Hoffmann & Krueger

Delivering Oncology Excellence

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We, at Hoffmann- Krueger do not turn complex issues into simple solutions

We make them understandable

Corporate overview – Who we are

- Hoffmann & Krueger is a company specialized in oncology
- Provides web-enabled insights and predictive analytics via proprietary databases, high-value analytical reports and tools

  • Enables customers to make fully-informed key investment and cost containment decisions in each phase of the business, science, politics, lifecycle

- Proprietary content and data streams created in-house through a rigorous, highly-analytical, knowledge-driven process

  • Staff of highly credentialed and experienced professionals: PhDs, MDs, MPHs, Pharmacists, MBAs and key industry experts. People with talent, drive and commitment who can achieve wonders, while rising above market medians

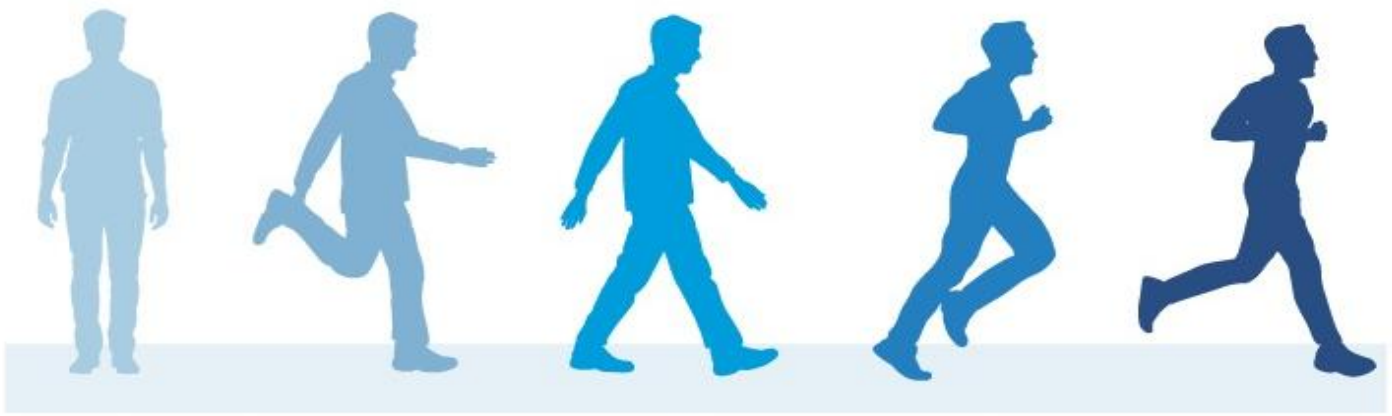
- Global scale and reach with offices in Geneva, and the Emirates

Support your business transformation

- Hoffmann & Krueger adopts a customized, responsive and personal approach towards client servicing and offers a complete range of services across oncology field

R E A C T

- We are delivering measurable value to clients through a global network of diverse professionals who bring unmatched depth and breadth of expertise



Rethink
The patient Connection

Evaluate
Your current Tactics

Accelerate
The Growth

Change
Your Model

Transform
Your Strategy

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Oncology generalities

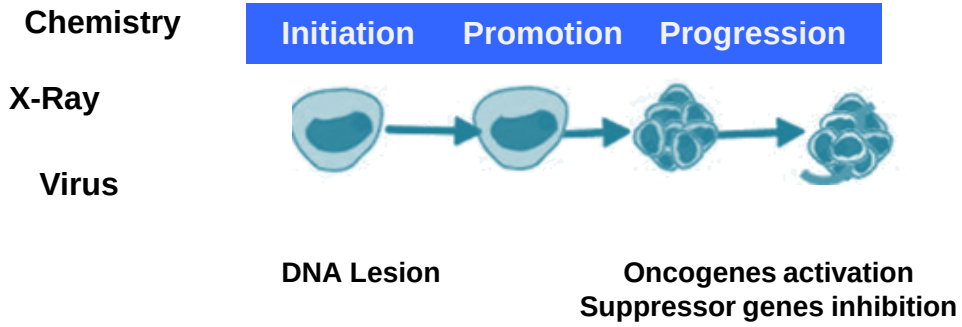
Initial steps of cancerogenesis

Three different steps may be schematically described during cancerogenesis, the first two are only known through experimental models and epidemiological studies of human cancers:

– **Initiation** is a rapid and irreversible DNA lesion which occurs after exposure to a carcinogen (physical carcinogen, chemical carcinogen, viral carcinogen)

–**Promotion** is due to prolonged, repetitive or continuous exposure to substances which maintain and stabilise the initiated lesion

–**Progression** is the acquisition of non controlled multiplication properties, independence acquisition, loss of differentiation, local invasion and metastasis



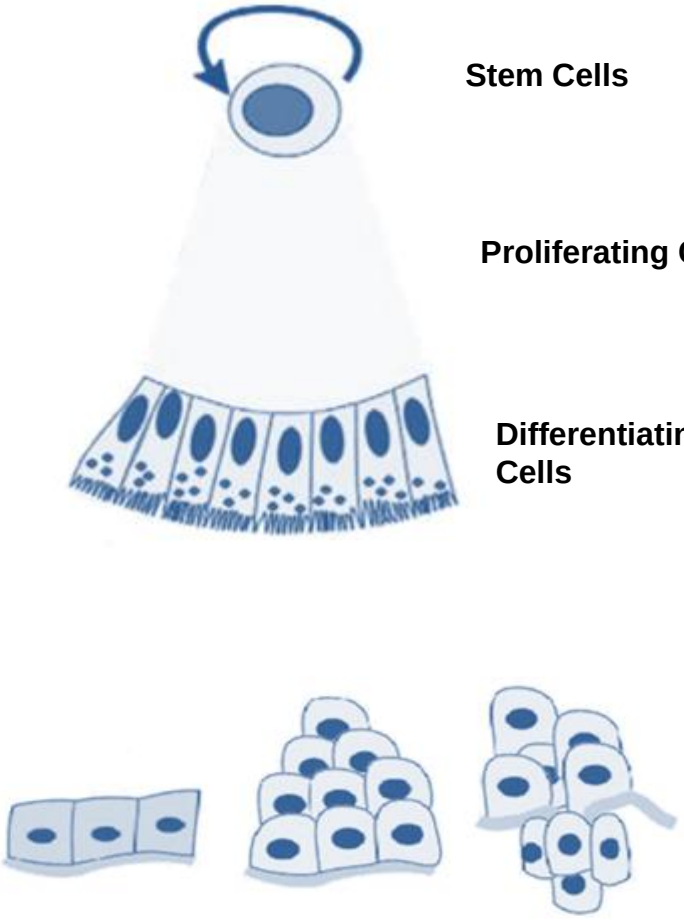
Cellular division and invasion

• **Cellular division:** local tumour development necessitates a great number of cellular divisions. The initial cancerous cells are stem cells, which divide giving birth to two daughter cells: some are identical to their mother cells, others will differentiate (differentiation). Many daughter cells will die without offspring

•**Invasion** : One of the major characteristics of cancer is the capacity of cancer cells to progressively invade neighbouring tissue. The breach and crossing of the basal membrane of the epithelium constitute the formal criteria to distinguish invasive cancers from in situ cancers.

Tumour invasion preferentially follows less resistant zones: organ capsules, nerve sheaths and small vessels.

Usually, resistant tissues are: cartilage, arteries, nerves, tendons and aponevroses



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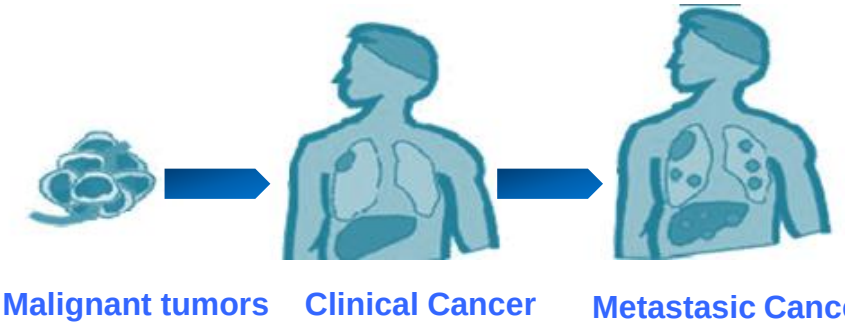
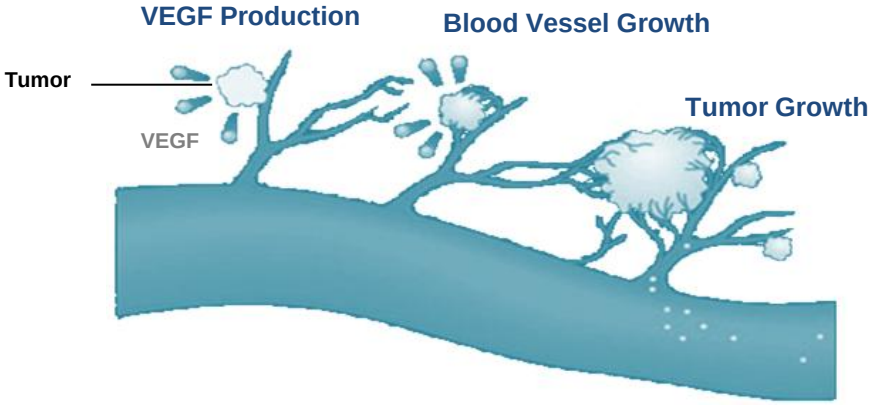
Cancer progression

- **Tumour :** this is an abnormal growth of tissue which can be either benign or malignant
- **Metastasis :** this is the spread of cancer from its primary site to other places in the body

Tumour development and metastases

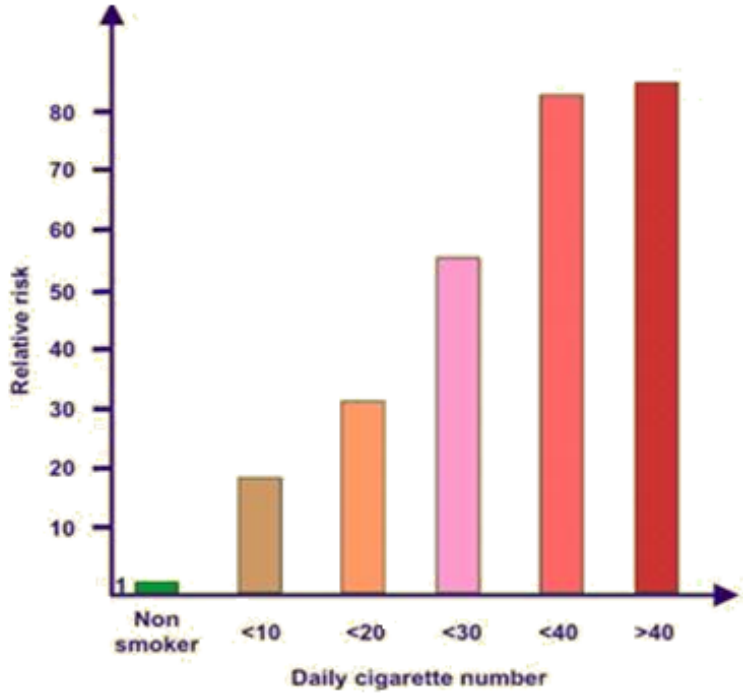


- **Angiogenesis :** cancerous tumour has to stimulate the creation of new blood vessels



Main causes of cancers

- **Chemical factors :**
  - **Tobacco:** Tobacco is nowadays the most important cause of cancer
  - **Alcohol:** consumption increases the effect of tobacco
  - **Products from industry:** aromatic amines, benzene, arsenic, chrome and wood for instance
- **Physical factors :** X-Rays, ultraviolet rays, nuclear radiations
- **Genetics predispositions**
- **Age :** important risk factor for cancer
- **Viral factors :** Human papilloma virus (HPV), Hepatitis B and C viruses, Human T-cell leukaemia virus, Epstein-Barr virus (EBV), Human immunodeficiency virus
- **Environmental factors :** pollution, feeding, drugs and pesticides



Cancer screening

The goal of cancer screening is to detect asymptomatic cancers by using diagnostic tests or methods which can be proposed to a great number of healthy person

- Cancers for screening should:
  - be frequent with heavy mortality
  - remain over a long period at a pre-clinical stage (without symptoms)
  - be detected at a stage when therapy is efficient
  - be detected using a high sensitivity and high specificity test, of a moderate cost and limiting inconvenience to the healthy person in order to be regularly repeated

- Cancers which can be screened are: breast, cervix uterin, skin, colon and rectum and prostate

Benefits	Negative aspects
• Improved prognosis for patients for whom cancer has been detected through screening • Reduction in aggressive treatment necessary to treat this screened cancer • Peace of mind for subjects with a negative test • Reduction of the general cost of cancer treatment • Reduction in mortality via the screening policy	• Discomfort brought by screening tests • Psychological and economical consequences of false positive results (more complex examinations which take place to finally reveal absence of pathology): precise description of morbidity (and sometimes mortality) induced for no reason • The more tragic consequences of false negative

Cancer diagnosis

Each cancer location has specific revealing symptoms

- **Once these symptoms have appeared**, physicians should request a few diagnostic procedures in order to:
  - **make diagnosis as soon as possible**, with the least disturbing and painful procedures for the patient
  - **quickly obtain tumour biopsy** offering precise histological knowledge
  - **request the necessary clinical and paraclinical check-ups** looking for local and remote cancer evolution
  - **precisely define** the cancer stage
  - **initiate the treatment schedule**, decided according to previously defined therapeutic protocols adjusted through a multidisciplinary study of the patient's medical record

- **Diagnostic procedures are** :
  - **Clinical examination** (letting the patient explain with his own words his disease; clinical questioning should be precise, well-mannered, discreet and empathetic)
  - **Radiological imaging** (X-Rays, mammography, computer tomography and RMI)
  - **Ultrasound imaging**
  - **Isotopic imaging**
  - **Endoscopic imaging**
  - **Diagnostic biology**
  - **Anatomopathological diagnosis** (cancer diagnosis always requires a biopsy and a pathological examination, except during terminal phases when therapy is not feasible)

Histological classification

- **Solid tumours :**
  - **Epithelioma :**

They originate from the epithelium and represent approximately 90% of all cancer types

- *malpighian or epidermoid* tumours : skin, oesophagus, head and neck epithelium, cervix uteri and lung
- *Adenocarcinoma :* breast, prostate, colon (most often mucinous), stomach, thyroid and lung
- *excreto-urinary cancer or transitional carcinoma :* excretory urinary epithelium (ureter, bladder, uretra)

- **Sarcoma :** They originate from mesenchymal structures: fibrosarcoma, liposarcoma, leiomyosarcoma, rhabdomyosarcoma, osteosarcoma, chondrosarcoma and synoviolosarcoma
- **Neuroectoblastic tumours :** They originate from central nervous system: glioma, ependymoma, or from other neurological systems: meninges (meningioma), nervous nodes (synmpathoblastoma), Schwann sheath (schwannoma), melanogenic system (melanoma) or diffuse endocrine system (apudoma).
- **Embryonic tumours :** Dysembryomas can be more or less mature: neuroblastoma, nephroblastoma, choriocarcinoma (placental, testicular, ovarian or extragonadal)

- **Hematopoiesis tumour :**

They are classified according to the cell type from which they originate: **leukaemia, lymphoma and myeloma** *(see definitions in appendix)*

Malignant and benign tumours

The following table roughly distinguishes malignant from benign tumours :

Benign tumour	Malignant tumour
Differentiated	Well to poorly differentiated
Rare mitoses	Frequent mitoses
Slow growth	Generally fast growing
No local invasion	Local invasion of neighbouring tissues
No destruction of normal tissue	Destruction of normal structures
Surrounded by a capsule	No clear limits
No relapse after complete exeresis	Quick local relapse if no complete exeresis
No node metastasis	Node metastases
No remote metastasis	Remote metastasis
No or little influence on the host	Death of the host

Classification

- **Classification according to stages :**

Stage	Description
Stage 0	In situ (non invasive) cancer
Stage 1	Very localised tumour with no remote metastases
Stage 2	Locally limited extension with/without minimal node satellite extension and with no remote metastases
Stage 3	Locally advanced extension with/without major node satellite extension and with no remote metastases
Stage 4	Locally advanced tumour and/or distant metastases

- **TNM classification : Tumor, Node and Metastasis**

T	Description
Tx	The primitive tumour cannot be studied
T0	There is no primitive tumour
T1	Very limited primitive tumour
T2	Larger tumour (generally more than 2 cm in diameter)
T3	Tumour with extension to adjacent connective tissue (fixed tumour)
T4	Extension to adjacent structures

N	Description
Nx	It is not possible to describe the node status
N0	The research for satellite nodes is negative
N1	Minimal invasion of regional nodes
N2	Major invasion of regional nodes
N3	Node invasion beyond regional nodes

M	Description
Mx	It is not possible to describe the metastasis status
M0	There are no remote metastases
M1	There are one or many remote metastases

Stages equivalence

TNM Stage	Primary tumour	Lymphatic nodes invasion	Metastases	Dukes classification (modified)
Stage 0	Tis	N0	M0	A
Stage I	T1 T2	N0 N0	M0 M0	A1 B1
Stage II	T3 T4	N0 N0	M0 M0	B2 B2
Stage III A B	 All T All T	 N1 N2-N3	 M0 M0	 C1/C2 C1/C2
Stage IV	All T	All N	M1	D

TNM stands for Tumor, Node, Metastasis; in the TNM system, T1 and T2 indicate a tumor at an early stage, T3 and T4 a tumor at an advanced stage. N reveals that tumor has reached the lymphatic nodes and M that cancer has spread other organs (metastases)

Classification

Grade classification : This component describes the histological grading of epithelial tumours

G	Description
Gx	No precision about histological grading
G1	Well differentiated tumour
G2	Moderately differentiated tumour
G3	Poorly differentiated or undifferentiated tumour

General status classifications :

- the performance status according to Karnofsky
- the ECOG performance status (Eastern Cooperative Oncology Group)

Cancer treatment

There is no single treatment for cancer; doctors will often combine several types of treatment for greater effect, taking into account all sorts of factors: the patient's age, history and lifestyle are very important in deciding on the best treatment

- Surgery : approximately half of curable cancer patients can be cured of their disease by surgery
- Radiotherapy : is the medical use of ionizing radiation as part of cancer treatment to control malignant cells
- Chemotherapy : The underlying principle of chemotherapy is to kill the cancer by treating them with chemicals that interfere with the process of cell division
- Targeted therapy : Type of medication which blocks the growth of cancer cells by interfering with specific targeted molecules needed for carcinogenesis and tumor growth
- Hormonotherapy : hormone systems are implicated in some types of cancer; by tinkering with the body's hormone system in the right way, doctors can stop some cancers growing and even kill them
- Immunotherapy : patients' tumours would occasionally shrink if their tumour became infected. This observation led to the idea that the body's immune system could be harnessed and made to fight cancer

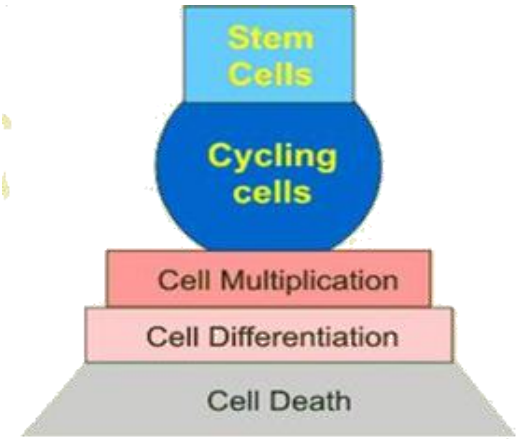
Cancer treatment

- **Treatment efficiency decreases as cancer progresses :**
  - The most efficient therapies are those applied to limited lesions: surgery and radiotherapy
  - Localised but more developed cancers can be the origin of silent metastases, which cannot be detected using standard clinical methods. In order to diminish the risk of metastasis, adjuvant treatments are proposed (chemotherapy or hormonotherapy), in complement to local treatment
  - Generalised cancers are generally not treated with a curative intention and only a palliative and temporary beneficial effect is obtained

General principles of chemotherapy

In a tumour, only the cells which have the capacity to indefinitely reproduce themselves (stem cells) are dangerous. These are the cells that we intend to kill.

The other cells, those which multiply themselves but cannot reproduce more than a few generations and those which are very well differentiated and cannot divide, will eventually die naturally without chemotherapy



Theoretical composition of a tumour from the top: stem cells which renew themselves indefinitely, then multiplying cells, then differentiating cells which result in cell death

Treatments with different objectives

Early Stages			Advanced stages	
Neoadjuvant therapy	**Induction therapy**	**Adjuvant therapy**	**Consolidation therapy**	**Palliative therapy**
Reduce the size of the primitive tumour thus making it easier to operate	Treatment designed to be used as a first step toward **shrinking the cancer** and in evaluating response to drugs. It is followed by additionnal therapy to eliminate whatever cancer remains	**Complementary treatment** prescribed after the main treatment modality	**high-dose** of chemotherapy given as the **second phase** (after induction) of a cancer treatment for leukemia	**Prolonge survival and improve patient comfort** There is no, or very little improvement on survival

Chemotherapy toxicity

Hematological toxicities	**Anaemia**	deficiency of red blood cells (RBCs) and/or hemoglobin. This results in a reduced ability of blood to transfer oxygen to the tissues
	Thrombocytopenia	presence of relatively few platelets in blood
	Neutropenia	hematological disorder characterized by an abnormally low number of neutrophil granulocytes
Acute digestive toxicities	**Acute vomiting**	intensity according to the chemotherapy drug, some being highly emetic such as cisplatin, dacarbazine or adriamycin
	Delayed vomiting	related to an other mechanism from acute vomiting and does not respond to new medicines ; generally more important if acute vomiting has been poorly controlled
	Mucitis	certain drugs are toxic for the mucosa: adriamycin, cytarabine, methotrexate. Mouth pains and dysphagia observed when mucosa abrasion occurs
	Acute diarrhoea	frequent with certain drugs such as tegafur and capecitabine
Other early toxicities	**Alopecia**	hair loss is quasi constant with products like VP 16, alkylating agents, anthracyclines, docetaxel, paclitaxel
	Acute renal insufficiency	occur particularly with the use of cisplatin or methotrexate
	Respiratory syndromes	rare complications but require urgent diagnosis
	Extravasation	fearsome complication which can induce extended and very painful skin necrosis
	Fatigue	constant component of chemotherapy

Grade examples

Adverse Event	Grade				
	0	1	2	3	4
Neuropathy-sensory	Normal	Loss of deep tendon reflexes of paresthesia (including tingling), interfering with function, but not interfering with activities of daily living	Objective sensory loss or paresthesia (including tingling), interfering with function, but not interfering with activities of daily living	Sensory loss or paresthesia interfering with activities of daily living	Permanent sensory loss that interferes with function
Emesis					
Nausea	None	Able to eat	Oral intake significantly decreased	No significant intake, requiring IV fluids	
Vomiting					
Vomiting (also consider dehydration)	None	1 episode in 24 hours over pretreatment	2-5 episodes in 24 hours over pretreatment	6 episodes in 24 hours over pretreatment; or need for IV fluids	Requiring parenteral nutrition: or physiologic consequences requiring intensive care: hemodynamic collapse

WNL = Within normal limits.

Key cytotoxic agents

Subclass	Mode of action	Brand	Generic	Company
Alkylating agents (L1A)	*Affect the process of cell division by alkylating nucleotides and preventing their assembly into DNA*	Temodar	Temozolomide	Schering-Plough
Antimetabolites (L1B)	*Inhibit the synthetis of nucleic acids*	Alimta Gemzar S-1 Xeloda	Pemetrexed Gemcitabine Tegafur, gimeracil+oteracil Capecitabine	Eli Lilly Eli Lilly Taiho/Sanofi-Aventis Roche/Chugai
Plant alkaloids and terpenoids (L1C)	*Bind specifically to tubulin, which prevents tubulin dimers from aggregating to form microtubules*	Camptosar Navelbine Taxol Taxotere	Irinotecan Vinorelbine Paclitaxel Docetaxel	Pfizer GSK BMS Sanofi-Aventis
Antineoplastic antibiotics (L1D)	*Prevent cell division, including the inhibition of topoisomerase II*	Doxil Ellence	Pegylated liposomal doxorubicin Epirubicin	Johnson & Johnson Pfizer
Platinum compounds (L1X2)	*Bind covalently to DNA and prevent the further synthetis of DNA, RNA and protein*	Eloxatin Paraplatin	Oxaliplatin Carboplatin	Sanofi-Aventis BMS

Key targeted therapies

Subclass	Mode of action	Brand	Generic	Company
Antineoplastics Monoclonal antibodies (L1X3)	*Target tumour specific antigens, thus enhance the host's immune response to tumour cells to which the agent attaches itself*	Avastin Erbitux Herceptin Rituxan	Bevacizumab Cetuximab Trastuzumab Rituximab	Genentech/Roche BMS/Merck KGaA Genentech/Roche Genentech/Roche
All other antineoplastics (L1X9)	*Signal tranduction inhibitor*	Gleevec Iressa Tarceva	Imatinib Gefitinib Erlotinib	Novartis AstraZeneca Genentech/Roche
	Apoptosis stimulators	Velcade	Bortezomib	Ortho Biotech/Millenium Pharmaceuticals

Key antihormonal therapies

Hormonal therapy is not chemotherapy. Cancer arising from certain tissues, including the mammary and prostate glands, may be inhibited or stimulated by appropriate changes in hormone balance

Subclass	Brand	Generic	Company
Luteinizing hormone-releasing hormone analog (L2A3)	Lupron Zoladex	Leuprorelin Goserelin	Abbott/Wyeth AstraZeneca
Anti-estrogens (L2B1)	Novaldex	Tamoxifen	AstraZeneca
Anti-androgens (L2B2)	Casodex	Bicalutamide	AstraZeneca
Aromatase inhibitors (L2B3)	Arimidex Femara	Anastrozole Letrozole	AstraZeneca Novartis

Appendix

Hematology definitions

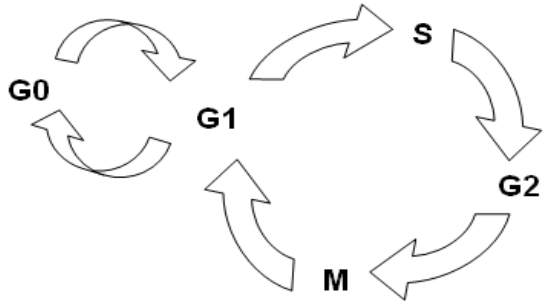
- **Hodgkin's lymphoma :** Cancer of the immune system that is marked by the presence of a type of cell called the Reed-Sternberg cell. Symptoms include the painless enlargement of lymph nodes, spleen, or other immune tissue. Other symptoms include fever, weight loss, fatigue, or night sweats.

- **Non-Hodgkin lymphoma :** (NHL) describes a group of cancers arising from lymphocytes, a type of white blood cell. Non-Hodgkin lymphoma may develop in any organ associated with the lymphatic system (spleen, lymph nodes, or tonsils)

- **Myeloma :** Type of cancer of plasma cells which are immune system cells in bone marrow that produce antibodies. Its prognosis, despite therapy, is generally poor, and treatment may involve chemotherapy and stem cell transplant

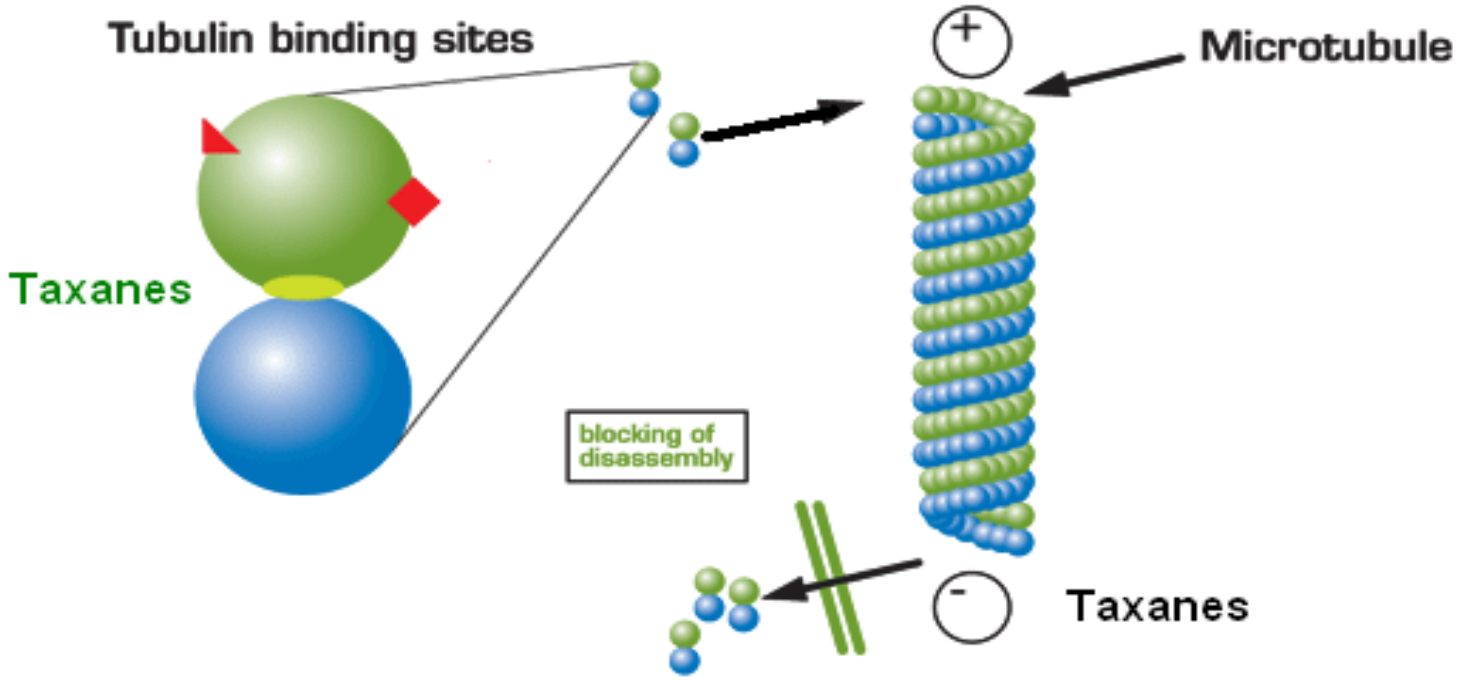
- **Leukaemia :** Cancer of the blood or bone marrow and is characterized by an abnormal proliferation of blood cells, usually white blood cells (leukocytes)

Phases of the cell cycle



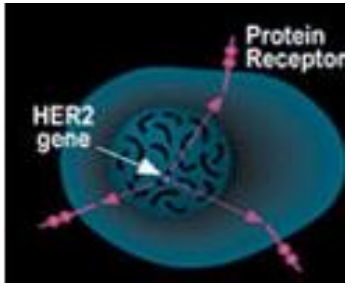
- **G0 phase** is a period in the cell cycle where cells exist in a quiescent state. Cells enter this phase from a cell cycle checkpoint in the G1 phase, such as the restriction point or the start point
- **M phase** consists of nuclear division (mitosis) and cytoplasmic division (cytokinesis). After M phase, the daughter cells each begin interphase of a new cycle. Although the various stages of interphase are not usually morphologically distinguishable, each phase of the cell cycle has a distinct set of specialized biochemical processes that prepare the cell for initiation of cell division
- **G1 phase** is the first phase within interphase; the biosynthetic activities of the cell, which had been considerably slowed down during M phase, resume at a high rate
- **S phase** starts when DNA synthesis commences; when it is complete, all of the chromosomes have been replicated
- **G2 phase** lasts until the cell enters the next round of mitosis. Metabolic activity, cell growth, and cell differentiation all occur during interphase

Mechanism of action: Taxanes (Paclitaxel, Docetaxel)

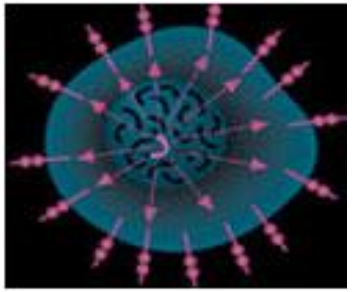


Mechanism of action: Trastuzumab

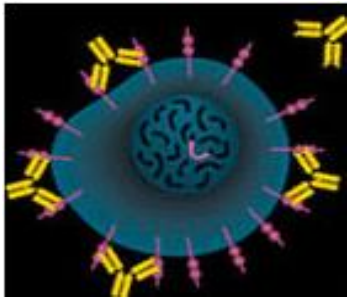
Normal Cell : in normal breast tissue cells, the HER2 gene produces a protein receptor on the cell surface. These growth factor play a role in normal cell growth by signaling the cell to divide and multiply



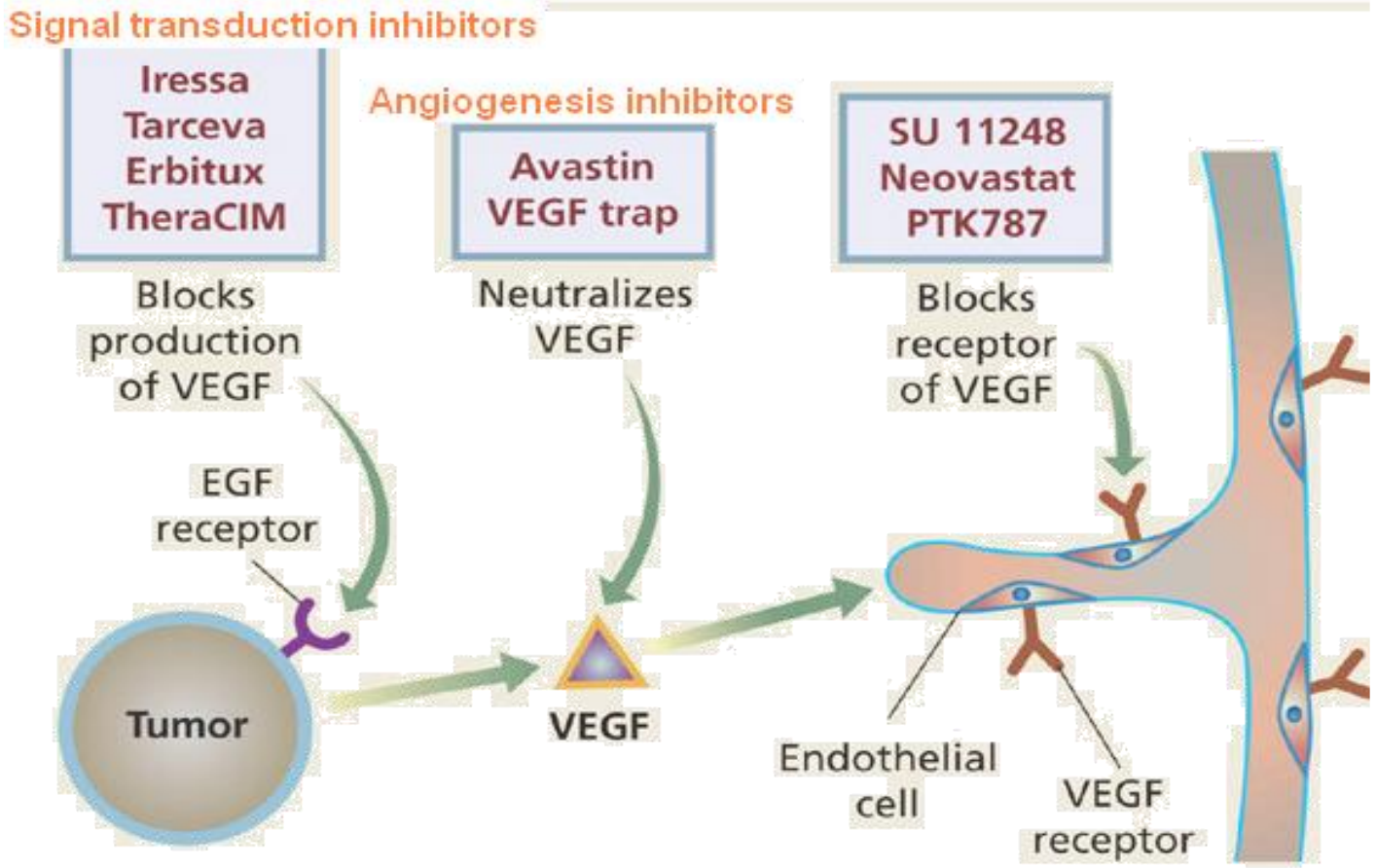
HER2 overexpressing cancer cell : cancerous breast tissue cells that overpress the HER2 gene produce extraprotein receptors on the cell surface which triggers the cell to divide and multiply at an accelerated rate thus contributing to tumour growth



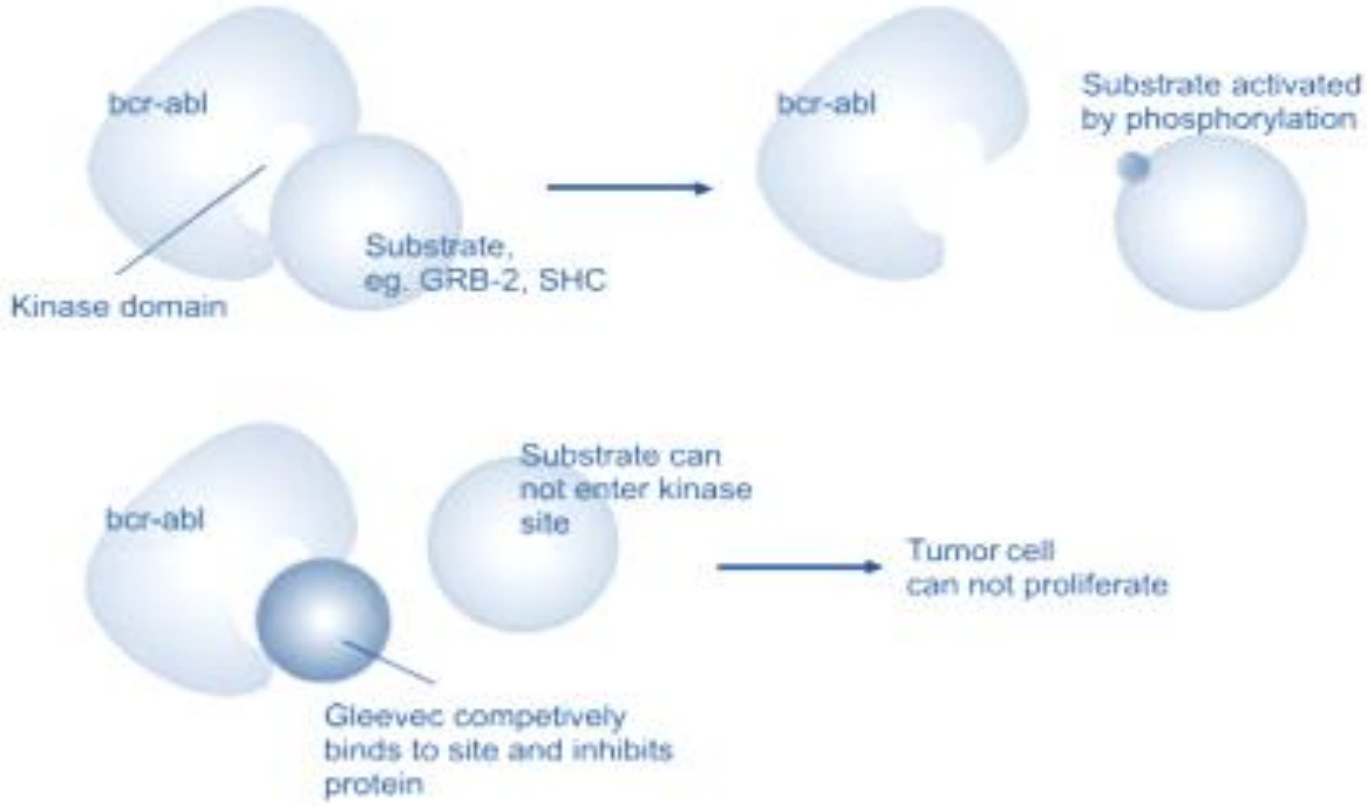
Trastuzumab binds to numerous HER2 receptor sites found on the cell surface, blocking the receptor sites and possibly preventing further growth by interrupting the growth signal. As a result, the HER2 antibody may slow progression of the disease



Angiogenesis and transduction inhibitors

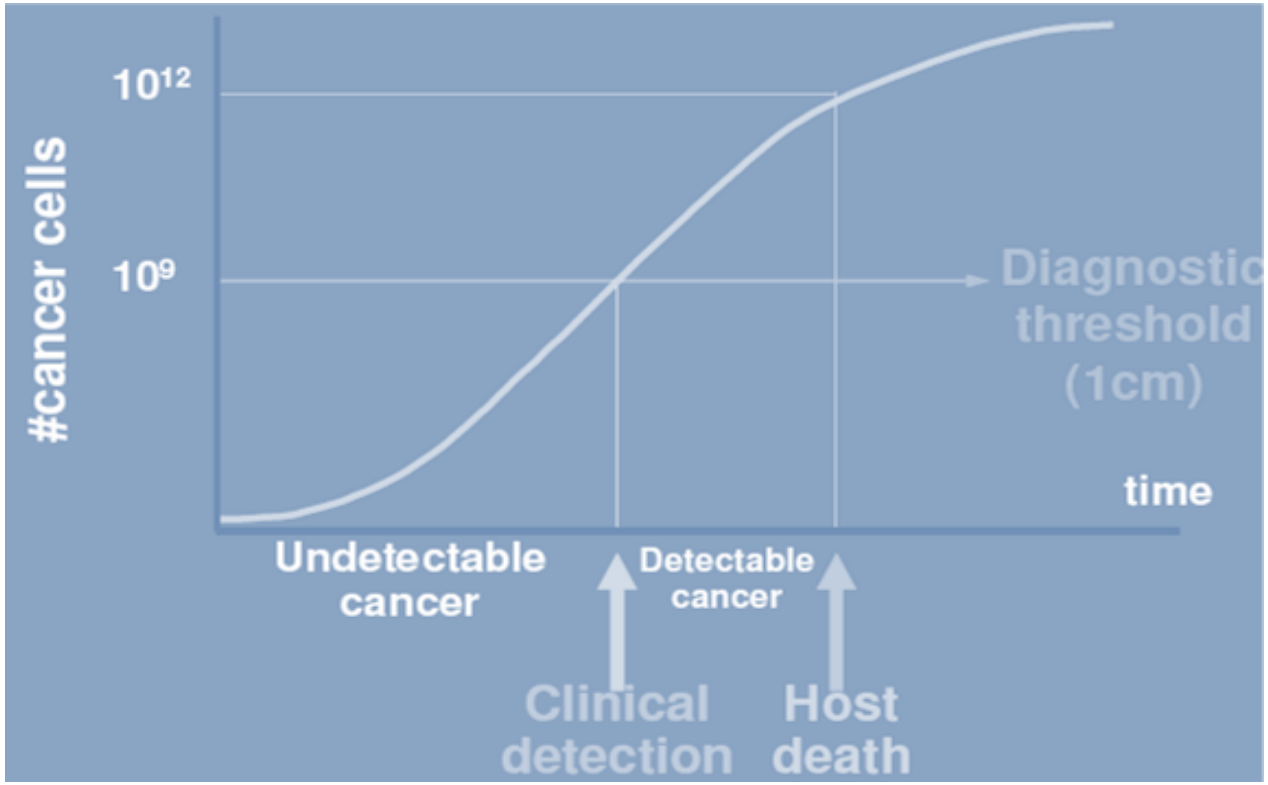


Mechanism of action: Imatinib



Gompertzian Tumor Growth

The gompertz curve represents the cell proliferation in the time and allows to learn about cancer kinetic



Clinical phases

- **Phase I study:** Their aim is to determine the useful clinical therapeutic dosage by finding the **maximal tolerated dose**. The initial dosage, the specific examinations and the potential toxicities are generally deducted from animal studies. Before beginning human studies, an initial dosage is determined.Generally, studies with other animals have been previously conducted in order to detect toxicities which do not exist in mice but may exist among other species. These toxicities are very carefully looked for during initial dosage of phase I

- **Phase II study:** The aim of phase II studies is to **evaluate the potential efficiency** of a new drug for a **particular cancer location**. They are performed with patients having exhausted all the usual therapeutic possibilities, in other words after one or two lines of standard treatment

- **Phase III study:** Phase III trials are **comparative studies**. Their aim is to show the superiority of a new treatment in comparison to a standard treatment. Whereas phase II studies look at responses, **phase III studies look at survival**

- **Phase IV study:** We refer to as phase IV the recording of treatment outside any randomisation, in order to **verify its efficiency** and study the observed toxicities on a larger scale. The main argument for such studies is that during randomised phase III trials, many patients are excluded due to **inclusion restrictions**, and that the phase III population does not therefore truly reflect the ‘true’ population

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All reports and data is the property of the client but all documents, questionnaires, cards etc. on which the data is recorded shall be the property of the Company. Copies of any or all of this material in an anonymous form can be made available to clients on request on payment of additional fees. If wider circulation of results is intended, Hoffmann&Krueger Consulting Ltd name may not be quoted in connection with the study until the exact form of any communication has been agreed by Hoffmann&Krueger Consulting Ltd. The Client undertakes to inform Hoffmann&Krueger Consulting Ltd of any intended wider publication prior to release and to offer identification of Hoffmann&Krueger Consulting Ltd as the supplier of the work to be published.

3. Disclosure of Interests

It is possible that Hoffmann&Krueger Consulting Ltd and/or one of its associates and/or one of Their respective officers, employees, representatives or agents or another client of the Hoffmann&Krueger Consulting Ltd may have interests, relationships and/or arrangements that give rise to conflicts or interests which are otherwise material in relation to the Engagement ("Other Interests"). So far as Hoffmann&Krueger Consulting Ltd is aware without having made enquiry of any of its clients, no other client of Hoffmann&Krueger Consulting Ltd has, as at the date of the project agreement, any Other Interests and Hoffmann&Krueger Consulting Ltd agrees to notify the Client if it becomes aware during the Engagement of any of its clients having any Other Interests provided that Hoffmann&Krueger Consulting Ltd shall not be required to disclose details of the names of any such clients or any other details concerning such clients to the Client pursuant to this Clause 3

The employees of the Hoffmann&Krueger Consulting Ltd assigned to the Engagement may be unaware of, and in any event are required to disregard, any Other Interests and the Client agrees that Hoffmann&Krueger Consulting Ltd may act for it despite Other Interests and that any profit or remuneration from such interests may be retained by Hoffmann&Krueger Ltd.

The Client agrees that Hoffmann&Krueger Consulting Ltd does not have a duty to disclose any matter which comes to its notice in the course of their business if doing so would constitute a breach of duty owed to other persons.
Hoffmann&Krueger Consulting Ltd will remain bound by any Confidentiality Agreement entered into between the parties.