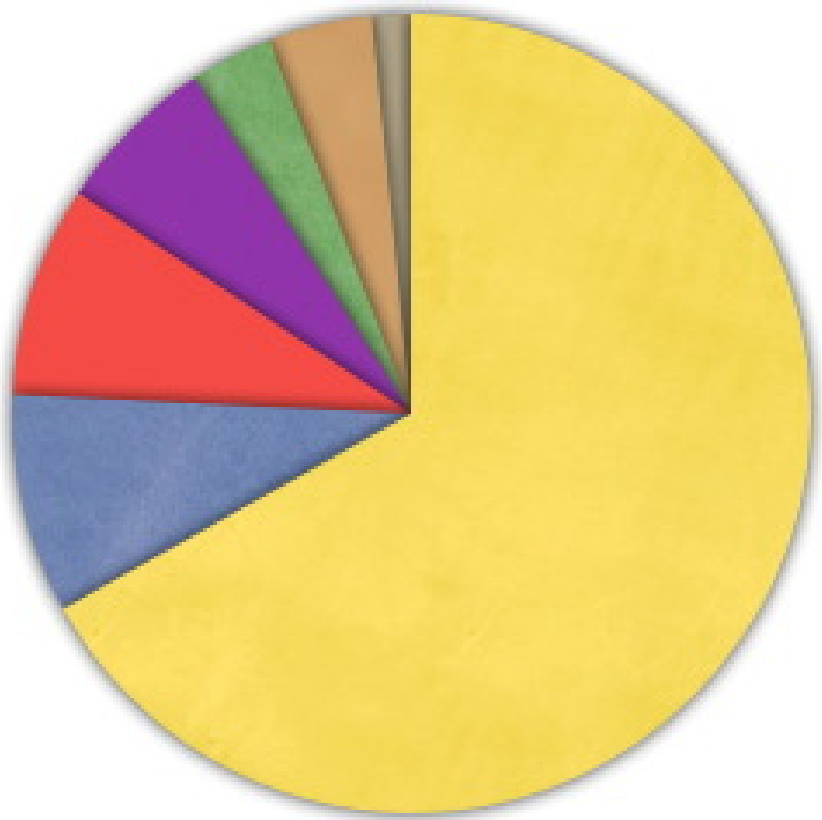
Lecture XII –
Gene therapy for cancer

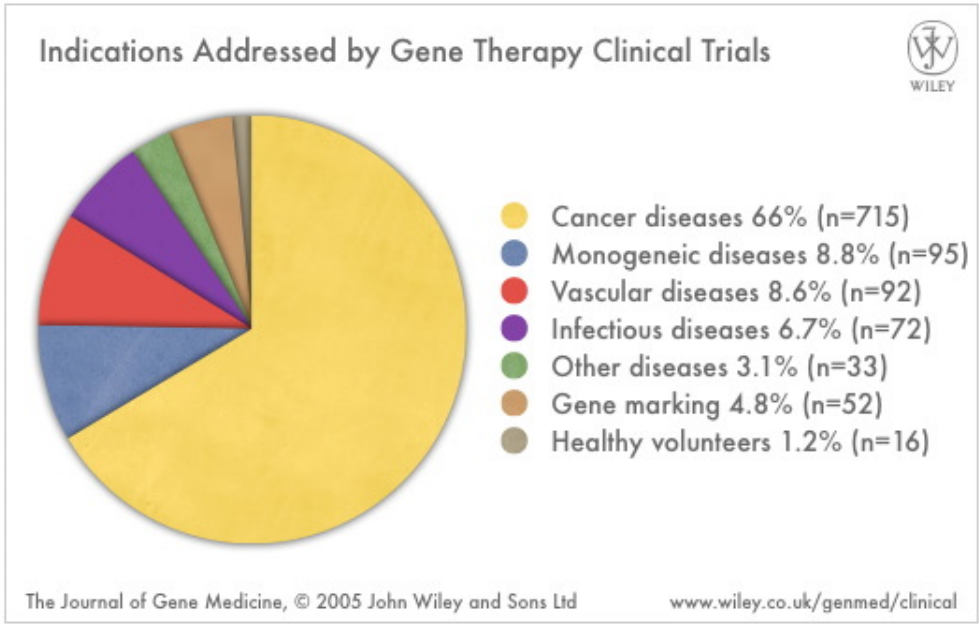
Indications Addressed by Gene Therapy Clinical Trials



- Cancer diseases 67% (n=797)
- Vascular diseases 8.9% (n=106)
- Monogeneic diseases 8.6% (n=102)
- Infectious diseases 6.5% (n=78)
- Other diseases 3.4% (n=40)
- Gene marking 4.2% (n=50)
- Healthy volunteers 1.6% (n=19)

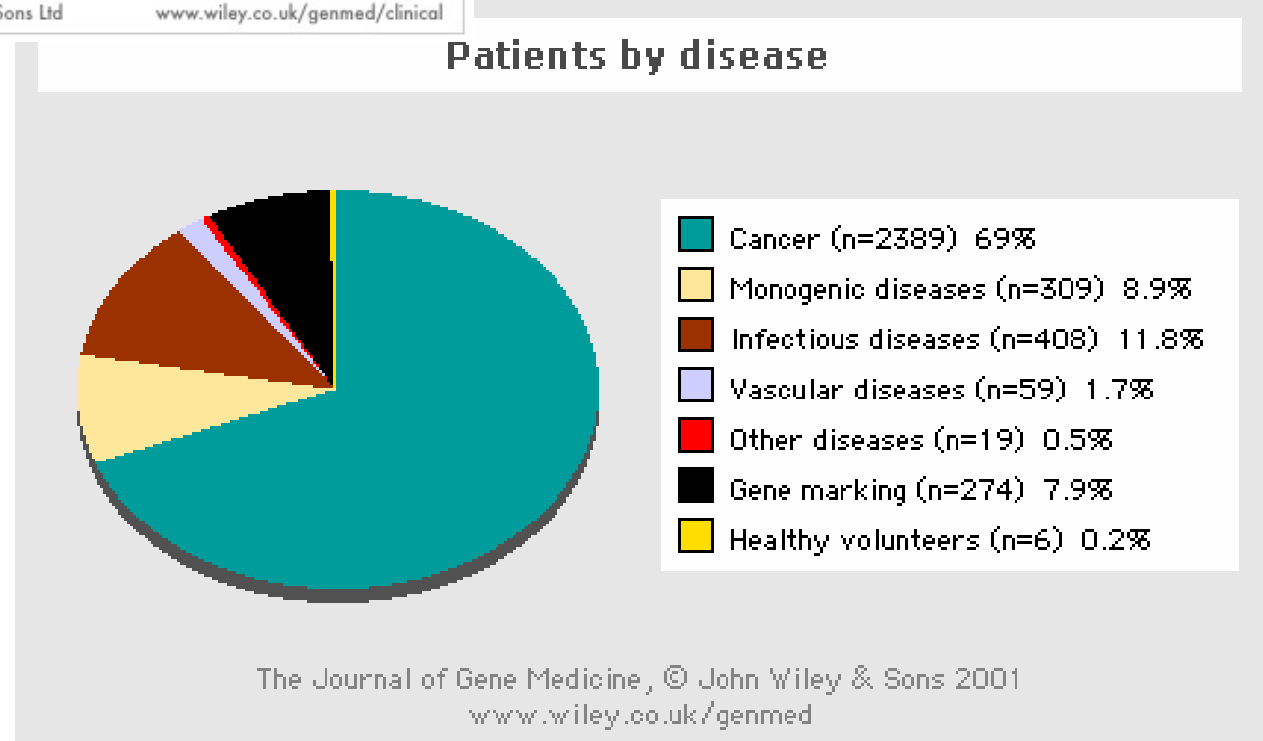
The Journal of Gene Medicine, © 2006 John Wiley and Sons Ltd

www.wiley.co.uk/genmed/clinical


Indications Addressed by Gene Therapy Clinical Trials
WILEY
Cancer diseases 66% (n=715)
Monogeneic diseases 8.8% (n=95)
Vascular diseases 8.6% (n=92)
Infectious diseases 6.7% (n=72)
Other diseases 3.1% (n=33)
Gene marking 4.8% (n=52)
Healthy volunteers 1.2% (n=16)
The Journal of Gene Medicine, © 2005 John Wiley and Sons Ltd
www.wiley.co.uk/genmed/clinical

2005

2001


Patients by disease
Cancer (n=2389) 69%
Monogenic diseases (n=309) 8.9%
Infectious diseases (n=408) 11.8%
Vascular diseases (n=59) 1.7%
Other diseases (n=19) 0.5%
Gene marking (n=274) 7.9%
Healthy volunteers (n=6) 0.2%
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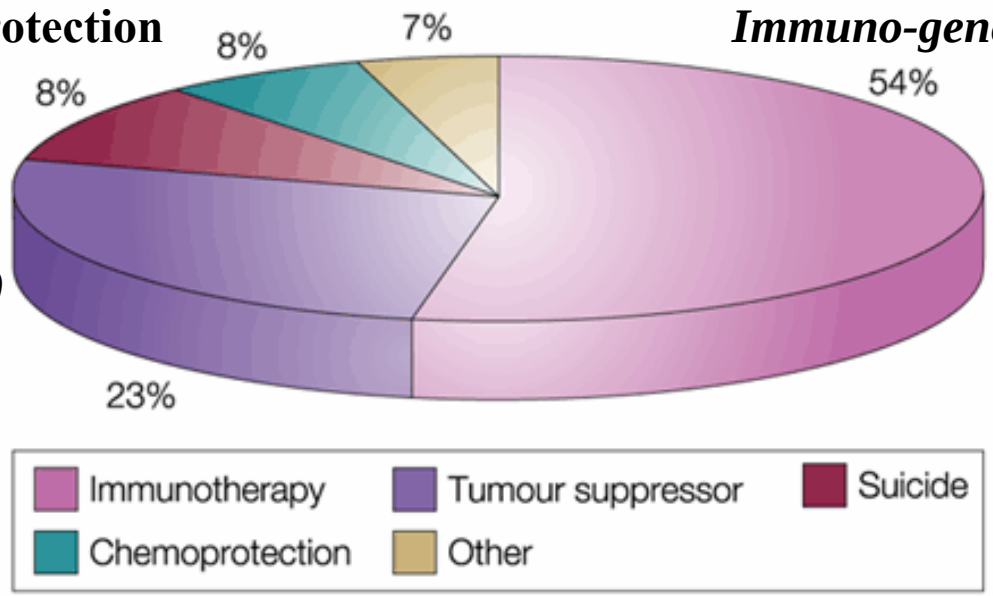
chemoprotection

Immuno-gene therapy

Suicide gene therapy

Tumor suppressor (p53)



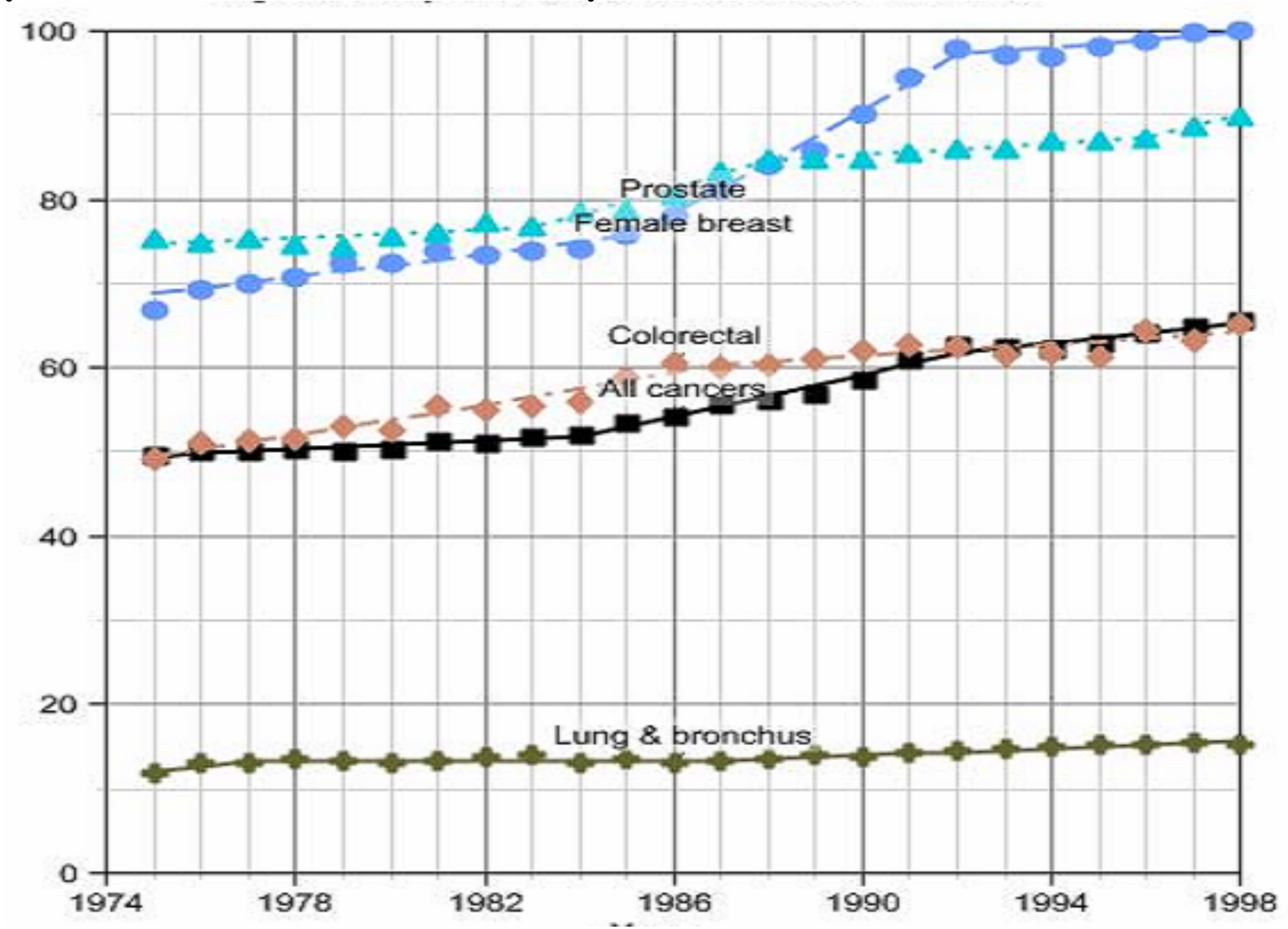
Nature Reviews | Cancer

Figure 1 | **Cancer gene therapy and immunotherapy trials currently listed as open by the US Recombinant Advisory Committee.** Over half of all gene-therapy-based protocols in the United States (113 currently open) are aimed at boosting the immune response to tumour antigens. Trials in melanoma alone account for 54% of immunotherapy trials. Delivery of the tumour-suppressor gene *TP53* accounts for the next largest group, followed by suicide gene delivery, in which viral vectors deliver enzymes that activate prodrugs to toxic products that kill tumour cells and their neighbours. Most of these use herpes simplex virus thymidine kinase (HSV-tk), which activates the prodrug ganciclovir. Chemoprotection is an indirect approach in which bone marrow cells are infected with viruses that protect them from the toxic effects of chemotherapy, by expressing drug-resistance genes.

Aim of cancer gene therapy

Effective killing of most (if not all) cancer cells without serious damage to normal cells and tissues

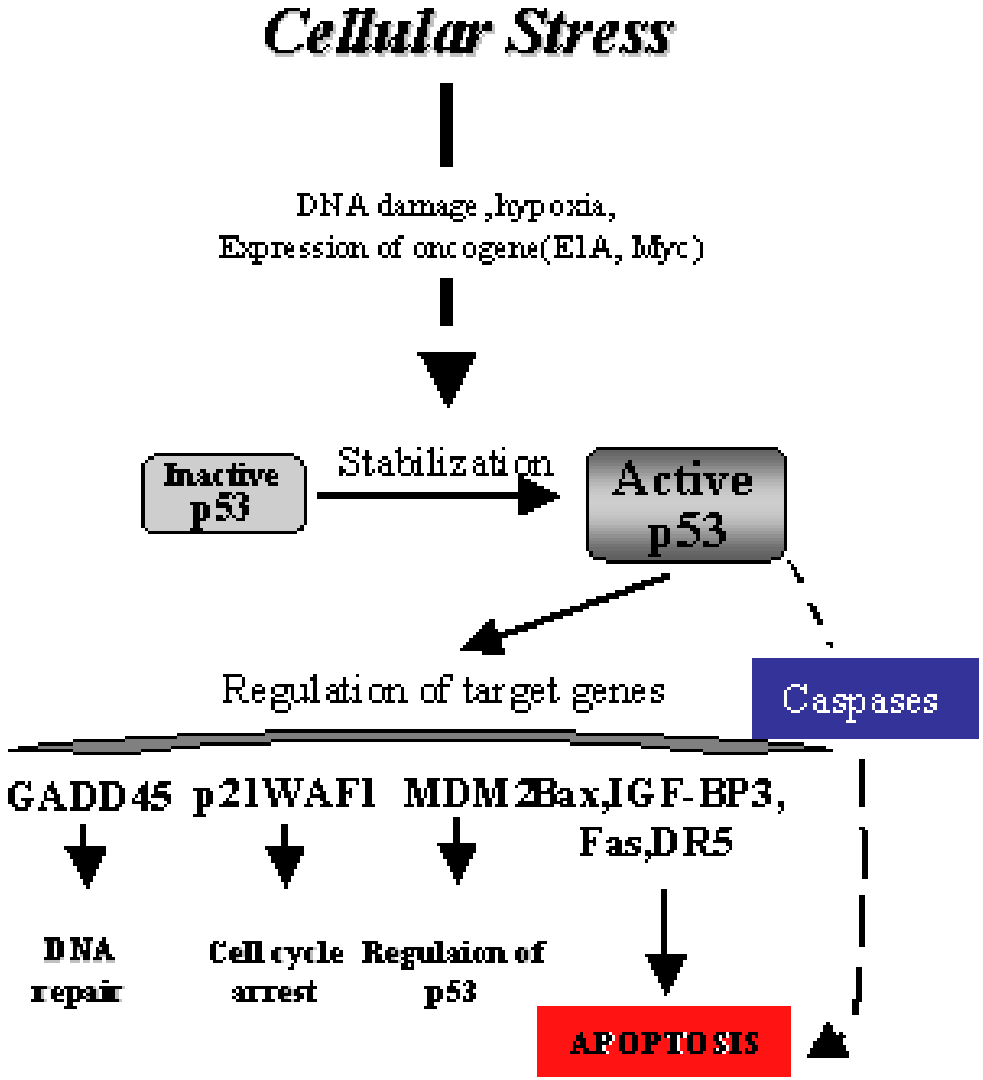
Why there is a need for cancer gene therapy
- 5-years survival rates of patients with different cancer



Cancer gene therapy

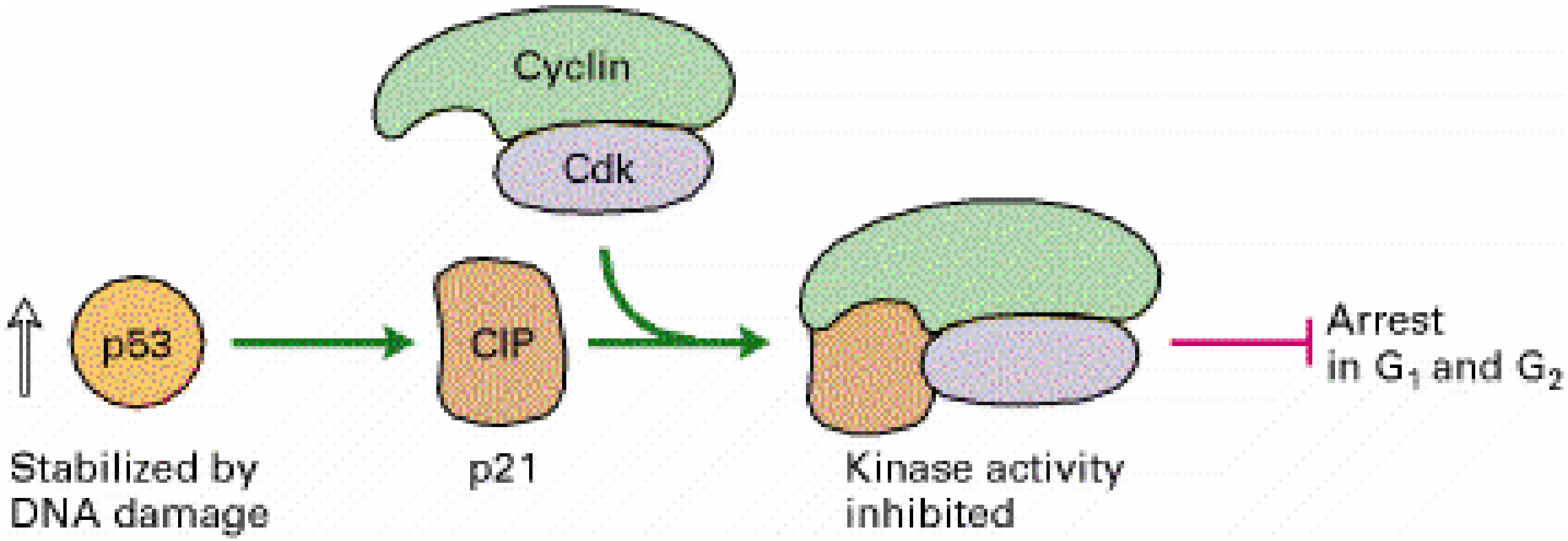
1. Direct attack on tumor cells
    - **a) transfer of tumor suppressor gene**
    - b) inhibition of oncogenes
    - c) suicide genes
    - d) oncolytic viruses (replication-competent viruses)
2. Harnessing immune response to tumor antigens
3. Chemoprotection
4. Anti-angiogenic therapy

p53 – the guardian of the genome

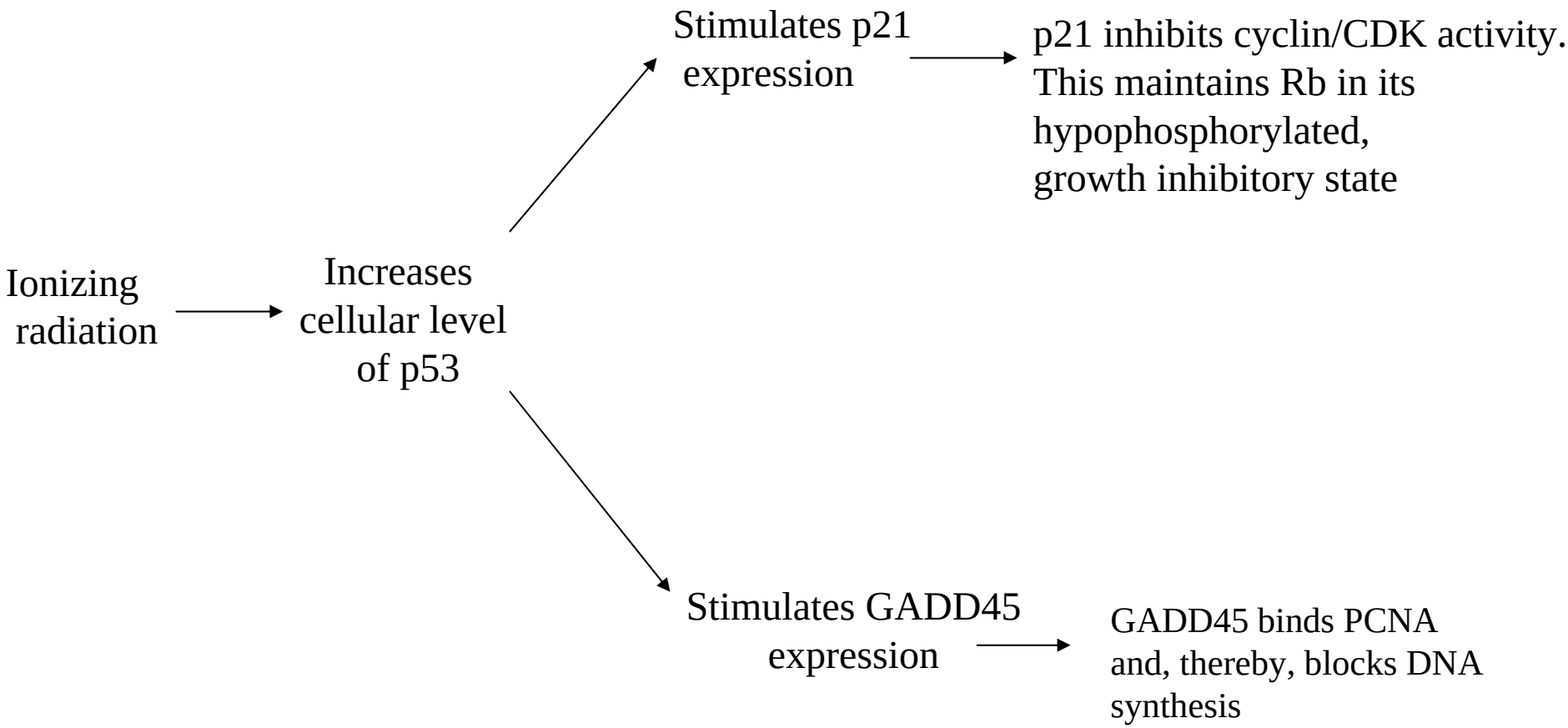


p53-induced cell-cycle arrest in response to DNA damage.

The normally unstable p53 protein is stabilized by damaged DNA, so its concentration increases. Acting as a transcription factor, p53 induces expression of $p21^{CIP}$, a cyclin-kinase inhibitor that inhibits all Cdk1-, Cdk2-, Cdk4-, and Cdk6-cyclin complexes. Binding of $p21^{CIP}$ to these Cdk-cyclin complexes leads to cell cycle arrest in G_1 and G_2



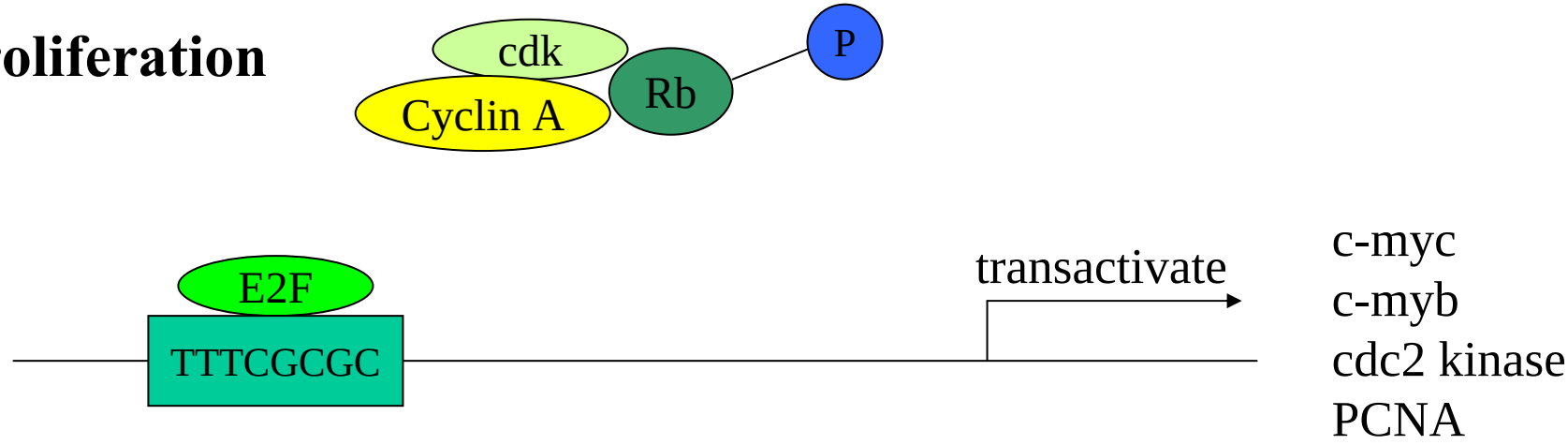
p53 and cell arrest



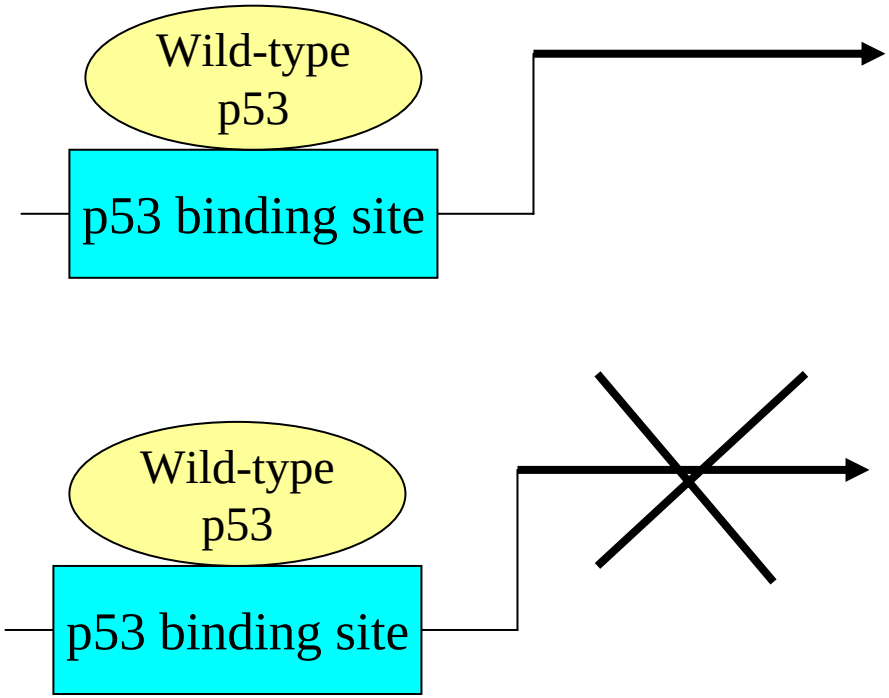
Transcription factor E2F

Quiescence

Proliferation



Mann MJ, Mol Med. Today 2000



The ability of p53 to stimulate transcription is essential for tumor suppression

p53 and gene therapy of cancer

Head and Neck Cancer

Overview

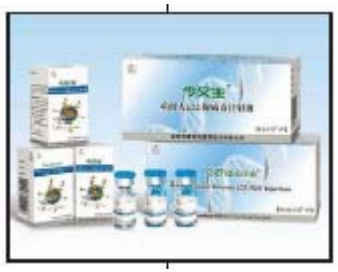
Head and neck cancer is the term given to a variety of malignant tumors that develop in the

- oral cavity (mouth);
- pharynx (throat);
- paranasal sinuses (small hollow spaces around the nose lined with cells that secrete mucus);
- nasal cavity (airway just behind the nose);
- larynx ("Adam's apple" or voice box); and
- salivary glands (parotid, submanidular, sublingual glands that secrete saliva).

Many authorities also include skin tumors of the face and neck and tumors of the cervical lymph nodes.

Excluding superficial skin cancers, but including cancer of the larynx and thyroid, it is conservatively estimated that about 60,000 people are diagnosed with head and neck cancer annually - about 5% of all cancers diagnosed in the United States. There are more than half a million survivors of oral, head, and neck cancer living in the United States today.

First officially registered therapeutic nucleic acid



Gendicine (SiBiono GeneTech, Chiny*)*

Adenoviral vector with a correct p53 gene

Efficient in patients with head and neck cancers

Claimed to be 3 x more efficient than radiotherapy alone

Registered on 16. X. 2003, after 5 years of clinical trials

Advexin – adenoviral vector expressing p53 gene

effective in patients with head and neck cancer

Alfredo and Elena Gonzalvo enjoyed visiting relatives recently in the Philippines.

Research nurse supervisor Marcelo Dolormente helps Bernis Teaters celebrate her fifth anniversary after gene therapy.

Examples of the most advanced clinical trials of cancer gene therapy

Table 1 Biotech firms with gene therapy products for cancer in phase 2 or later of clinical development

Company or research institute	Indication	Delivered gene	Vector	Phase of clinical development
Shenzhen SiBiono Gene Technologies (Shenzhen, China)	HNSCC	Tumor protein p53	Adenovirus	Approved
Shanghai Sunway Biotech (Shanghai, China)	HNSCC	HAdv5 oncolytic virus	Adenovirus	Phase 3
AnGes MG (Osaka, Japan)	Arteriosclerosis obliterans	Hepatocyte growth factor	Plasmid	Phase2
GenVec, Inc. (Gaithersburg, MD, USA)	Pancreatic, esophageal and rectal cancers	Human tumor necrosis factor-?	Adenovirus	Phase 2
Introgen (Austin, TX, USA)	Head and neck, lung, breast, esophageal, ovarian, bladder, brain, prostate and bronchoalveolar cancers	Tumor protein p53	Adenovirus	Phases 1–3
Transgene (Strasbourg, France)	Cervical cancer	Human papilloma virus type 16 E6 and E7 antigens and interleukin 2	Vaccinia virus	Phase 2
Transgene (Strasbourg, France)	Breast, lung, prostate and renal cancers	Human mucin 1 antigen and interleukin 2	Vaccinia virus	Phase 2

Nature Biotechnology, January 2004

ADVEXIN® Clinical Trials**

Cancer Type	Status
Head and Neck (both monotherapy and combined with chemotherapy)	Phase 1 / Phase 2 / Phase 3
Non-Small Cell Lung (combined with radiation therapy)	Phase 1 / Phase 2 / Phase 3
Breast (combined with chemotherapy)	Phase 1 / Phase 2 / Phase 3
Perioperative (and surgery)	Phase 1 / Phase 2 / Phase 3
Esophageal	Phase 1 / Phase 2 / Phase 3
Prostate	* Phase 1 / Phase 2 / Phase 3
Intravenous Administration	Phase 1 / Phase 2 / Phase 3
Ovarian	* Phase 1 / Phase 2 / Phase 3
Bladder	* Phase 1 / Phase 2 / Phase 3
Bronchoalveolar	* Phase 1 / Phase 2 / Phase 3
Brain (glioblastoma)	* Phase 1 / Phase 2 / Phase 3

* Conducted in conjunction with the National Cancer Institute.

** We hold the worldwide commercial rights to the product candidates related to each of these programs.

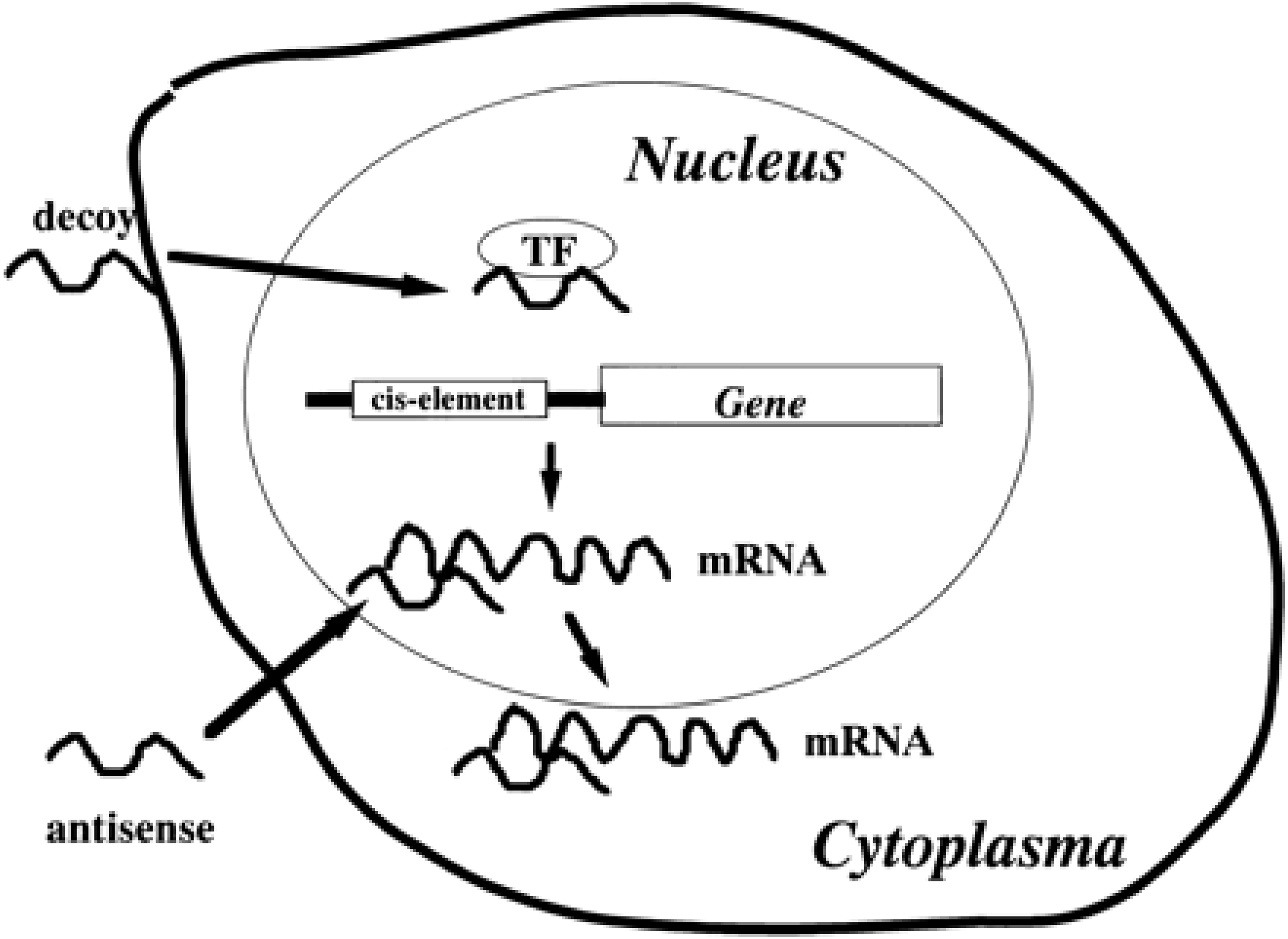
Other therapeutic nucleic acids developed by Introgen

INGN 241 ** - *mda-7*

INGN 241 uses the mda-7 gene, a tumor suppressor gene that, like p53, induces cell death, or apoptosis, in many types of cancer. In addition to its known activity as a tumor suppressor gene, the protein produced by the mda-7 gene is secreted and may also stimulate the body's immune system to kill metastatic tumor cells and to protect the body against cancer. This offers an advantage in treating various cancers because it may attack cancer using two different mechanisms. The mda-7 gene acts as a cytokine, or immune system modulator, and is also known as interleukin-24, or IL-24.

Cancer gene therapy

1. Direct attack on tumor cells
    a) transfer of tumor suppressor gene
    b) **inhibition of oncogenes**
        - antisense therapy
        - ribozymes
    c) suicide genes
    d) oncolytic viruses (replication-competent viruses)
2. Harnessing immune response to tumor antigens
3. Chemoprotection
4. Anti-angiogenic therapy


Nucleus
decoy
TF
cis-element
Gene
mRNA
mRNA
antisense
Cytoplasma

Blocking oncogenes in tumors

Sis - growth factor

erB-2

abl

ras

jun

myc

DNA decoys for cancer gene therapy

Will they be effective?

Remember the story of Edifoligide

When Bad Gene Transfer Is Good...

Cancer gene therapy

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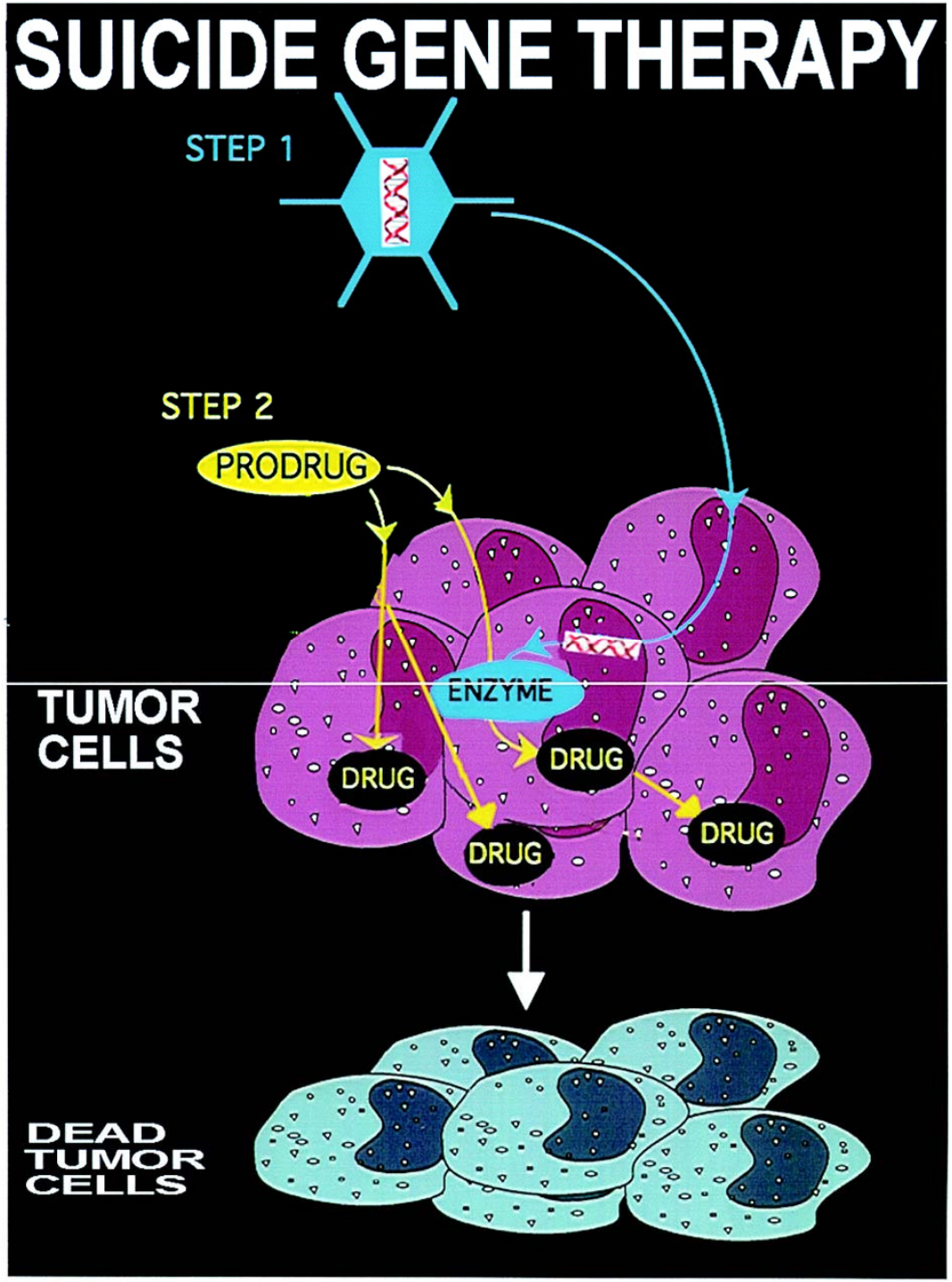
Suicide gene therapy –pro-drug activation

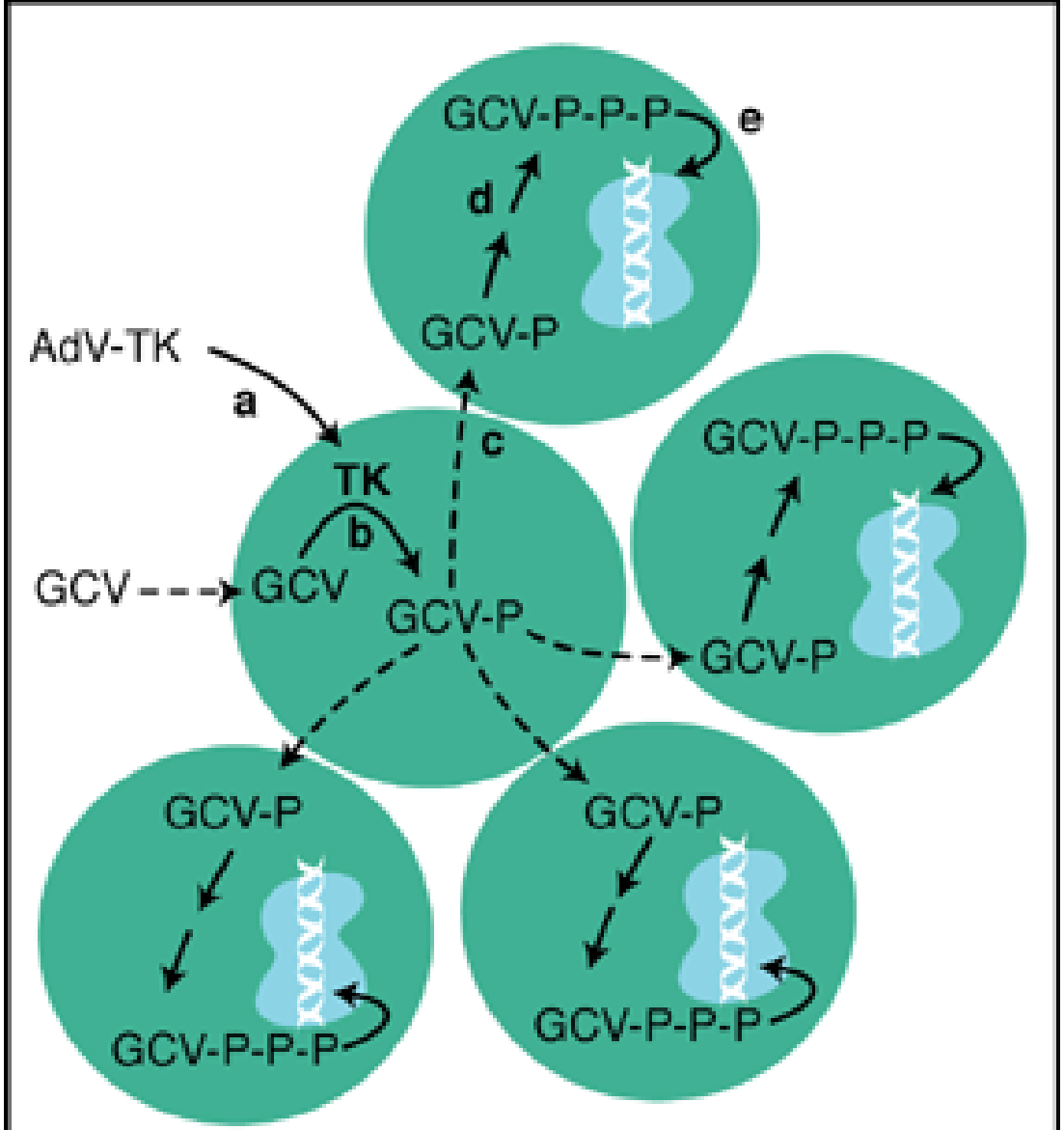
The objective of pro-drug activation therapy is to express an activating enzyme within the tumor, which will then activate a systemically delivered, inactive pro-drug at the target site only

Bystander effect
(efekt sąsiedztwa)



Enzyme-prodrug combination for suicide gene therapy

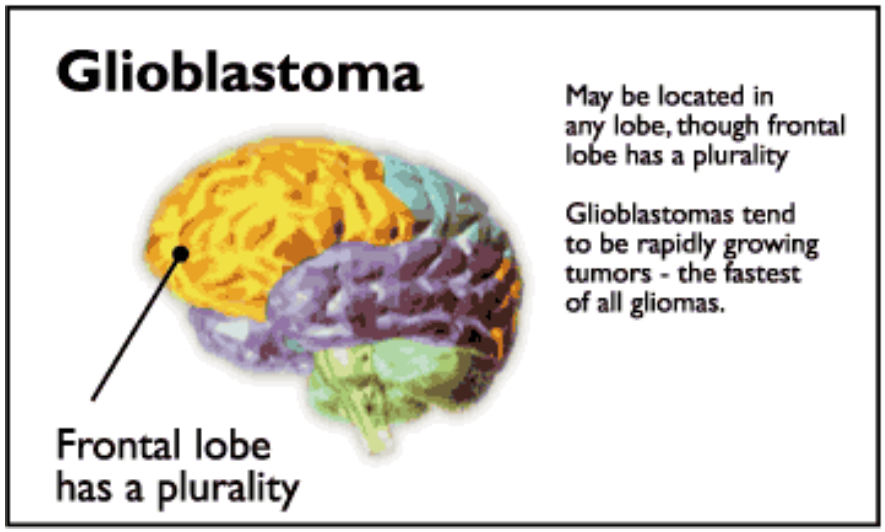
Enzyme	Prodrug	Product	Mechanism
HSV-tk	ganciclovir	ganciclovir triphosphate	blocks DNA synthesis
cytosine deaminase	5-fluorocytosine	5-fluorouracil (5-FU)	blocks DNA and RNA synthesis (pyrimidine antagonist)
cytochrome P450	cyclophosphamide	phosphoramide mustard	DNA alkylating agent; blocks DNA synthesis



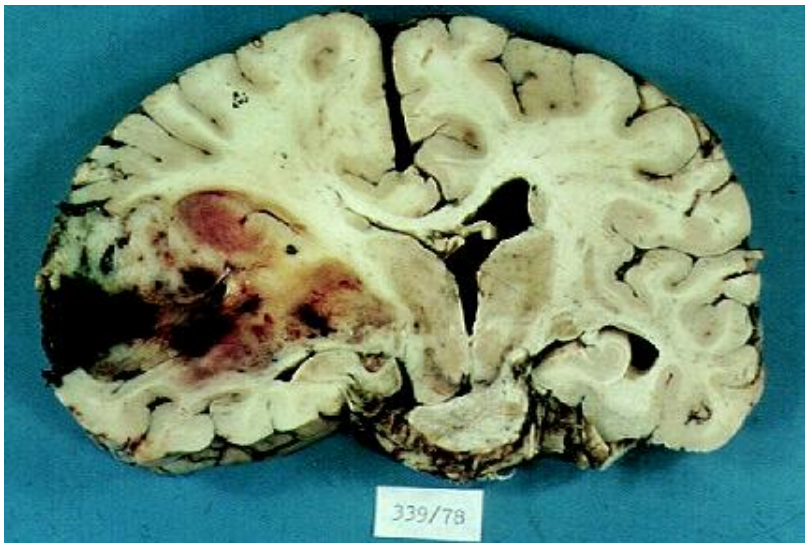
Suicide gene therapy

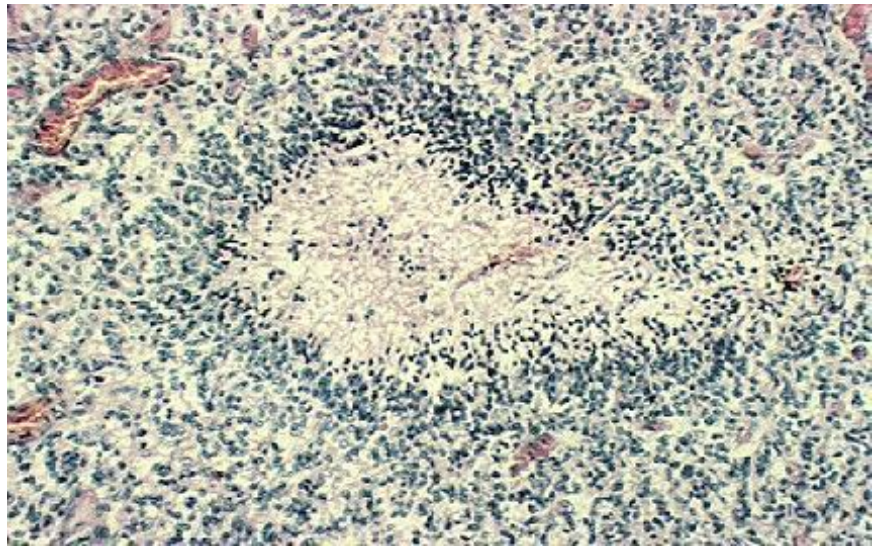
Expert Reviews in Molecular Medicine

©2003 Cambridge University Press



Glioblastoma multiforme - Infiltrative; rapid-growing; occurs: most frequently in mid-aged; apt to involve both cerebral hemispheres via the corpus callosum; Average Survival: 1 year





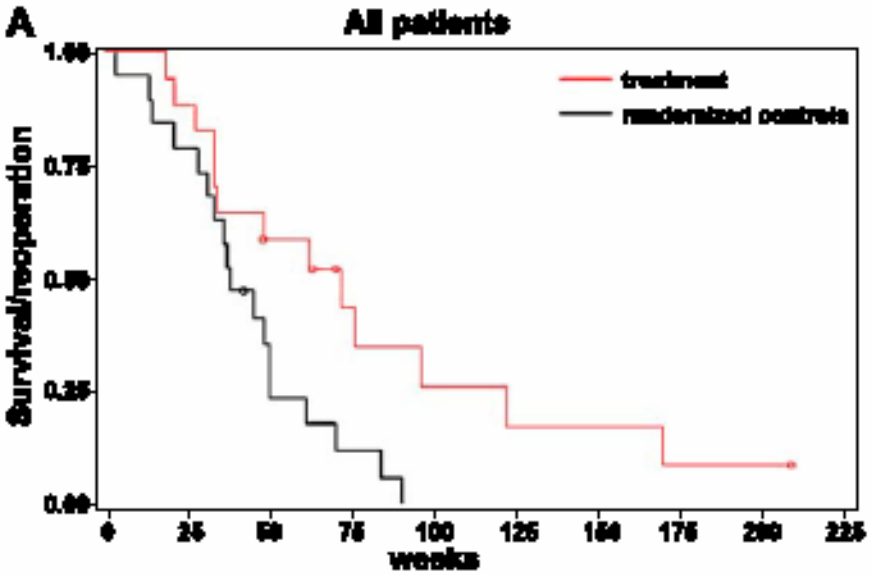
Mol Ther. 2004 Nov;10(5):967-72

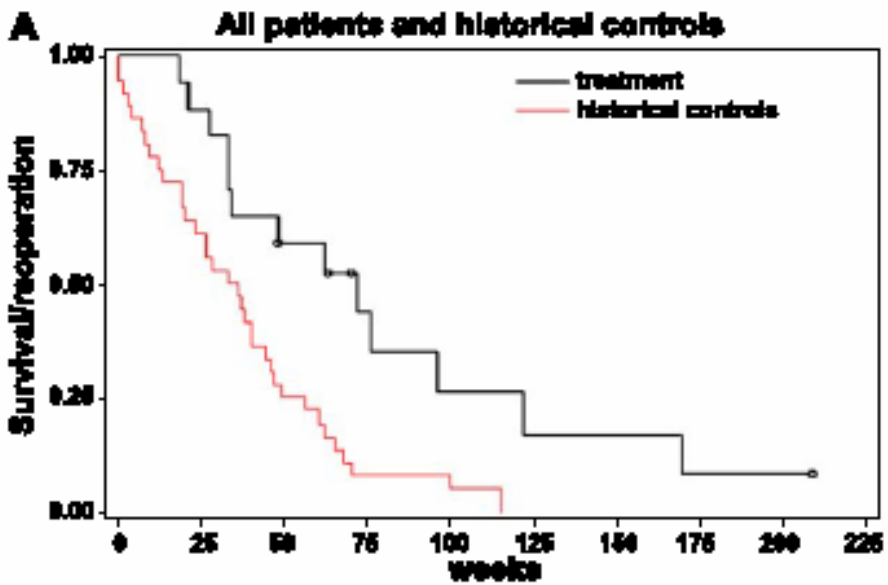
AdvHSV-tk gene therapy with intravenous ganciclovir improves survival in human malignant glioma: a randomised, controlled study.

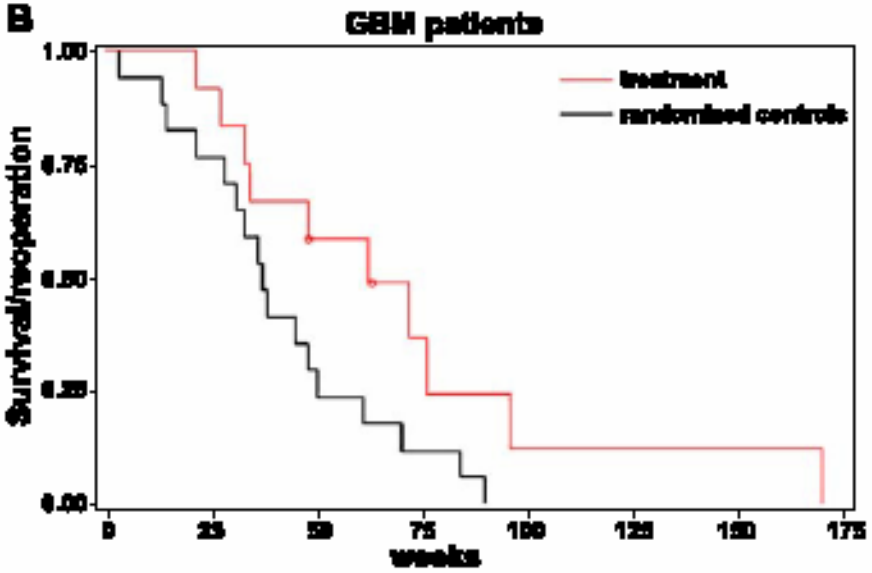
Immonen A, Vapalahti M, Tyynela K, Hurskainen H, Sandmair A, Vanninen R, Langford G, Murray N, Yla-Herttuala S.
Department of Neurosurgery, University of Kuopio, A.I. Virtanen Institute, Finland.

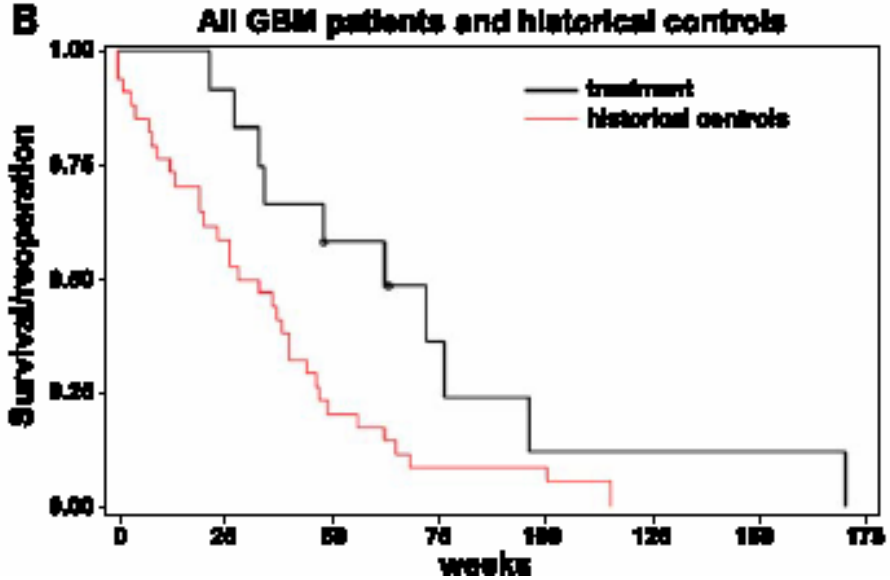
Malignant glioma is a devastating brain tumor with no effective treatment. This randomised, controlled study involved 36 patients with operable primary or recurrent malignant glioma. Seventeen patients were randomized to receive AdvHSV-tk gene therapy (3 x 10(10) pfu) by local injection into the wound bed after tumor resection, followed by intravenous ganciclovir (GCV), 5 mg/kg twice daily for 14 days. The control group of 19 patients received standard care consisting of radical excision followed by radiotherapy in those patients with primary tumors. The primary end-point was survival as defined by death or surgery for recurrence. Secondary end-points were all-cause mortality and tumour progression as determined by MRI. Overall safety and quality of life were also assessed. Findings were also compared with historical controls (n = 36) from the same unit over 2 years preceding the study. AdvHSV-tk treatment produced a clinically and statistically significant increase in mean survival from 39.0 +/- 19.7 (SD) to 70.6 +/- 52.9 weeks (P = 0.0095, log-rank regression vs. randomized controls). **The median survival time increased from 37.7 to 62.4 weeks.** Six patients had increased anti-adenovirus antibody titers, without adverse effects. The treatment was well tolerated. **It is concluded that AdvHSV-tk gene therapy with GCV is a potential new treatment for operable primary or recurrent high-grade glioma.**

AdvHSV-tk gene therapy with intravenous ganciclovir improves survival in human malignant glioma: a randomised, controlled study









Cerepro™ has been granted Orphan Drug Status by the European Committee for Orphan Medicinal Products and by the Office of Orphan Products Development, FDA

LONDON, Jan. 5, 2007 - **Ark Therapeutics Group plc** (LSE: AKT) ("Ark" or the "Company) today provides an update on the Phase III trial (Study 904) being undertaken on its lead product, CereproTM, for the treatment of high grade glioma (malignant brain tumour).

The independent Data and Safety Monitoring Board (DSMB) met on December 13th 2006 to review the data from the first 130 patients entered into the trial. Ark has now been notified by the DSMB that the side effect profile observed to date in Study 904 is in line with that previously reported, giving the DSMB no cause for concern nor requiring any alteration in the design and architecture of the trial. The DSMB has unanimously recommended that the Company continue the study without modification.

Recruitment into Study 904 has now passed 160 patients, in line with the Company's previous guidance.

Trials completed to date have shown that Cerepro™ treatment produces an average extension of 7.5 months of life, giving around 15.5 months survival in a disease where most patients will only live for around 8 months

Cancer gene therapy

1. Direct attack on tumor cells
    a) transfer of tumor suppressor gene
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    d) oncolytic viruses (replication-competent viruses)
2. Harnessing immune response to tumor antigens
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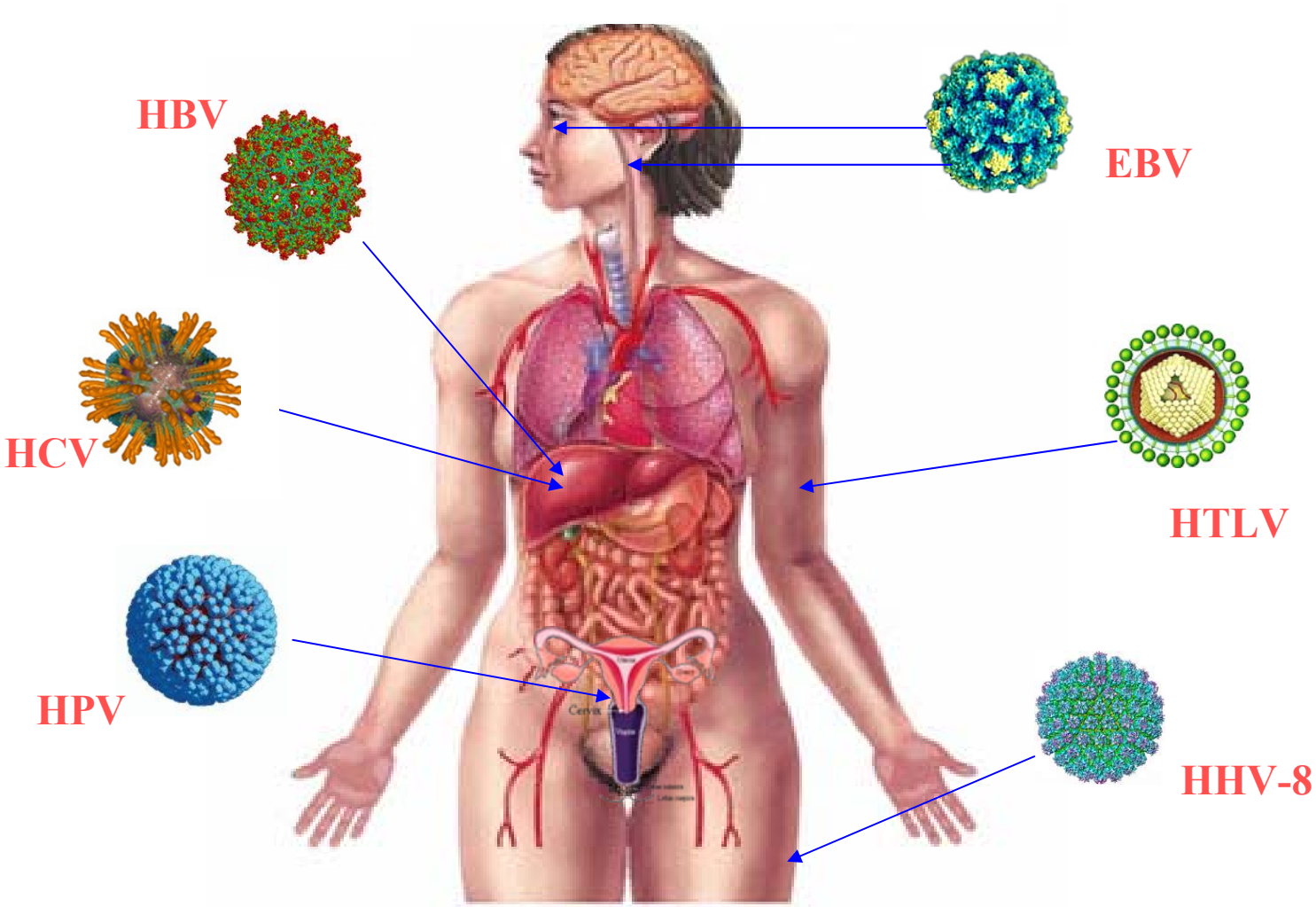
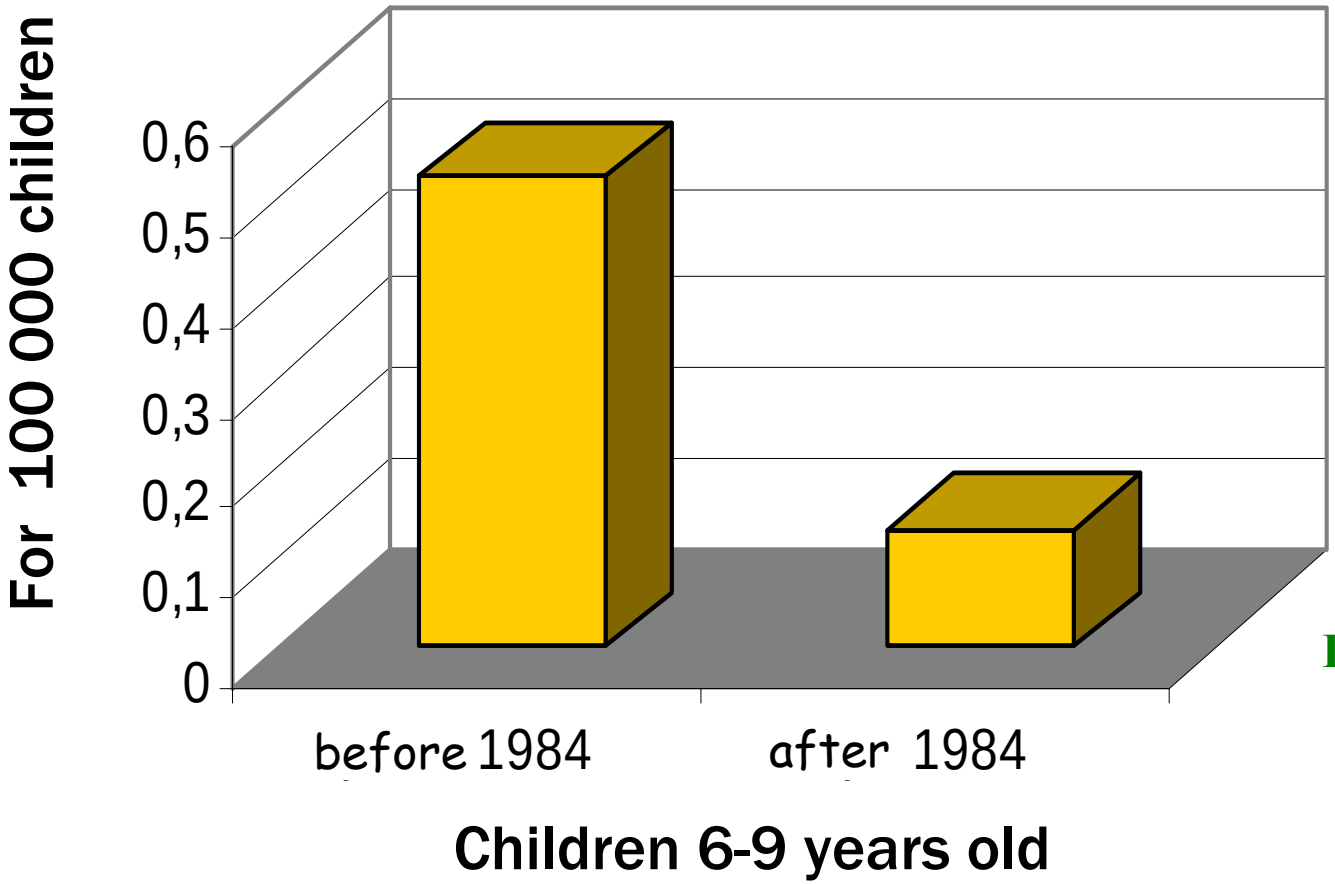
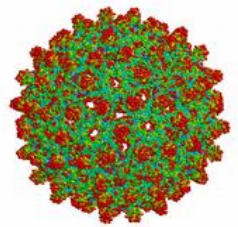
Viruses which increase the risk of cancer



Vaccination against hepatitis B decreases the risk of liver cancer





Data: 1995, Taiwan

Replication competent viruses

Naturally occuring viruses

- autonomously replicating parvoviruses
- human reoviruses
- vesicular stomatitis virus
- Newcastle disease virus

Engineered oncotropic viruses

- conditionally replicating adenoviruses (such as ONYX-15)

Cancer gene therapy

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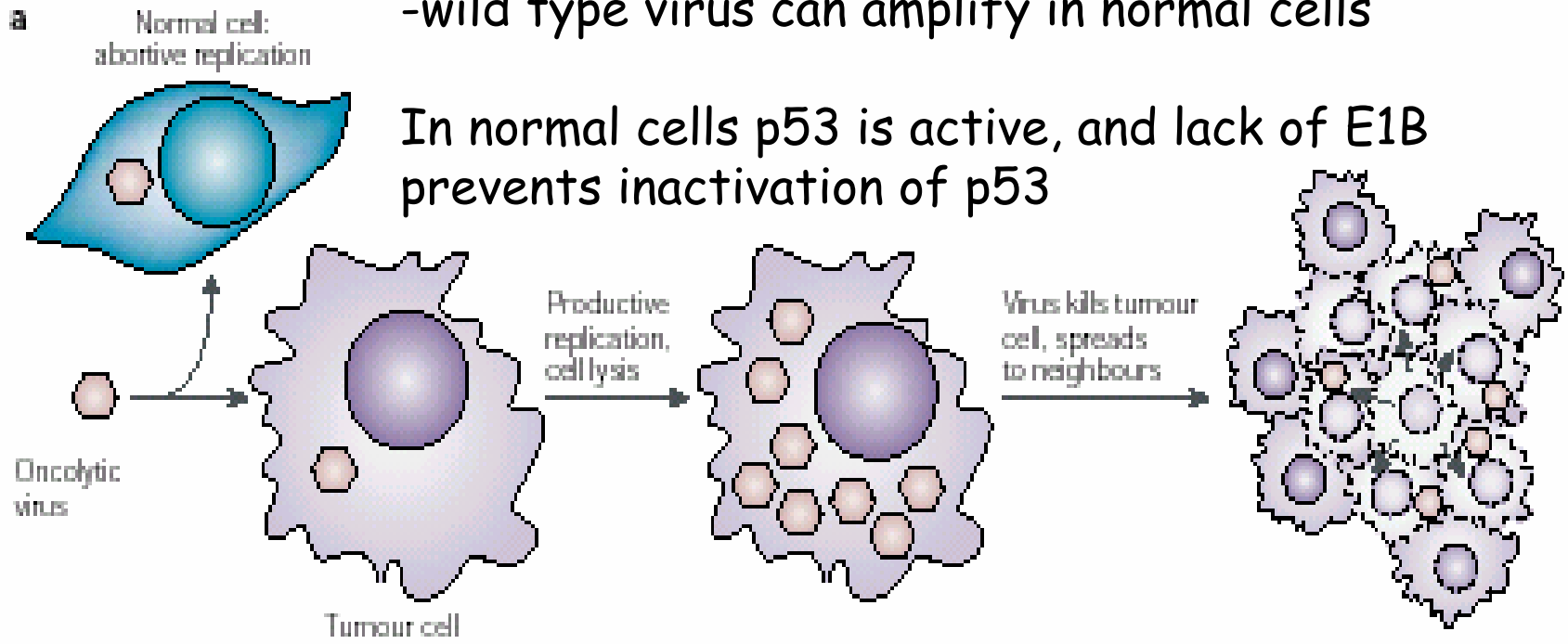
        - ribozymes
    c) suicide genes
    d) oncolytic viruses (replication-competent viruses)
2. Harnessing immune response to tumor antigens
    - overexpression of cytokines genes
    - tumor vaccines
3. Anti-angiogenic therapy

Oncolytic viruses

Lyse only tumor cells,
Adenovirus ONYX-015 , with deletion in E1B gene, may amplify only in cells with mutated p53 gene

E1B 55K protein binds to p53 and blocks its function
-wild type virus can amplify in normal cells

In normal cells p53 is active, and lack of E1B prevents inactivation of p53



Genetically modified tumor vaccines

- Immune system may play a role in controlling tumor growth and development
- However, antigens present on tumor cells are not sufficient to boost immune response

hence

Modification of tumor cells – i.e, overexpressing certain genes, may stimulate immune system to respond to tumor cells

Strategy of genetically modified tumor vaccines

1. Isolate tumor cells from a patient – however, it is often not possible to use autologic cells

2. Alternative – culture other tumor cells- eg, cell line of the same type – i.e. allogeneic cell line

3. Transduce such cells with vector – eg. Retroviral vector harboring cytokine gene

4. Inject such modified cells into patients

5. Antigens present on allogeneic tumor cells stimulate immune system, which respond to the same antigens present on patient's tumor
   Cytokines enhance the response

TABLE 3 Cytokine gene therapy trials

Cytokine	Vector	Malignancy	Immune response	Clinical response	Reference
IL-12	Vaccinia	Mesothelioma	T cell infiltrate	0/6	78
GM-CSF	Retrovirus	Melanoma	Infiltrate at vaccine site	1/ 5 CR	79
IFN-γ	Retrovirus	Melanoma	Antibodies to tumor antigens	5/8 SD 3/8 CR/PR	80

CR, complete response; PR, partial response; SD, stable disease.

IL-6 and sIL-6R retrovirus melanoma (allogeneic)

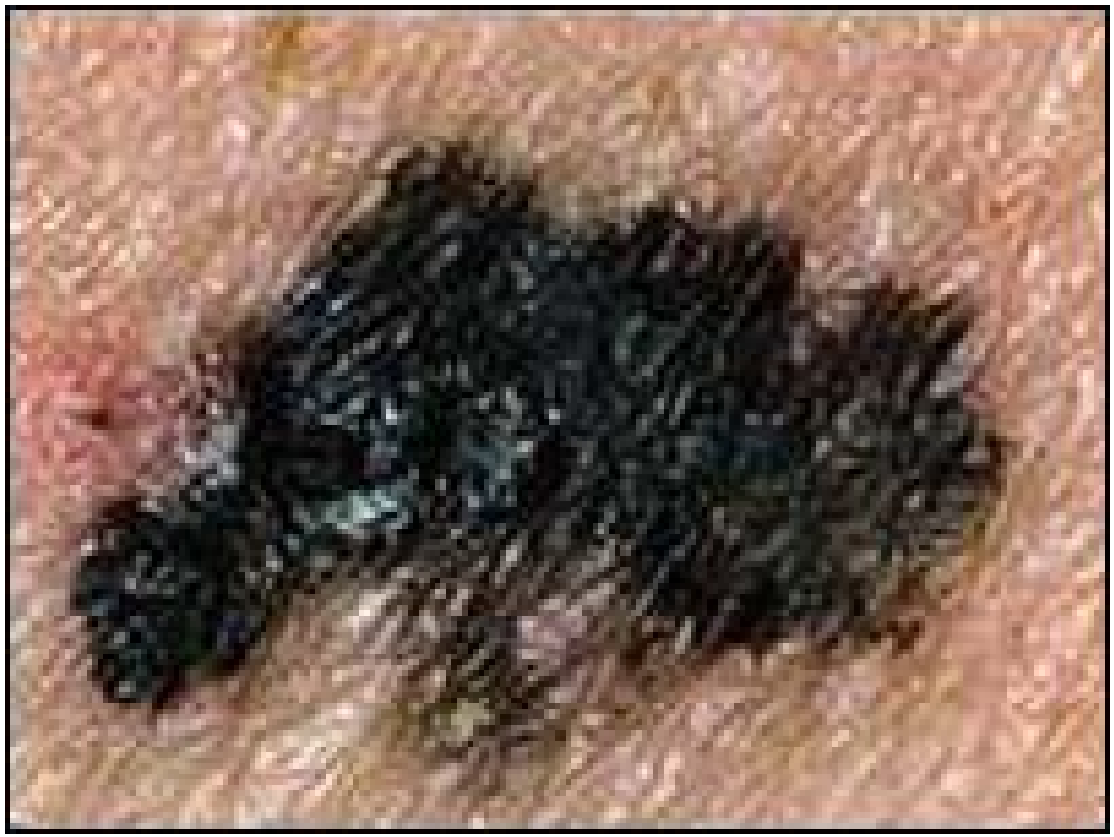
A. Mackiewicz et al., - Poznań

Wadhwa et al., Ann Rev Med. 2002

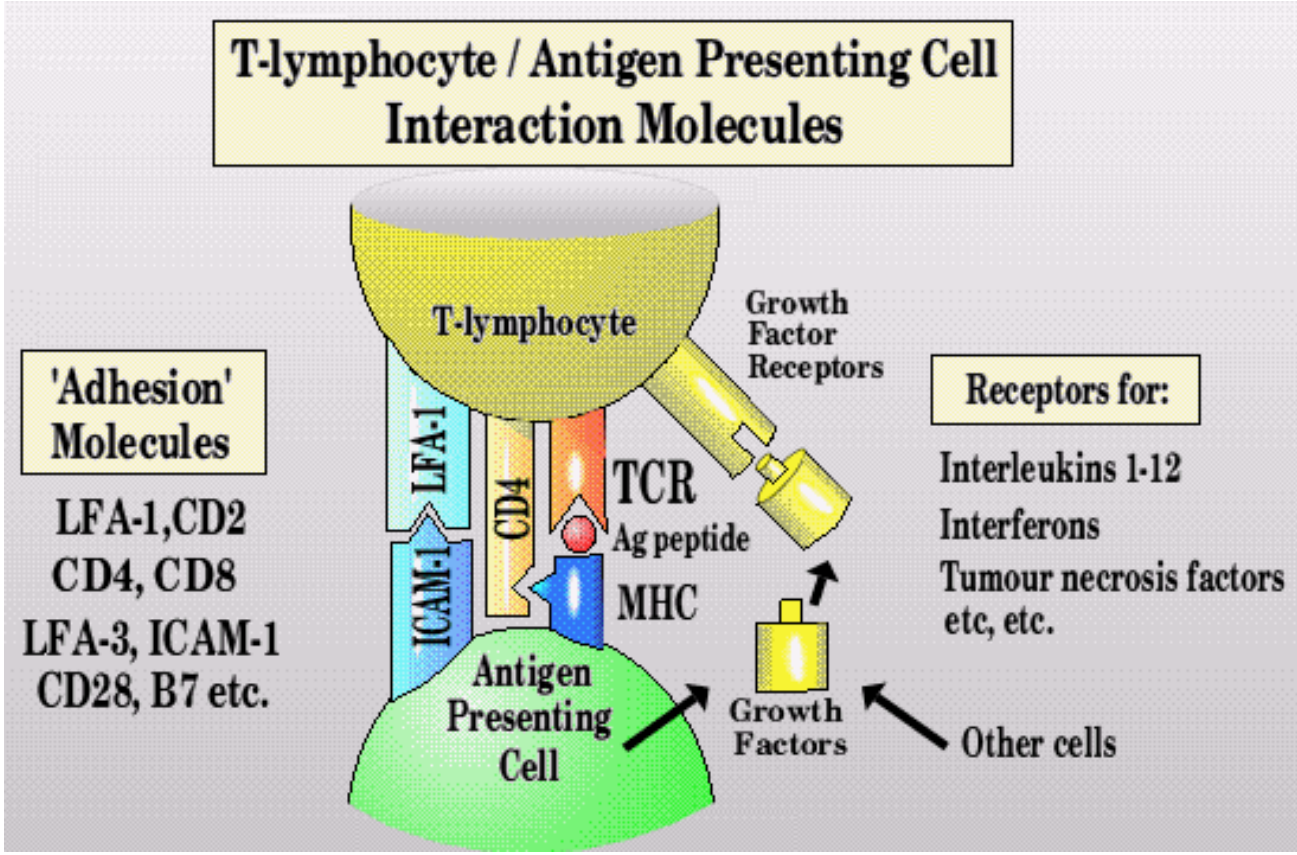
Gene therapy for treatment of melanoma

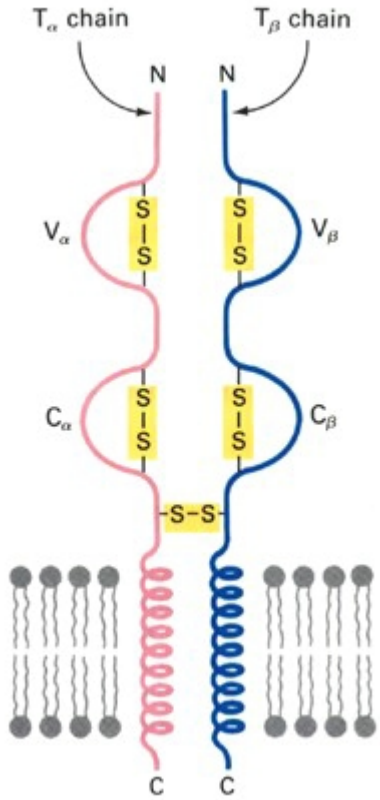
Cancer Regression in Patients After Transfer of Genetically Engineered Lymphocytes.

Morgan at al.. (Rosenberg), Science, 31 August 2006



Tumor associated antigens (TAA) are recognized by T cell receptor (TCR) on the T lymphocyte surface, which is composed of the TCR α and β-chains





Genes encoding TCR specific for a variety opf TAA have now been cloned and these include:

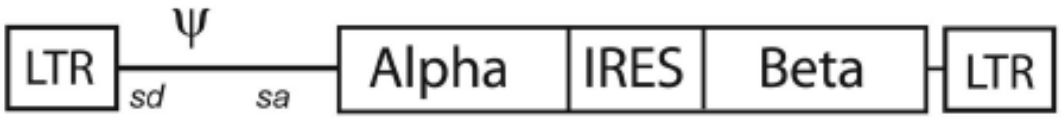
1) the MART-1 and gp100 melanoma/melanocyte differentiation antigens
2) the NY-ESO-1 cancer-testis antigen present on many epithelial cancers
3) the epitope from the p53 molecule, expressed on the surface of approx. 50% of cancers of common epithelial origin

Cancer Regression in Patients After Transfer of Genetically Engineered Lymphocytes

Morgan at al.. (Rosenberg), Science, 31 August 2006

Peripheral blood lymphocytes (PBL) of the patients with refractory melanoma were transduced ex vivo with retroviral vector encoding T cell receptor (TCR), recognizing:

1) the MART-1 and gp100 melanoma/melanocyte differentiation antigens



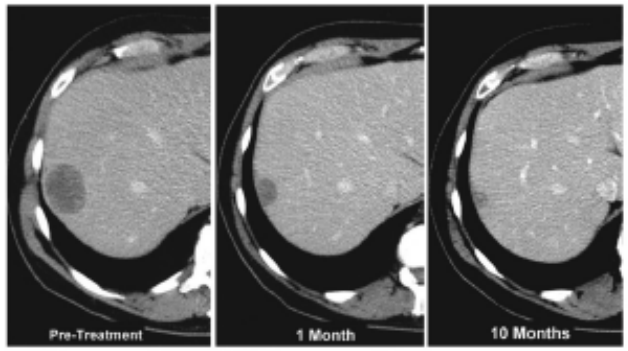
Transduction with these TCR encoding retroviral vectors converted normal PBL to cells capable of sepcifically recognizing and destroying both fresh and cultured cells from multiple common cancers

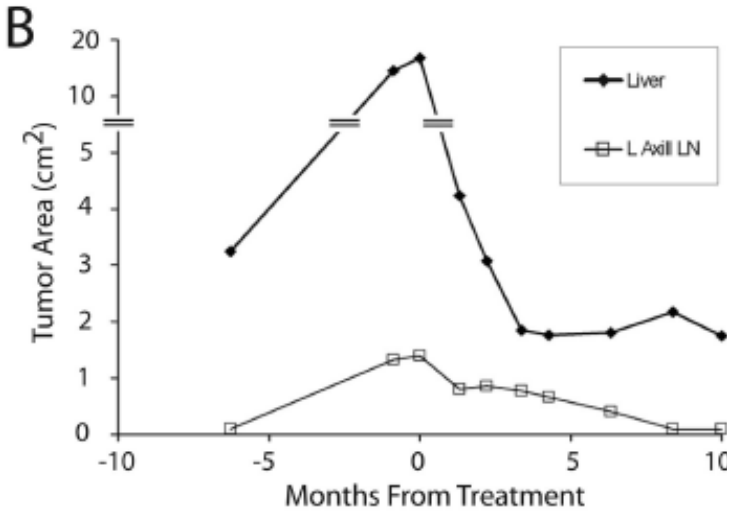
Those TCR transduced T cells secreted IFN-γ following co-culture with melanoma cells

Cancer Regression in Patients After Transfer of Genetically Engineered Lymphocytes

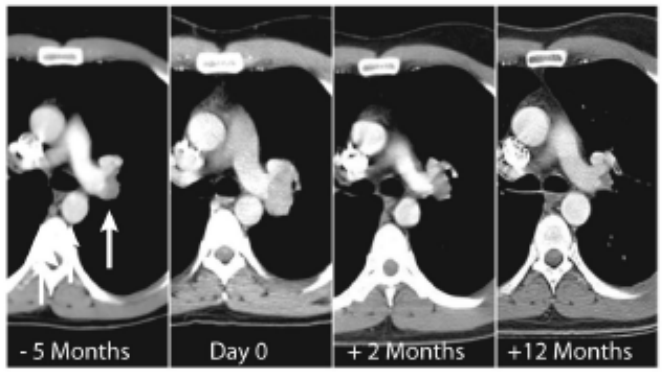
Morgan at al.. (Rosenberg), Science, 31 August 2006

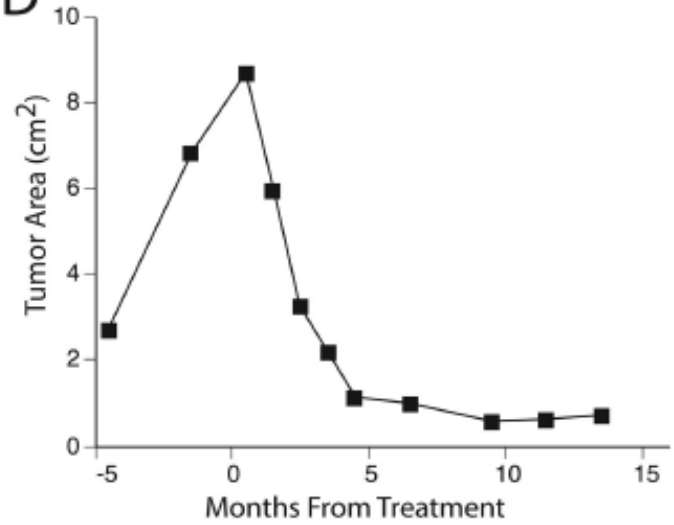
Patient 4 liver metastasis





Patient 14 lymph node metastasis

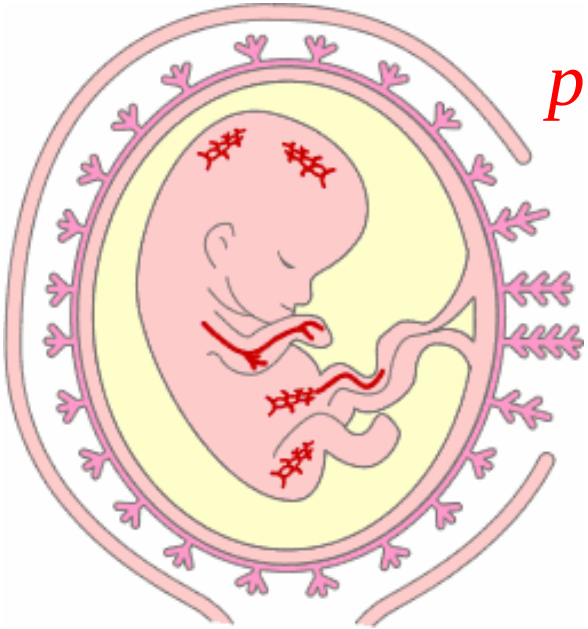




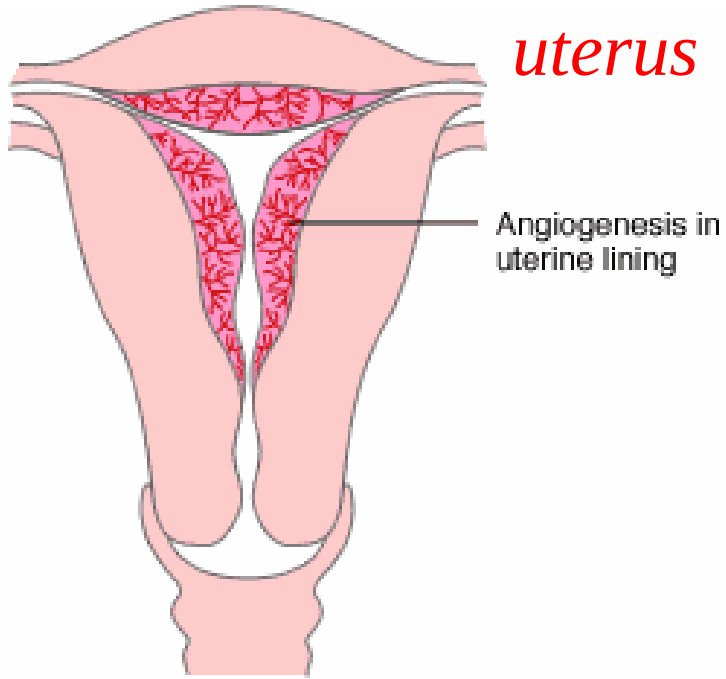
How to starve tumor to death...

Anti-angiogenic gene therapy

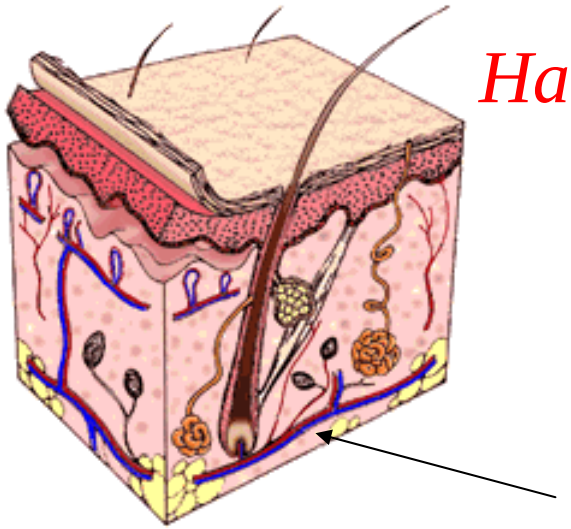
Physiological angiogenesis in adults is restricted



placenta

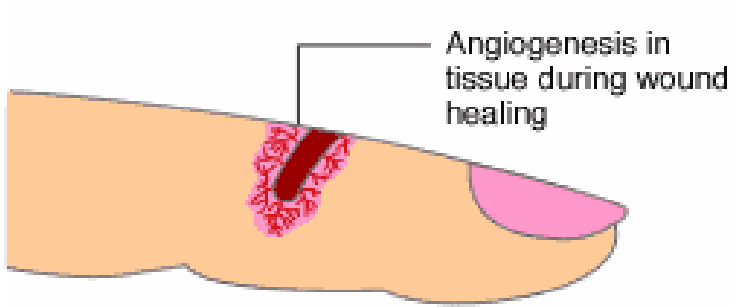


uterus

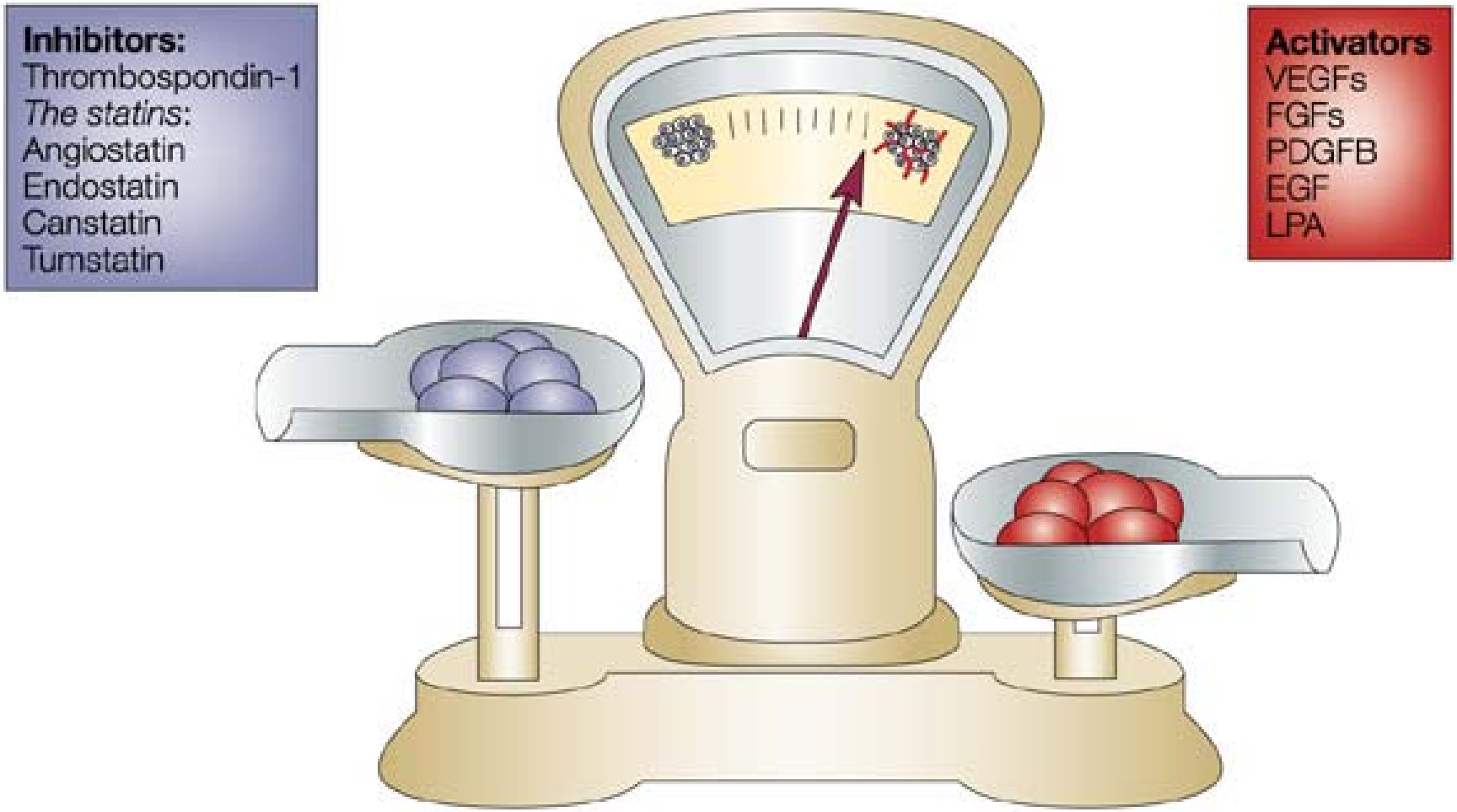


Hair growth

Wound healing

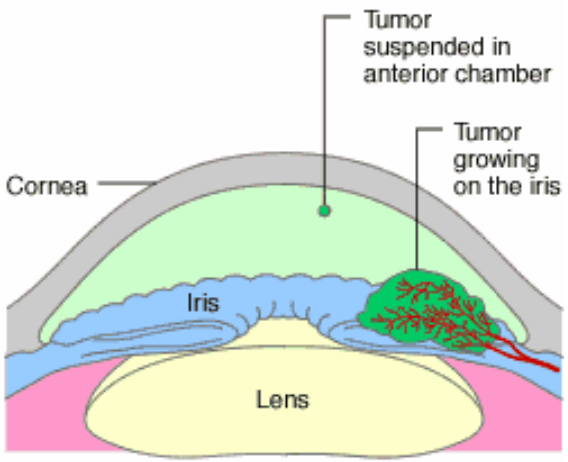


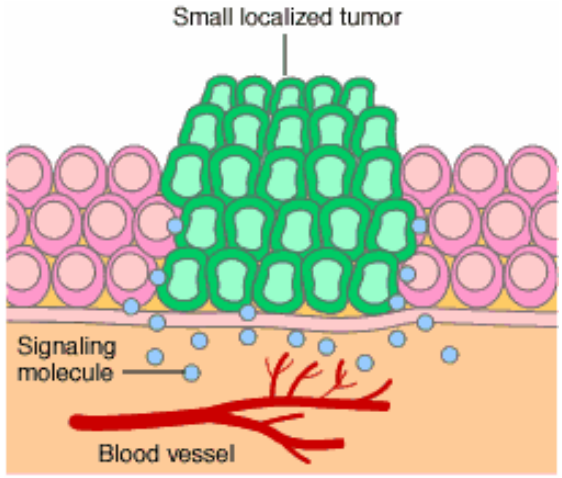
In healthy organism the action of activators and inhibitors of angiogenesis is balanced

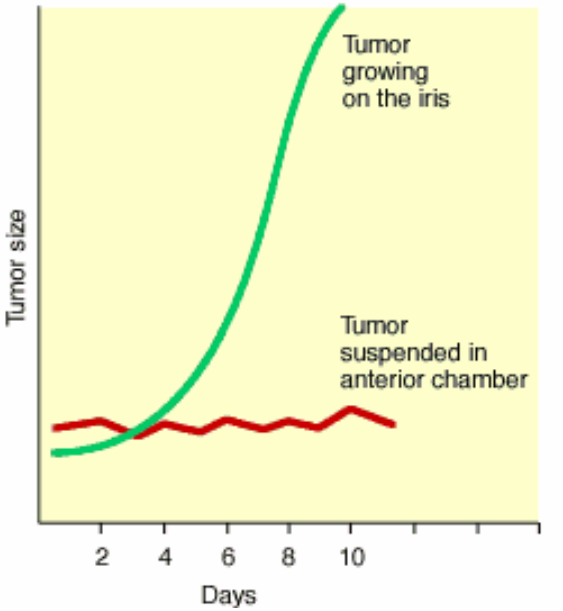


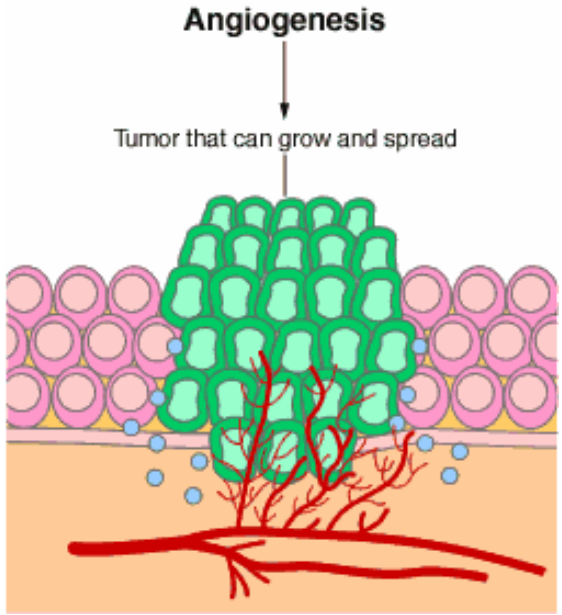
Nature Review Cancer

Tumor growth is dependent on angiogenesis

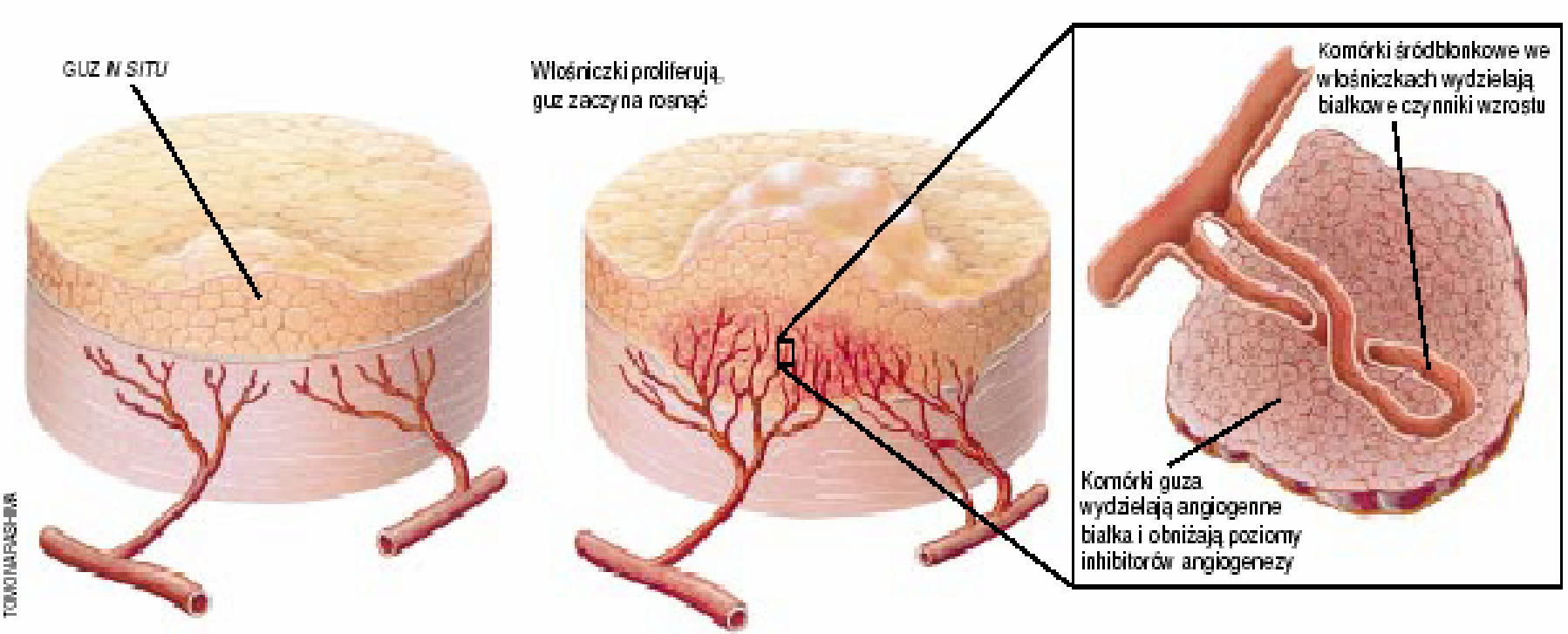




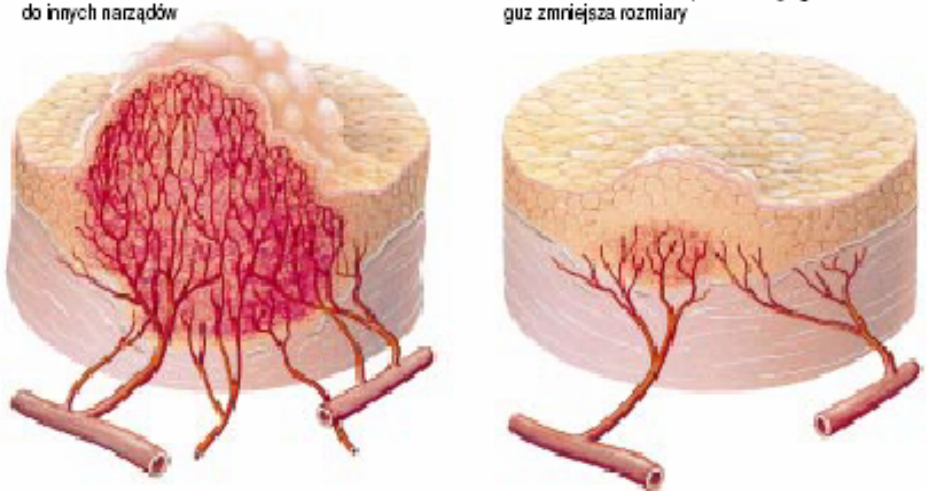




Anti-angiogenic therapy

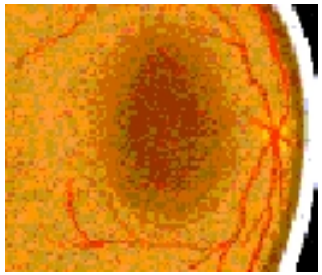


122 ŚWIAT NAUKI *Listopad 1996*

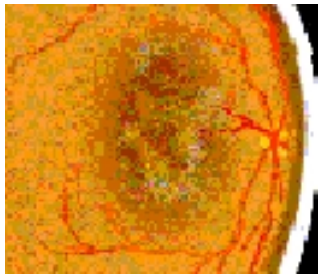


siRNA against mRNA VEGF

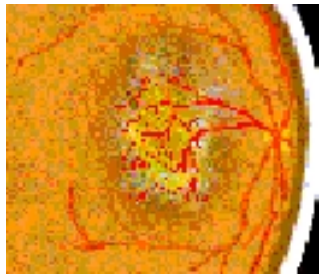
Acuity Pharmaceuticals



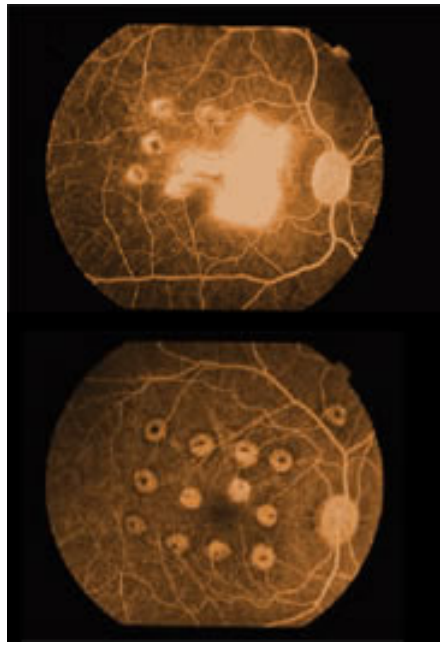
Normal Macula



Dry AMD:Drusen formation under the Macula



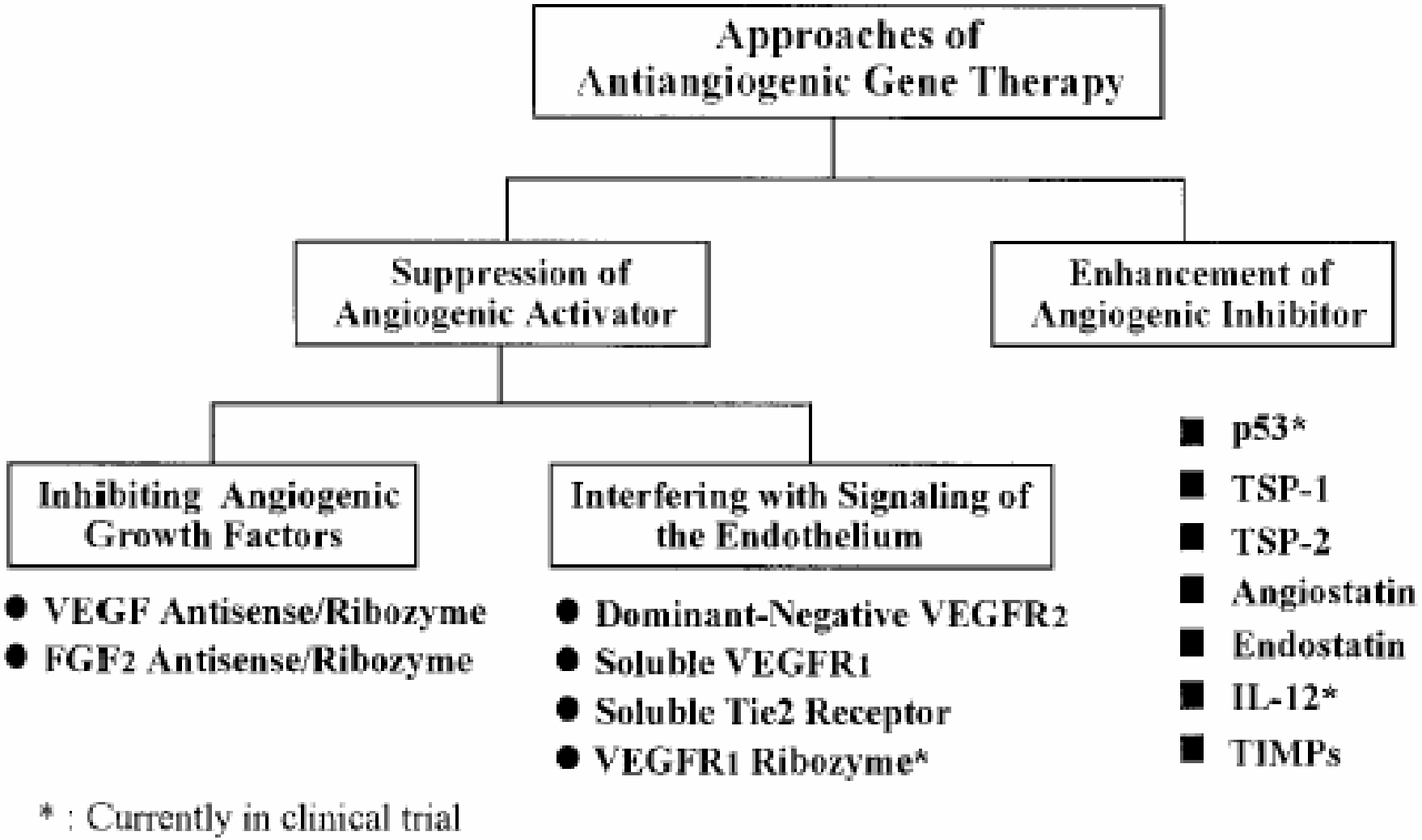
Wet AMD:Macula with abnormal blood vessels



+ siRNA

Carla McCullough, a 79-year-old woman in Cleveland, Ohio - 9 November 2004 – first injection of siRNA

Strategies of anti-angiogenic gene therapy



* : Currently in clinical trial

Gene therapy of cancer: anti-cancer vs anti-angiogenic

Anti-cancer cells- introduction of genes that:

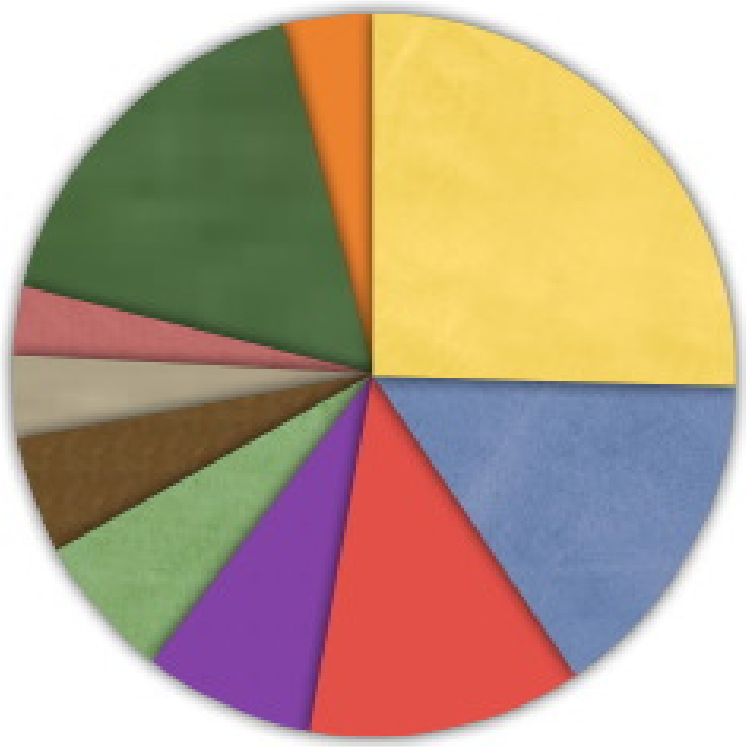
1. Permit tumor cells to express toxic molecules
2. Prevent or correct genetic defects
3. Increase the immunogenecity of tumor cells
4. Increase the sensitivity of tumor cells to drugs

Anti-angiogenic - anti-endothelial cells: introduction of genes that:

1. Specifically target tumor endothelial cells
2. Block tumor-derived angiogenic activity

They may be employed together or separately

Gene Types Transferred in Gene Therapy Clinical Trials



- Cytokine 25% (n=274)
- Antigen 15% (n=162)
- Tumor supressor 12% (n=132)
- Suicide 7.6% (n=82)
- Deficiency 6.5% (n=70)
- Drug resistance 5.2% (n=56)
- Replication inhibitor 3.7% (n=40)
- Receptor 3.1% (n=33)
- Others 18% (n=183)
- N/C 4.1% (n=44)

The Journal of Gene Medicine, © 2005 John Wiley and Sons Ltd

www.wiley.co.uk/genmed/clinical

Gene therapy trials in Poland

Trial ID	Title
PL-001	Gene Therapy of Human Melanoma. Immunization of Patients with Autologous Tumor Cells Admixed with Allogeneic Melanoma Cells Secreting Interleukin 6 and Soluble Interleukin 6 Receptor
PL-001	Gene therapy of vulvar cancer with plasmid vector encoding soluble receptor FLT-1 (psFLT-1)
PL-002	**IGF-I (Insulin like growth factor 1) triple helix cellular therapy of digestive tube tumors**
PL-002	Gene therapy of coronary artery disease with phvegf165
PL-003	**IGF-I (Insulin like growth factor 1) triple helix cellular therapy of brain tumors**
PL-003	Therapeutic angiogenesis with gene therapy using bicistronic VEGF/bFGF plazmid (pVIF) in "no option" patients with refractory coronary artery disease - efficacy and safety evaluation. Double-blind, placebo controlled, clinical study

Gene therapy trials in Poland

PL-002	IGF-I (Insulin like growth factor 1) triple helix cellular therapy of digestive tube tumors

PL-003	IGF-I (Insulin like growth factor 1) triple helix cellular therapy of brain tumors

Jerzy Trojan – was employed in Krakow and in Bydgoszcz; He claimed that it is possible to cure children from glioma using the IGF-triplex helix

The contract with him in Krakow was terminated; still is present at the web page of Collegium Medicum in Bydgoszcz

[Usefulness of the antisense and triplex anti-IGF-1 techniques for postoperative cellular gene therapy of malignant gliomas expressing IGF-1.] [Article in Polish]

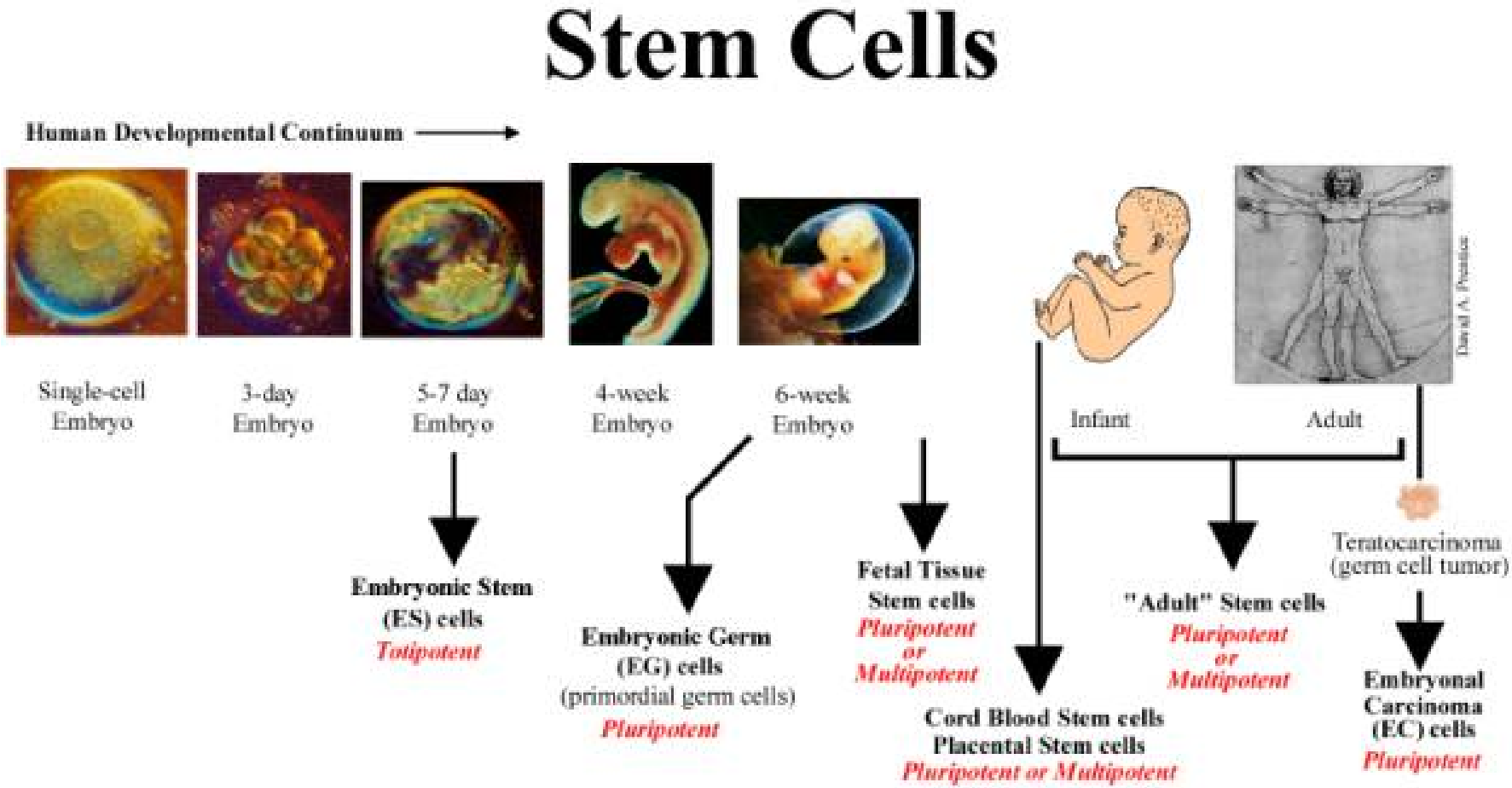
Neurol Neurochir Pol. 2006 Nov-Dec;40(6):509-15

Kasprzak HA, Trojan J, Bierwagen M, Kopinski P, Jarocki P, Bartczak K, Czapiewski J ul. Wyspianskiego 8, 85-073 Bydgoszcz, tel. +48 602 237 978.

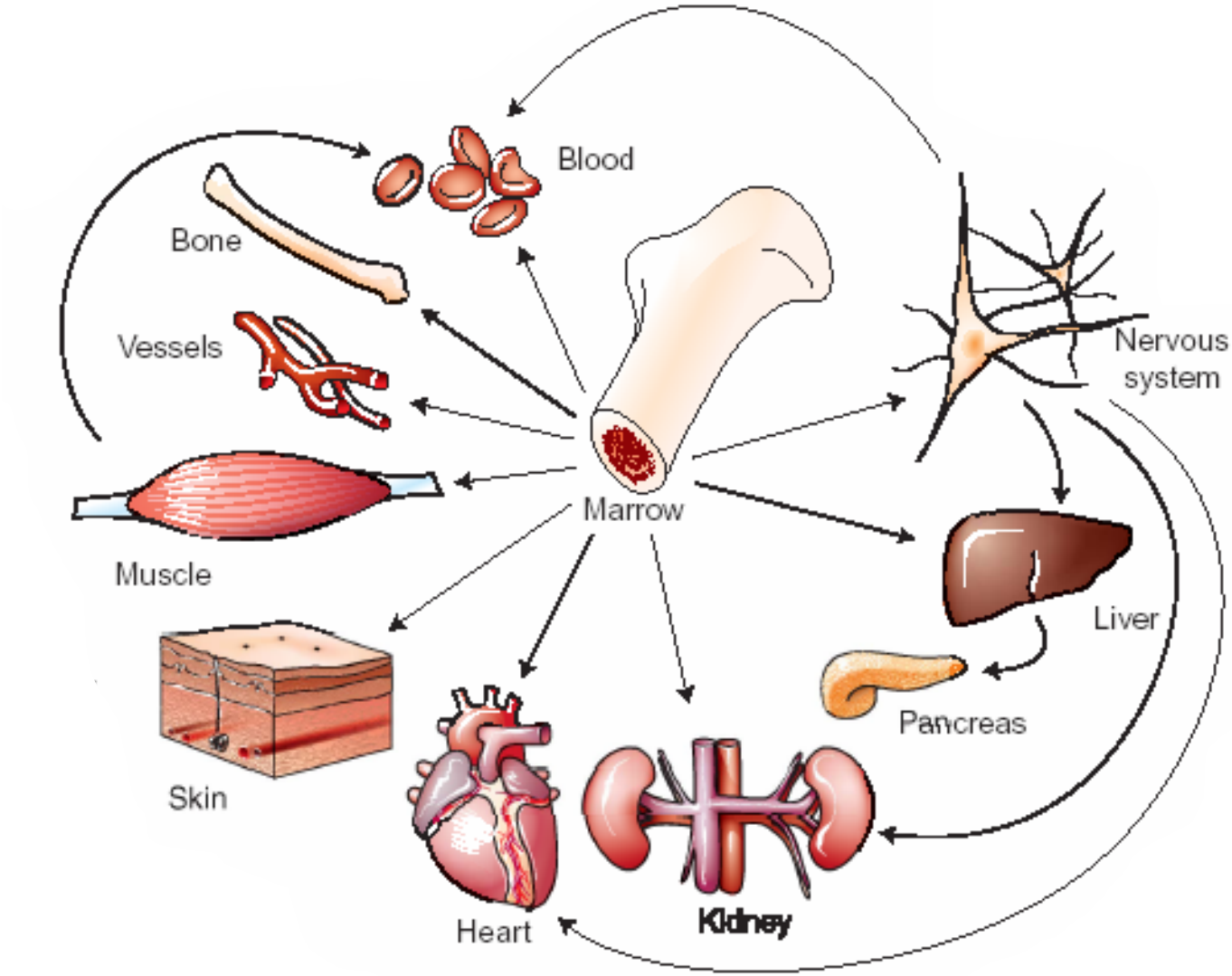
Background and purpose: The aim of the study was to estimate the usefulness of the antineoplastic vaccination in treatment of malignant brain tumors. According to medical knowledge there is no cure for this kind of tumors. Material and methods: Between 2001 and 2005, ten patients suffering from malignant glial tumors were treated. There were 5 male and 5 female individuals, aged from 17 to 76 (mean age: 40.8 years). The histopathological examination showed 4 cases of glioblastoma and 6 cases of anaplastic astrocytoma. Initially, patients were operated on with dissection of 1 cm3 of the most representative part of tumor. The neoplasm cells were cultured, transfected with episomal pMT EP vector (expressing alternatively oligonucleotide sequence forming triple helix with IGF-I gene or antisense against IGF-1 mRNA), re-cultured, irradiated and resuspended in medium to prepare antineoplastic vaccine. The patients were vaccinated subcutaneously. We examined peripheral blood lymphocyte subsets to assess the immunological response of the patients. **Results**: We observed prolongation of the survival time to 21.7 months compared to 9-11 months observed in literature. The patients were additionally treated oncologically with radiotherapy and chemotherapy (temozolomide) according to the reasonable indications. Therefore, the comprehensive assessment of the genotherapy as the supplemental monotherapy was impossible. **Conclusions**: The method of treatment used in this study prolongs the survival time of patients with high-grade gliomas of the central nervous system. This gene therapy needs further investigations as a method of oncological monotherapy of brain malignant gliomas.

Future of gene therapy

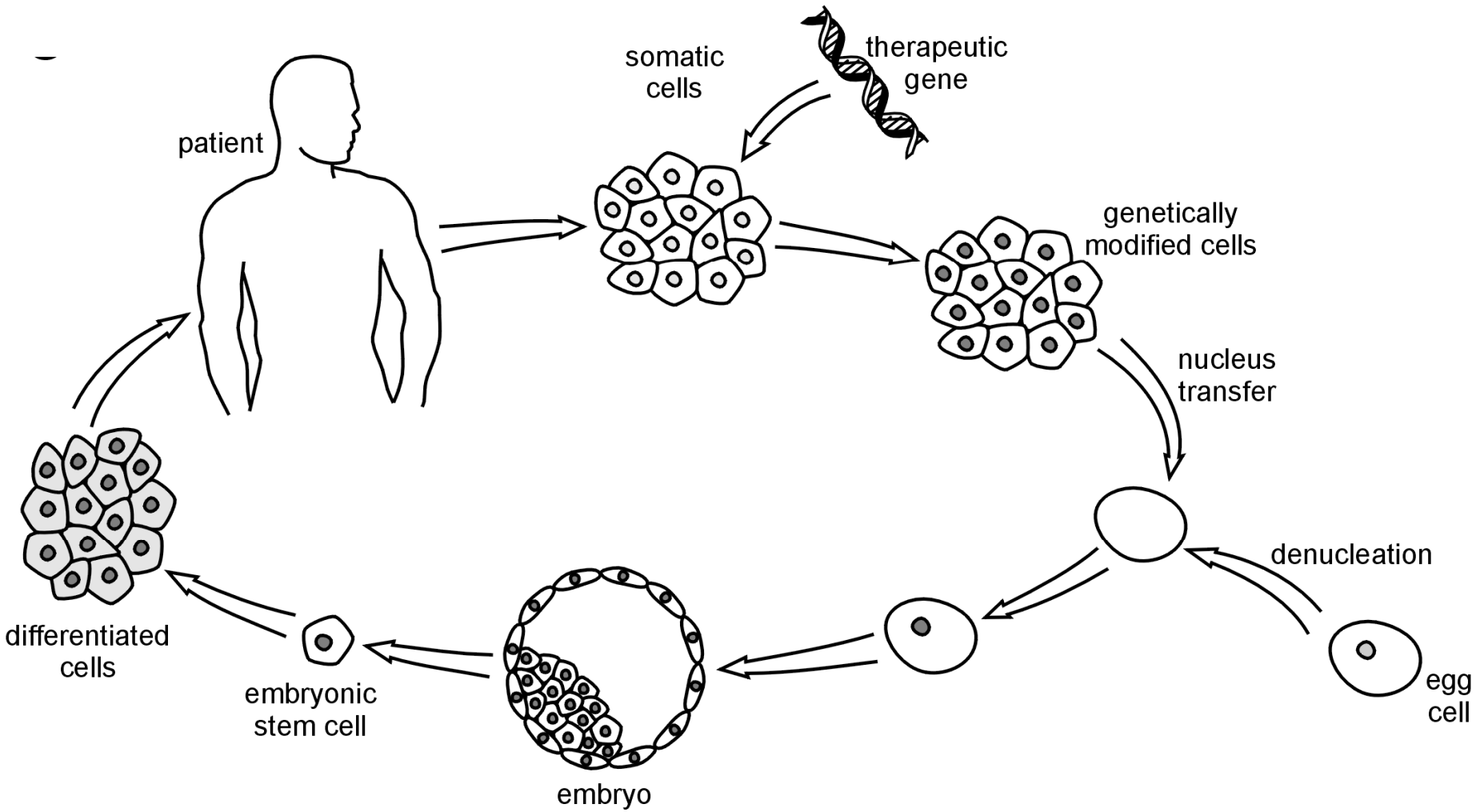
Gene therapy vs cell therapy



Adult stem cells

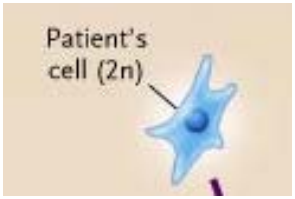


Somatic cell nuclear transfer and gene therapy

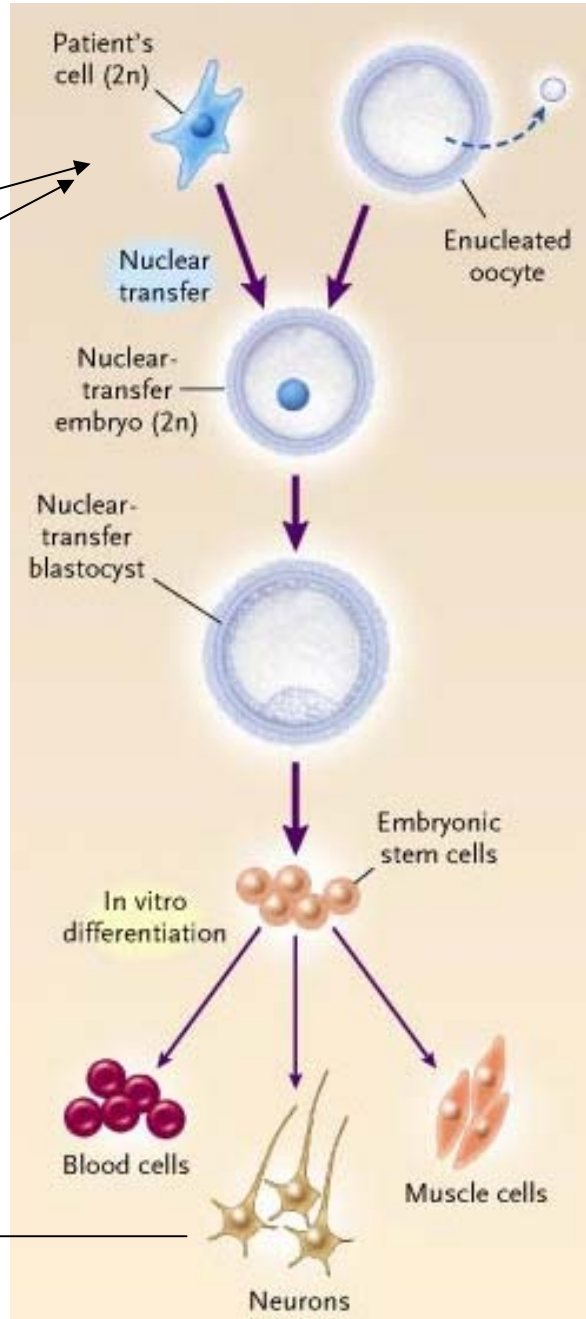


Somatic cell nuclear transfer and gene therapy

Eg, cells of the boy
with Lesh-Nyhan syndrome



Correct HPRT gene



Cure?

implantation of autologous
(embryonic stem cells derived)
neural celsl

Summary

Problems of gene therapy

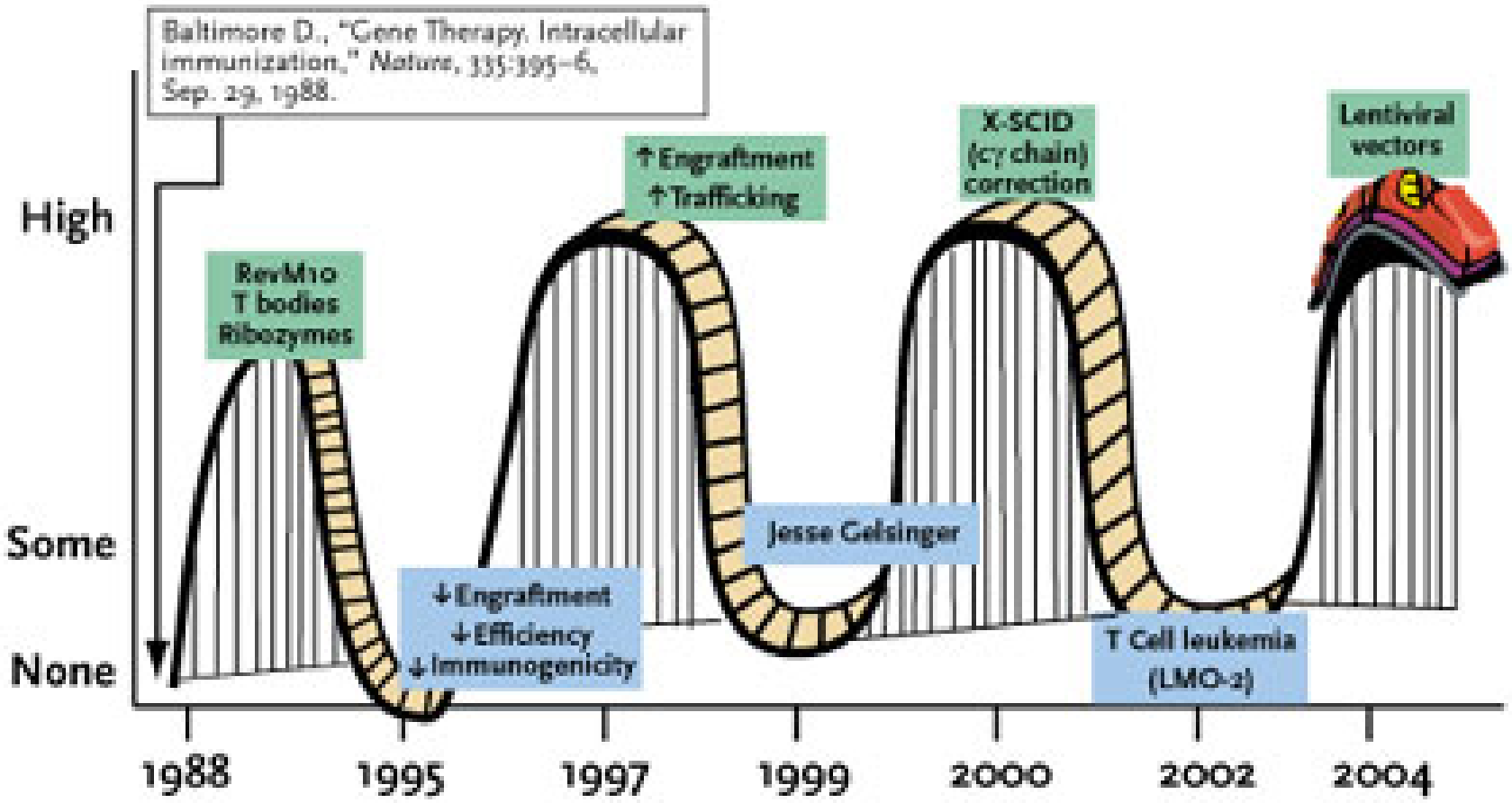
1. Efficacy vs safety
   - vector choice
   - target choice

2. Bioethical problems?

   a) embryonic progenitor cells in gene therapy – so called therapeutic cloning (experimental nuclear transfer)
   b) gene therapy of germ line
   c) „cosmetic” gene therapy/gene doping
   d) quality of life vs gene therapy
   e) costs of gene therapy

3.Commersialisation

Gene Therapy's Fall and Rise (Again)



http://www.the-scientist.com/yr2004/sep/research_040927.html

The Scientist, Sept 27, 2004

Gene therapy can cure the disease not only in experimental animals but also in human – SCID trials

Gene therapy will never be a simple method effective in treatment of every disease

Treatment of diseases by means of gene therapy is a fascinating idea, but scientists have to work a lot before it will be fully implemented and before all hopes connected with it will be realized

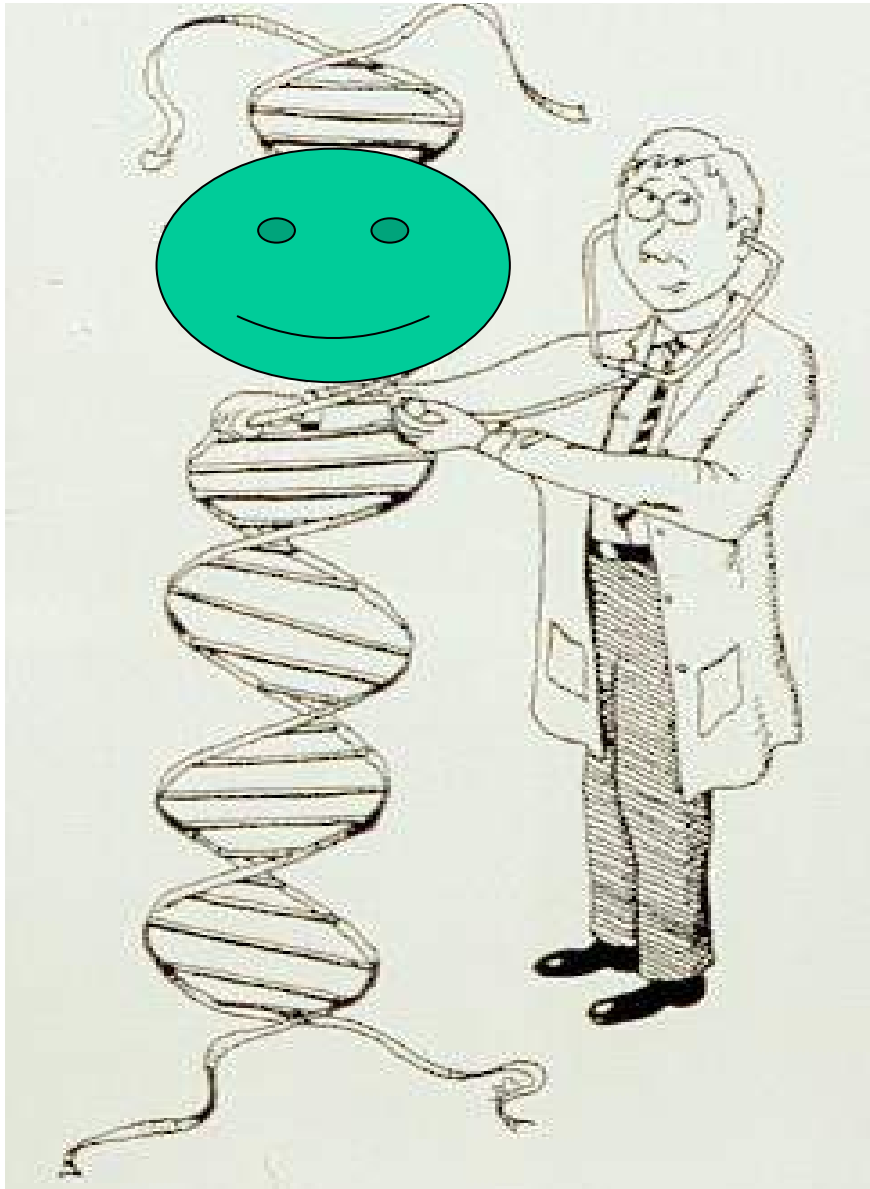
Theodore Friedmann, 1997

The difficulty is making it work..."

Ronald Crystal

"It's too easy to forget the incredible progress that has been made and to say, 'Well, why haven't we cured all these diseases?' " says Daniel Salomon, an associate professor at The Scripps Research Institute in La Jolla, Calif. "But when you're pioneering a new field, there are going to be advances and setbacks that are going to frustrate both the scientists and the public.

Society cannot expect the immediate success of every approach and scientists and physicians may not generate hype and promise effective treatment when it is not yet available



THANK YOU FOR YOUR ATTENTION

GOOD LUCK AT EXAM!

29th January - only Socrates students

10th February, Saturday

9.00 am

Spring semester course

Molecular mechanisms of angiogenesis

First lecture

Monday, 26th February 2006

3 – 5 pm, room D104

7 lectures (15 hours)
30 h practical course (limited number of participants!)