leukemia | 25,860 | 3% |
| **All sites** | **870,970** | **100%** | | | **All sites** | **891,480** | **100%** |

+ ممكن بالأردن أعلى إشي بال males هوه ال Lung cancer أو ال Colorectal Cancer

Estimated 2019 cancer incidences in the United States for males and females.

* سواء بال US أو عنّا هون وجنّو إنو ال highest incedince of cancer in females is the breast cancer (30%) and the lowest is the pancreas (3%)

* for males, the highest incidence is Prostate cancer (20%) and the lungs (13%) because it's correlated to smoking.

* ال hematological cancer هوه سرطان بالدم وهوه أنواع زي Leukemia و Lymphoma و الـ Lymphoma كمان إلها أنواع

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* بتميزهم عن الـ normal cells

- Cancer cells manifest, to varying degrees, four characteristics that distinguish them from normal cells:
  1) *Uncontrolled proliferation* (cell division)
  2) *Dedifferentiation & loss of function*
  3) *Invasiveness* (إنتشار)
  4) *Metastasis*

* حيث إنه خلايا جسمنا لما بالبداية بيصيرلهم إنقسام فهمه بيصيرلهم differentiation يعني بتخصص لـ functions معينة مثلاً إشي بيصير epithelial وإشي بروح عالـ endocrine وإشي بروح عالـ neurons . وبالسرطان هاي الخلايا بتبطل تتعرف عالـ identity تاعتها و بتفقد الـ function تاعها

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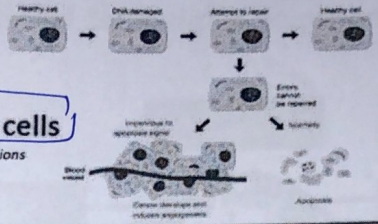

**Cancer cells**

*Multiple mutations*

**Normal cells**

| | | |
|---|---|---|
| ~~proto~~ – oncogenes (GFs)<br>tumour ~~suppressors~~ | **1. Uncontrolled proliferation** | proto - oncogenes<br>tumour suppressors |
| immature, loss of function, 'immortal' | **2. De-differentiation** | mature, defined function, defined lifetime |
| actively leave tissue of origin and go to other tissues | **3. Invasiveness** | stays in tissue of origin *(need survival signals)* |
| can proliferate in foreign tissues | **4. Metastasis** | die if escape own tissue |

(well differentiated)

(no invasiveness)

↓

abnormality أي * يكون لو عندهم
apoptosis يصير عندهم

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Cell

* شو بالي بميز السرطان ٥ وليه بميز ٥ وليه بتطور.

Leading Edge
Review

# Hallmarks of Cancer: The Next Generation

Douglas Hanahan[1,2,*] and Robert A. Weinberg[3,*]
[1]The Swiss Institute for Experimental Cancer Research (ISREC), School of Life Sciences, EPFL, Lausanne CH-1015, Switzerland
[2]The Department of Biochemistry & Biophysics, UCSF, San Francisco, CA 94158, USA
[3]Whitehead Institute for Biomedical Research, Ludwig/MIT Center for Molecular Oncology, and MIT Department of Biology, Cambridge, MA 02142, USA
*Correspondence: dh@epfl.ch (D.H.), weinberg@wi.mit.edu (R.A.W.)
DOI 10.1016/j.cell.2011.02.013

The hallmarks of cancer comprise six biological capabilities acquired during the multistep development of human tumors. The hallmarks constitute an organizing principle for rationalizing the complexities of neoplastic disease. They include sustaining proliferative signaling, evading growth suppressors, resisting cell death, enabling replicative immortality, inducing angiogenesis, and activating invasion and metastasis. Underlying these hallmarks are genome instability, which generates the genetic diversity that expedites their acquisition, and inflammation, which fosters multiple hallmark functions. Conceptual progress in the last decade has added two emerging hallmarks of potential generality to this list—reprogramming of energy metabolism and evading immune destruction. In addition to cancer cells, tumors exhibit another dimension of complexity: they contain a repertoire of recruited, ostensibly normal cells that contribute to the acquisition of hallmark traits by creating the "tumor microenvironment." Recognition of the widespread applicability of these concepts will increasingly affect the development of new means to treat human cancer.

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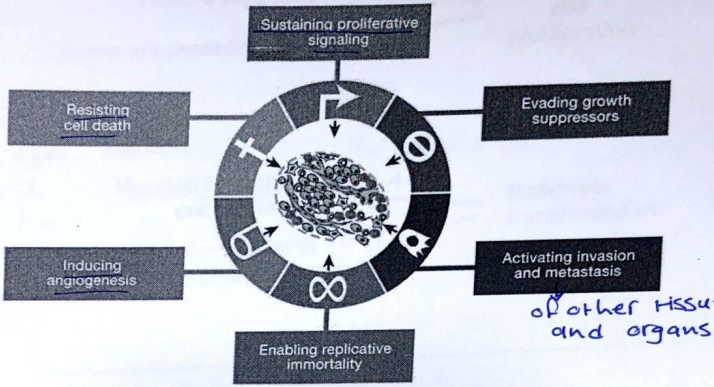
# Functional capabilities acquired by cancer cells




of other tissues and organs

* كل هاي الـ Factors بتساعد بالـ Survival of the Cancer Cells.

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* لما بصير metastasis و invasivness و unprogramed cell proliferation و dedifferentiation كلّه بصير بسبب abnormalities بالكروموسومات داخل الـ cancer cell، أو بتكون الـ abnormalities على مستوى الجين أو على مستوى الـ resistance to cell death . حيث بالـ normal conditions عنّا الـ apoptosis إلي هوّه الـ programed cell death حيث لما تكون الخليّة فيها خلل بتفعل الـ cell death enhancment لحتى تموتها ، في عنّا oncogene معين بعمل لـ apoptosis ، لكن الـ cancer cell بتكون عندها مقاومة فبكون عندها إنقسام كتير وبنفس الوقت بتقاوم الـ death .

* في كمان suppresor gene. إذا الـ cancer cell بتكون محاجة للغذاء فراح بصير عندها angiogenesis إلي هوه تكوّن new blood vessels بالتالي بتصير عندها its own supplement من الأكسجين والغذاء.

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* Colon Cancer, breast Cancer, Prostate Cancer, they have genetic factors
* Enviromental factors Such as Lung Cancer which is related to Smoking, abestoses

# Causes

- A normal cell turns into a cancer cell because of one or more mutation in its DNA, which can be inherited or acquired through exposure to viruses or *carcinogens* (e.g. tobacco products, asbestos)

*ممكن تكون genetic أو لأ و الي عندهم بالعيلة family history في السرطان لازم يفحصو يعملو checkups

- 2 main categories of cancer genes:

على مستوى الجين

**Oncogenes** (Genes which normally function to PROMOTE cell growth/division in a controlled manner)

**Tumour suppressor genes** (These genes normally function to PREVENT cell growth/division)

*لازم هدول الـ genes يكونو في بينهم balance فلو كان في خلل بخليه لازم الـ Tumour suppresor gene يتفعل و يثبط الـ cell growth

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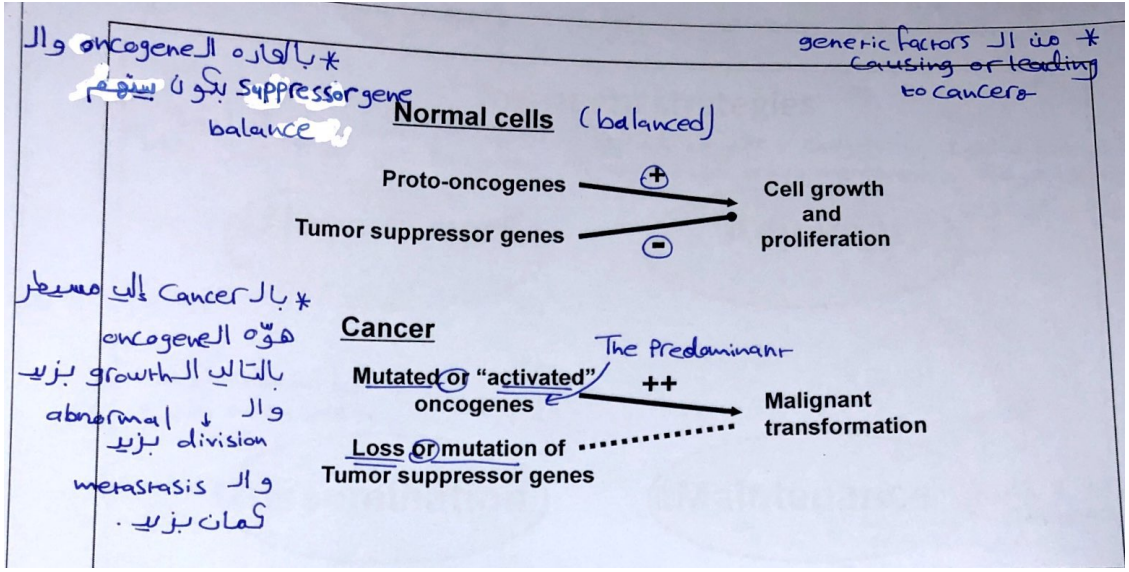


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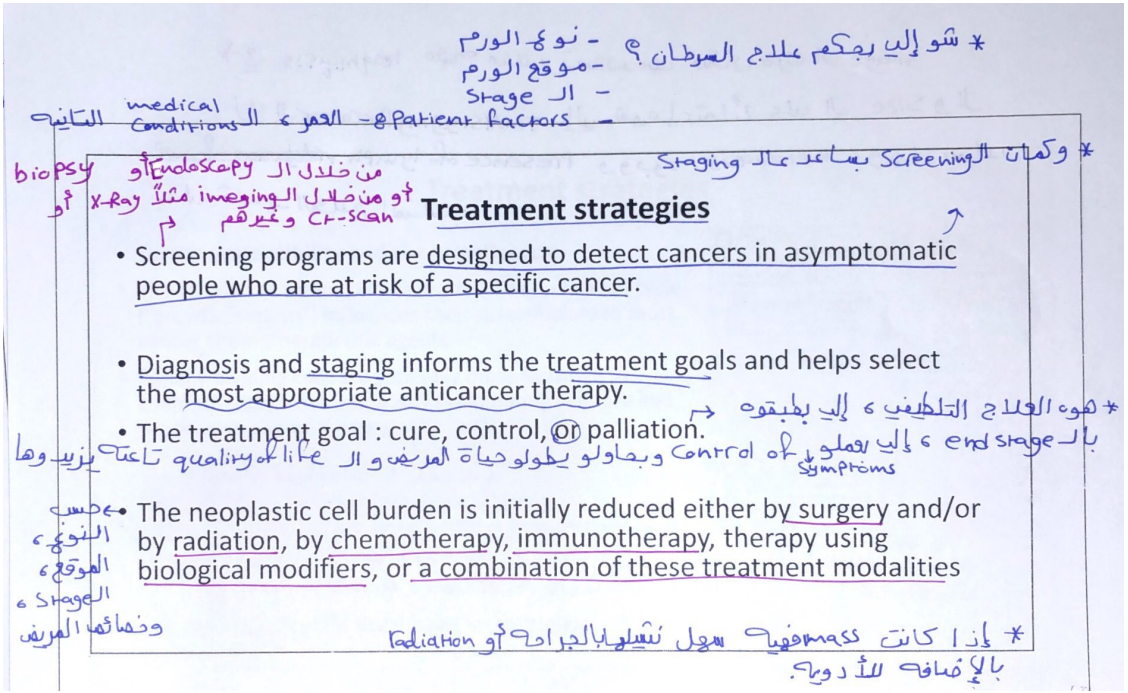


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* في techniques معينة منقدر نستخدمها عشان نعرف الـ Stage
زي الـ TNM Staging for Cancer اللي هوه إعتماداً على الـ Size والـ
Presence of lymph nodes ووجود metastasis وهاد بساعد
على تحديد العلاج المناسب

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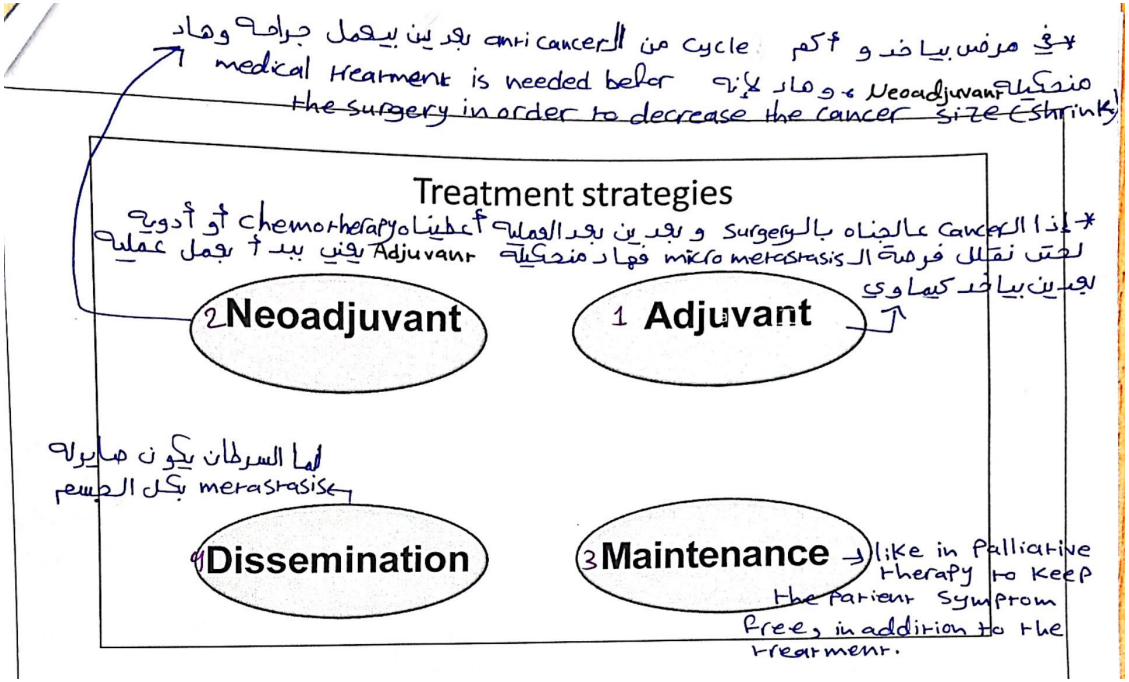


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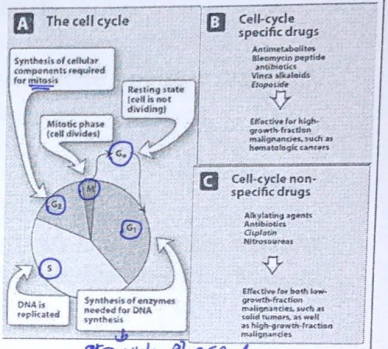
# Treatment strategies

- **Tumor susceptibility and the growth cycle**
- The fraction of tumor cells that are in the replicative cycle ("growth fraction") influences their susceptibility to most cancer chemotherapeutic agents.
- Rapidly dividing cells are generally more sensitive to chemotherapy, whereas slowly proliferating cells are less sensitive to chemotherapy.




* منصنف الـ Cancer medication لـ 2

- Agents falling into these two major classes:

a. **Cell-cycle specific drugs:** are effective only against replicating cells (that is, those cells that are cycling). → * هدول بأثروا على highly replicating cells و منفضلها الـ cell cycle محدد بال stage

Growth Phase 1 بتحضر حالها لحتى تدخل بال S.phase

b. **Cell-cycle non-specific drugs:** used for replicating and non-replicating cells

↳ it affects any stage.

* بالغالب الـ cell-cycle specific drugs ما منعطيهم لحالهم و منعطيهم بـ Combinations لأنه هو بيكون بستهدف stage معين لكن الخلية ممكن إنها تكون موجودة بالـ stage إلي بعده.

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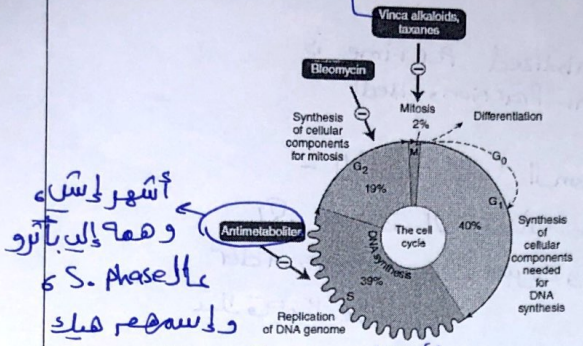
# Cell cycle effects of major classes of anti-cancer drugs.

neuropathy ويعمل highly neuro toxic هذول هم




أشهر إشي وهم إلي بأثرو على S. Phase وإسمهم هيك

لأنها structural تشبه الـ Purine and Pyridine bases بتشتغل كـ competitive inhibitors

- DNA replication بوقف destructing
- DNA synthesis بوقف stoping

| Cell Cycle-Specific (CCS) Agents | Cell Cycle-Nonspecific (CCNS) Agents |
|---|---|
| **Antimetabolites (S phase)** | **Alkylating agents** |
| Capecitabine | Altretamine |
| Cladribine | Bendamustine |
| Clofarabine | Busulfan |
| Cytarabine (ara-C) | Carmustine |
| Fludarabine | Chlorambucil |
| 5-Fluorouracil (5-FU) | Cyclophosphamide |
| Gemcitabine | Dacarbazine |
| 6-Mercaptopurine (6-MP) | Lomustine |
| Methotrexate (MTX) | Mechlorethamine |
| Nelarabine | Melphalan |
| Pralatrexate | Temozolomide |
| 6-Thioguanine (6-TG) | Thiotepa |
| **Topoisomerase II inhibitor ($G_2$–S phase)** | **Antitumor antibiotics** |
| Etoposide | Dactinomycin |
| **Topoisomerase I inhibitors (Camptothecins, $G_2$–M)** | Mitomycin |
| Irinotecan | **Platinum analogs** |
| Topotecan | Carboplatin |
| **Taxanes (M phase)** | Cisplatin |
| Albumin-bound paclitaxel | Oxaliplatin |
| Cabazitaxel | **Anthracyclines** |
| Docetaxel | Daunorubicin |
| Paclitaxel | Doxorubicin |
| **Vinca alkaloids (M phase)** | Epirubicin |
| Vinblastine | Idarubicin |
| Vincristine | Mitoxantrone |
| Vinorelbine | |
| **Antimicrotubule inhibitor (M phase)** | |
| Ixabepilone | |
| Eribulin | |
| **Antitumor antibiotics ($G_2$–M phase)** | |
| Bleomycin | |

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* ال action تأخذ شكل
under 1st order kinetics

## The log cell kill hypothesis

* يعني (Killed) بتتغير بعدد ال Cancer cells من number و amount مع Constant Proportion
مع الوقت

- **Cytotoxic drugs act with first-order kinetics**: a given dose killing a constant ***proportion*** of cells, not a constant *number* of cells.

- Cell kill is, therefore, proportional, regardless of tumor burden
- A drug with 3 log killing of cancer cells would reduces the tumour burden from $10^{10}$ to $10^{7}$ cells. The same dose used at a tumor burden of $10^{5}$ cells reduces the tumor mass to $10^{2}$ cells

* $\text{Log}10^{3}$ is Killed per unit time

* يعني كان عندي ال Cancer cells بالبداية $10^{10}$ ومع أول cycle صار $10^{3}$ kill فـ Proportion
Constant Proportion of cells are killed per unit time
بتصير $10^{7}$ و بال second cycle بتصير $10^{4}$ فهيك بصير

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* First and zero order Kinetics :-

- Constant amount of drug metabolized per time is for zero order kinetics (constant fraction, fixed)

- الأدوية كلها تاكها يكون metabolism first order kinetics
لكن مع بعض الإستثناءات حيث في بعض الأدوية بال high doses بيروح
ما بيكون إلا zero order زي الأسبرين والكحول بالتالي ال metabolism
علاقة بال dose

~~* The rate of zero~~

* The rate of zero order kinetics does not depend on the conc. of reactants.

* The rate of 1st order depends on the conc. of one reactant.

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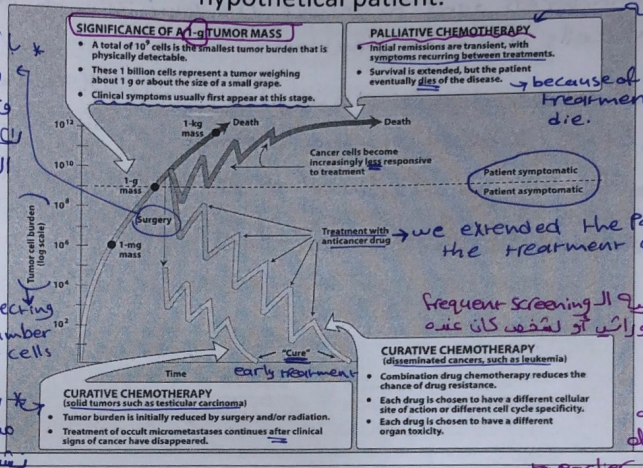
* الـ anticancers بينقطو as cycles وهاد مهم لحتى to decrease the chance of resistance against the anticancers and to enhance the remission of the normal cells and their functions.

حيث إنو الـ normal cells بتأثر من هاي الأدوية وهاد أحد أسباب تساقط الشعر والـ diarrhea وغيرهم زي الـ infertility ، حيث إنهم بأثروا على highly dividing cells وهو الـ cancer فقط . مشان هيك منقطيهم بـ cycles لحتى نعطي وقت للـ normal cells إنهم يرجعو لوضعهم

# Effects of various treatments on the cancer cell burden in a hypothetical patient.




* بالإعتماد على الـ Cancer size و لما تكون solid mass و فنقدر نتعالجها كعملية لكن إذا ما تعالج راح الـ size يكبر ويلش يصير بالـ end stage و يصير death

reflecting the number of cells

* بعد الـ Surgery كمان منعطيه علاج لحتى نقتل micrometastasis و نعمل Complete cure

because of late diagnosis & treatment the patient will die.

we extended the patients life with the treatment cycles

frequent screening

* من الاشغلات المهمة في الطب إذا كان في عامل وراثي أو شخص كان عنده cancer و إنقال لحتى تتأكد إنه ما رجع and the earlier the diagnosis will lead to earlier treatment so better prognosis.

* إذا إكتشفناه بـ late stage فمنعمله

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— - أو Surgery of Chemotherapy يعني

* Type of treatment is based on the Choice of modalities
  - The type of cancer
  - The patient condition

## Principles of cancer chemotherapy

- Chemotherapy dosing may be based on **body weight** ①, body surface area **(BSA)** ②. BSA is commonly used as an estimate of cardiac output and subsequent distribution to the liver and kidneys, the primary determinants of drug elimination.

(بالمختصر بأيش رح نعتمد)

- **Individual patient characteristics determine the choice of modalities**

- **Not all patients can tolerate drugs**, and **not all drug regimens are appropriate for a given patient**

* يعني diff. patients عندهم نفس الـ cancer بس عالجناهم بـ diff. regimens وهاد بسبب factors مختلفة.

- **Renal and hepatic function, bone marrow reserve, and concurrent medical problems** all come into consideration in making a therapeutic plan

* وزي ما حكينا الـ normal highly dividing cells كمان بتأثر بالعلاج

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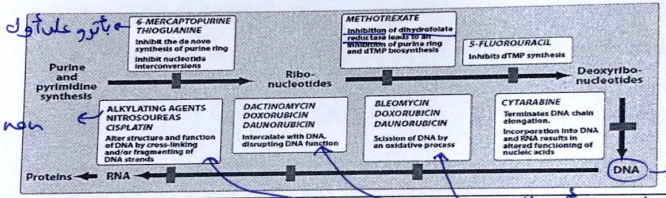
* ال anticancer dose منحسبوها ممكن بالإعتماد عالـ Body weight
و الأكثر دقة هيه بالأعتماد على ال BSA ، ال BSA كمان بساعد
الـ كتور يعمل estimation لـ Cardiac output و الـ distribution
لـ kidney and liver بالتالي ممكن يدل على drug elimination ،
لكن الـ renal function و الـ liver screening لازم ينعملو قبل بدء العلاج
بأي anticancer drug B. صحة الـ renal function test

* تأثير الدوا على highly dividing normal & cancer cells كانت أوضح بالسابق لما كانت chemotherapy هي الـ old medications زي الـ cysplatin.

## PRINCIPLES OF CANCER CHEMOTHERAPY

- Cause a lethal cytotoxic event or apoptosis in the cancer.
- Generally directed toward DNA or against metabolic sites essential to cell replication...for example, of purines and pyrimidines.
- Ideally, these anticancer drugs should interfere only with cellular processes that are unique to malignant cells.
- Unfortunately, most anticancer drugs do not specifically recognize neoplastic cells but, rather, affect both normal and abnormal cells.




So this will lead to many advers reactions.

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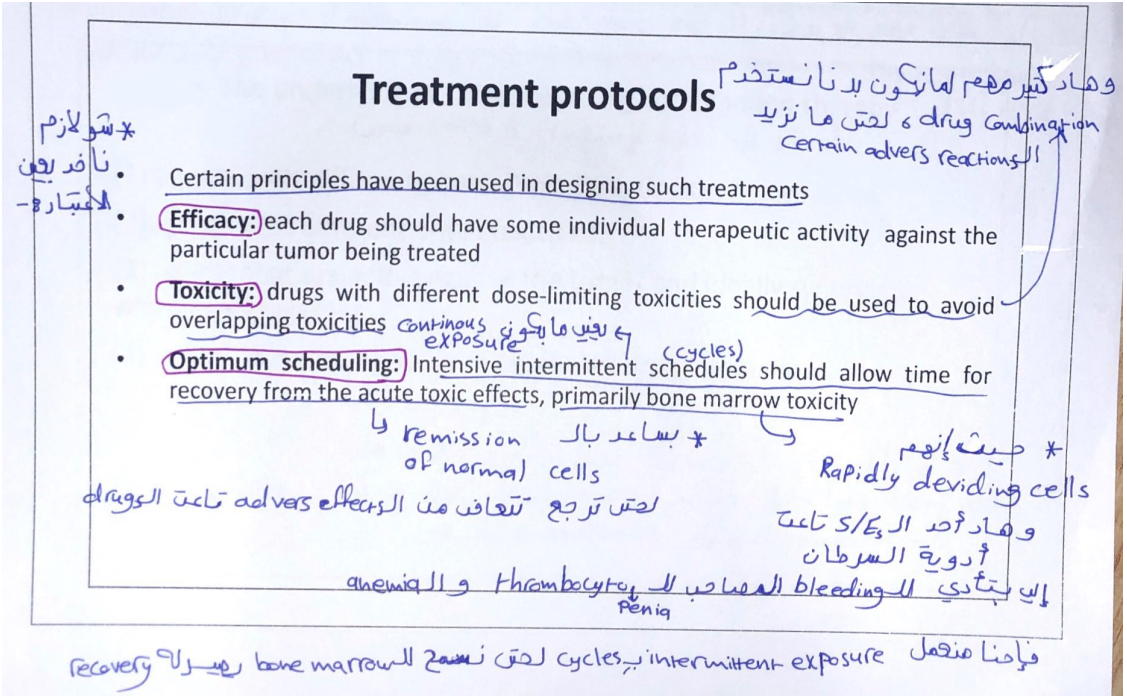
# Treatment protocols

- Certain principles have been used in designing such treatments
- **Efficacy:** each drug should have some individual therapeutic activity against the particular tumor being treated
- **Toxicity:** drugs with different dose-limiting toxicities should be used to avoid overlapping toxicities
- **Optimum scheduling:** Intensive intermittent schedules should allow time for recovery from the acute toxic effects, primarily bone marrow toxicity



* من الRecamindation إنه نستخدم drug Combination

## Treatment protocols

- Combination drug chemotherapy is more successful than single-drug treatment in most of the cancers for which chemotherapy is effective.

* benifits تزيد الeffect تاعتك

1) provide maximal cell killing within the range of tolerated toxicity
2) are effective against a broader range of cell lines in the heterogeneous tumor population.
3) may delay or prevent the development of resistant cell lines.

* يعني الkilling effect راح يزيد ، لكن toxic effect وكمان يفضل well tolarated

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* كل دوا لازم يكون active ضد هاد الـ cancer ولازم يكون إلهم diff M.O.A
بالتالي لما نستخدم مع بعض راح يصير potentiate their action of maximization in their effect
و الـ ~~cross resistance between these medications~~ لازم يكون minimal و يكون عند هم diff toxic effect

The underlying principles of using combination therapy
أو شو الـ adv. لإستخدام الـ Combinations :-

(1) agents with different pharmacologic actions

(2)agents with different organ toxicities.

(3) agents that are active against the tumor and ideally synergistic when used together.

(4) agents that do not result in significant drug interactions

* لما ناخد هاي الـ factors بعين الإعتبار راح نحصل على الـ whole benifits و الـ benifits اللي منحصل عليها من إستخدام الـ drug combination صح بالسلايد إلي قبل .

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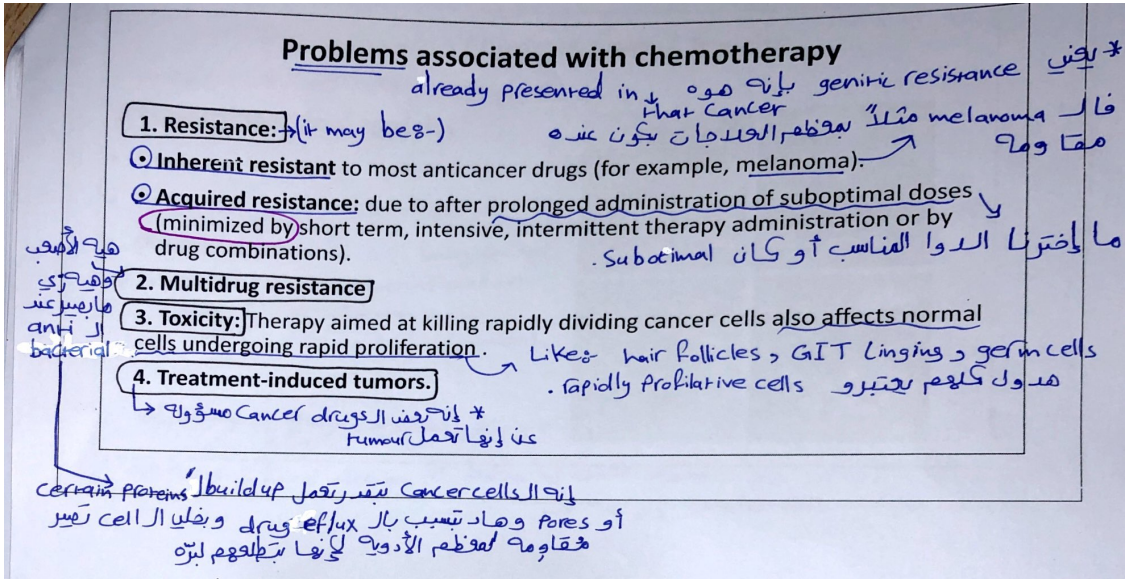

## Problems associated with chemotherapy

1. **Resistance:** (it may be a-)
   - **Inherent resistant** to most anticancer drugs (for example, melanoma).
   - **Acquired resistance:** due to after prolonged administration of suboptimal doses (minimized by short term, intensive, intermittent therapy administration or by drug combinations).
2. **Multidrug resistance**
3. **Toxicity:** Therapy aimed at killing rapidly dividing cancer cells also affects normal cells undergoing rapid proliferation.
4. **Treatment-induced tumors.**

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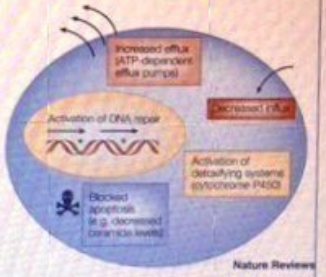
## Mechanisms of resistance

- ***Increased DNA repair***—An increased rate of DNA repair in tumor cells can be responsible for resistance particularly important for alkylating agents and cisplatin.
- ***Changes in target enzymes***—Changes in the drug sensitivity of a target enzyme, dihydrofolate reductase, is a mechanism of resistance of tumor cells to methotrexate.
- ***Decreased activation of prodrugs***—Resistance to the purine antimetabolites and pyrimidine antimetabolites
- ***Inactivation of anticancer drugs***—Increased activity of enzymes capable of inactivating anticancer drugs.

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mechanism of resistance

① ال alkylating agents هو ال DNA حيث بعمل target ال DNA بتكون عنده distruction in DNA synthesis و وفي بعض الأحيان ال increased repair ability و Cancer resistance بالتالي هاد بزيد ال Combination فبهاي الحالة فنعطي لدوا بشتغل على Phase تانية من ال cycle، يعني عند different MOA فهاد راح يقلل ال drug resistance وبتزيد ال efficacy to kill cancer cells.

② أحيانًا الدوية drugs بتكون ال target تبعها هوه إنزيمات زي ال DHFA enzyme اللي هوه target ال methotrexate drug، لكن لما يصير Changes in drug sensitivity على ال target enzyme يعني ببطل الإنزيم يستجيب للدوا فهون بصير resistance

③ معظم الـ drugs بكونو Prodrugs بحتاجو للتفعيل فأحيانًا بتصير resistance عن طريق decreasing the prodrug activation

④ في إنزيمات معينة بتزيد ال destruction أو بتزيد ال metabolism اللي بأدي لزيادة تكوين ال inactive metabolites، بالتالي بتخلي ال drugs غير فعّالة

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* ال anticancers بتختلف بال SIEs تانها بتقوم بدرجتها

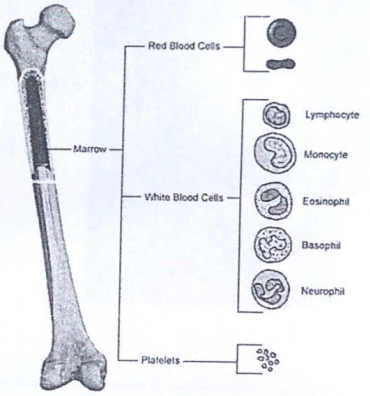
هوه مسؤول عن تصنيع ال RBCs وال WBCs و ال Platelets

→ because of the effect on highly proliferative cells

## Problems with Chemotherapy/ Toxicity

➢ **Bone marrow toxicity: Myelosuppression**

- **Anemia: decrease red blood cells**
- **Neutropenia: decrease in neutrophils – main defence against bacterial infection**
- **Thrombocytopenia: decrease in platelets**
- **Pancytopenia: decrease in red and white blood cells plus platelets**

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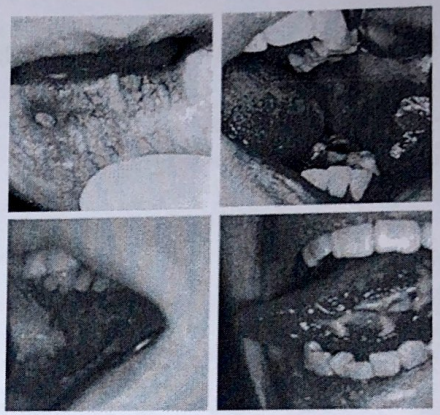
# Problems with Chemotherapy/ Toxicity

epithelial cells are rapidly deviding cells

## ➢ Gastrointestinal toxicity

1. Nausea and vomiting
2. Mucositosis. → inflammation in the GI Lining.
3. Diarrhea → because of GI lining damage.
- Stomatitis → inflammation / ulceration or infection in the oral cavity. (Common)



oropharyngial ↓ infection // highly susceptabil بتصير و ✱

و opportunistic fungal infection و bacterial infection سواء كانت

Common إلي هيه كتير candidiasis

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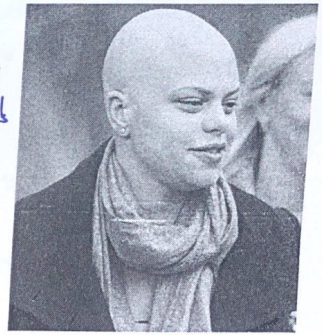
## Problems with Chemotherapy/ Toxicity

- **Hair toxicities**
  - Alopecia: common side effect and the most visual
  - Phsycologically distressing
  - Cytotoxic drugs (particularly doxorubicin and etoposide, taxanes) target the dividing hair follicle



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## Problems with Chemotherapy/ Toxicity

- Effect on germ cells → sterility, teratogenicity and mutagenic
- Hyperuricemia : cell break down → purine → uric acid → gout and renal damage

*uric acid accumulation is also called tumur lysis syndrom

* بسبب الـ break down للخلايا فبتأدي لخروج كميات كبيره من الـ purine و الـ purine بيصير metabolism لـ uric acid وبتأدي الـ Hyperuricemia واللي بتراكم وبعمل gout و renal dameg.

* مشان هيك بالعلاج بالغالب بضيفو allopurinol عالـ regimen حيث هوه إله anticancer effect و بنفس الوقت بعمل inhibition لتكوين الـ uric acid عن طريق تثبيط الـ xanthine oxidase فبتقل فرصة تراكم الـ uric acid.

منقلل من التراكم عن طريقة زيادة الـ fluid intake خاصة" لما بصير renal dameg وإنه ناخد allopurinol.

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Specific adverse effects:-

- **Kidney toxicity**

Cisplatin – renal toxicity is dose limiting

In early studies, 75% of patients experienced renal toxicity

- **Cardiotoxicity**

لما تصير منقلك ال dose

Daunorubicin and doxorubicin – cardiotoxicity is dose limiting/ Caused by free radical generation

- **Neurotoxicity**

Distal neuropathy (peripheral neuropathy) can occur with vincristine

- **Carcinogenicity**

Many drugs damage DNA of normal cells – can cause mutations Secondary malignancies may occur

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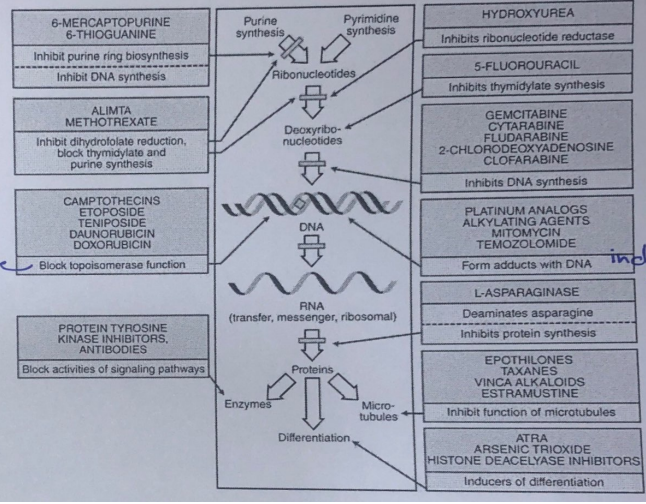
إلي مابنذكرو بال
anticancer Part 2
مو مطلوبين

## Summary of cytotoxic drug action




So inhibits the DNA re replication

induce DNA dameg

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## Classification of Anticancer Drugs

- 1. Antimetabolites.
- 2. Alkylating agents.
- 3. Antibiotics.
- 4. Mytotic spindle poisons. Like taxanes
- 5. Hormones.
- 6. Others.

↳ That have higher efficacy because they are targeted treatment so they have minimal systemic adverse reactions.

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