

IPT-FACT OR FICTION

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George 16 September 2009

Absorption of glucose by cancer cells is the basis of PET scans

This confirms that there is an important difference in glucose metabolism between cancer and normal cells.



1) PET

2) CT

3) fused PET/CT

These scans were taken in April 2007, before the patient underwent chemotherapy.



4) PET

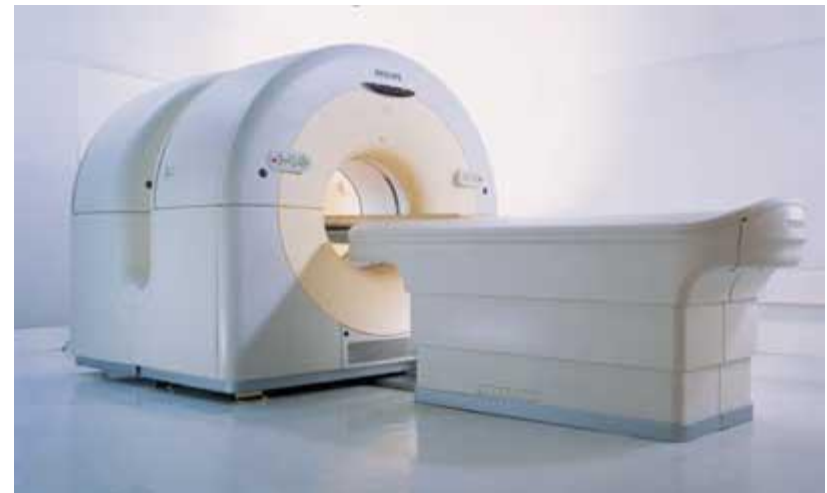
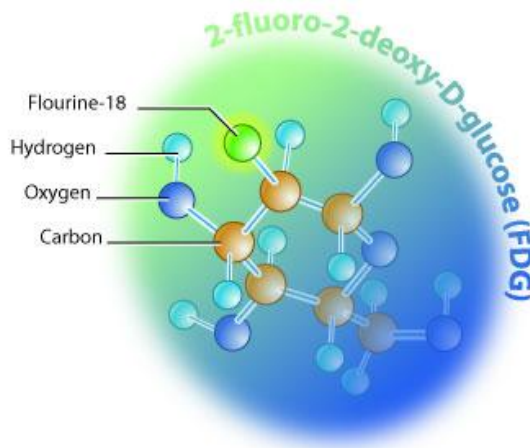
5) CT

6) fused PET/CT

These scans were taken in July 2007, after chemotherapy.

How PET works

Positron emission tomography, PET, is currently accepted to be the most accurate way to stage and monitor many types of cancer. PET is a Nuclear Medicine imaging technique that uses very short-lived radioactive compounds that localize in cancer cells. Recent advances in molecular biology research have identified many abnormalities of cellular function in tumors. **Cancer cells have alterations in the normal metabolism of the sugar, glucose, and have increased glucose uptake and decreased glucose clearance.** Glucose can be bound to the positron 18-F to make a compound 18 F-fluorodeoxyglucose, FDG. When injected into a patient's blood stream, FDG is taken up by the cancer cells at a more rapid rate than normal cells and allows cancers to be seen as “hot spots” on the PET scan.



WHAT IS IPT?

- IPT (Insulin Potentiation Therapy) Low Dose Chemotherapy is an alternative cancer therapy which is now available in South Africa for patients who don't have the courage to submit themselves to the side effects of conventional cancer therapy. (This is the definition used by Dr Pretorius on his website - <http://www.camaa.co.za/index.html>).
- It is promoted in South Africa by Dr Eugene Pretorius of Centurion.



This technique was developed in Mexico by Dr. Donato Perez Garcia the 1st more than 65 years ago.

When the technique of IPT is being utilized a patient's blood sugar is lowered to a desired therapeutic level through the administration of an individually calculated dose of insulin.

Chemotherapy (the combination of chemotherapeutic agents as prescribed by the National Cancer Institute of America for the specific type of cancer being treated) is then administered in lower than conventional dosages according to the IPT protocol.

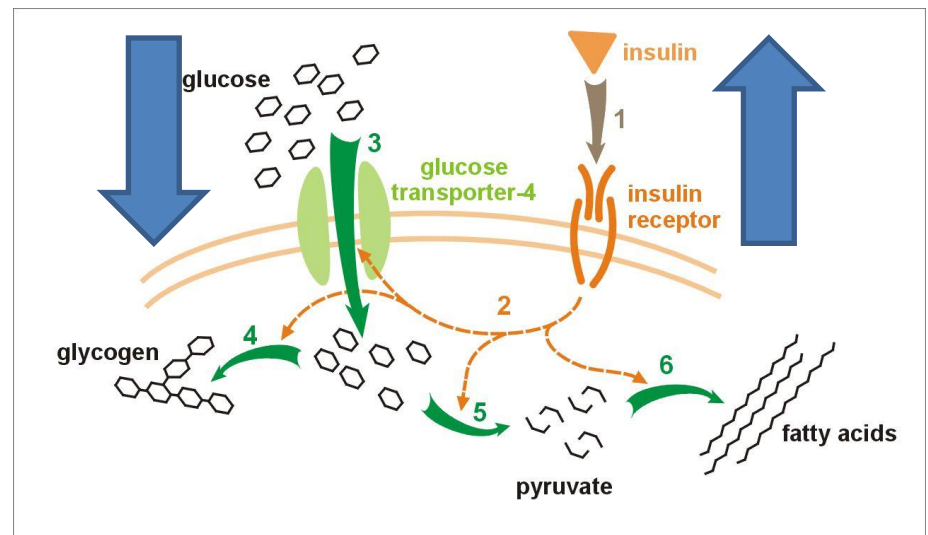
Thereafter the patient's blood sugar is reversed back to normal through the administration of intravenous glucose.

<http://www.camaa.co.za/index.html>

How does it work?

All cancer cells grow on glucose. For every receptor (channel) which allows sugar to pass into a normal cell, a cancer cell has between 16 and 20. When the blood sugar is lowered, cancer cells are thus starved 16x to 20x more than normal cells. Under these circumstances we get that most of the chemotherapeutic agents can concentrate up to 10,000x more in cancer cells than in the rest of the body. We thus achieve a targeted attack on the cancer cells without damaging the normal body too much, especially the immune system.

<http://www.camaa.co.za/index.html>



What are the benefits of IPT?

Many patients with different types of cancer in different stages had **tumours disappear or shrink dramatically** after a course of IPT therapy. In many instances one can thus achieve anti-cancer results without having to lose limbs or organs. This statement is based on 65 years of anecdotal evidence in the practicing of IPT in Mexico, the USA and now in South Africa.

Minimal side-effects. Clinical experience has shown that the side-effects of IPT Low Dose Chemotherapy is minimal. Hair loss is minimal if any, nausea is minimal and energy levels tend to return back to normal usually day 2 after therapy. The patient can thus continue with his or her normal life without being bedridden for days during therapy

<http://www.camaa.co.za/index.html>

Med Hypotheses. 1986 Jun;20(2):199-210

Insulin potentiation therapy: a new concept in the management of chronic degenerative disease.

[Ayre SG, Perez Garcia y Bellon D, Perez Garcia D Jr.](#)

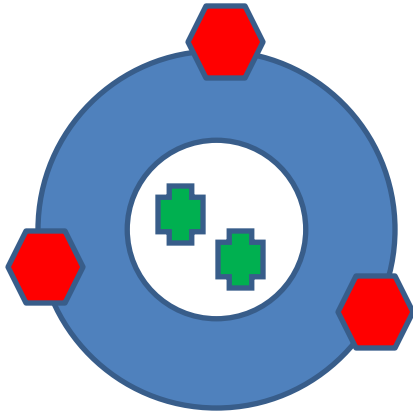
In insulin potentiation therapy the hormone insulin is used as an adjunct in the medical management of the chronic degenerative diseases including malignant neoplasia. In this, the recognized physiological action of insulin--that of increasing cell membrane permeability--is taken advantage of to potentiate the pharmacological actions of medications administered concurrently in the therapy. This potentiation occurs because of the heretofore unrecognized applicability of this membrane permeabilizing effect of insulin to a much wider range of tissues than is classically accepted, and further the observed effect of this permeabilizing phenomenon as it relates to drug molecules, most importantly the antineoplastic agents. The historical context of insulin potentiation therapy is described, and scientific corroboration for its novel hypotheses is given. Insulin potentiation therapy represents a potentially revolutionary concept in the medical management of diseases and is, in the authors' opinion, deserving of intensive scientific investigation through in vitro and in vivo experimentation and properly conducted human clinical trials in a university teaching hospital setting.

(This is the only peer-reviewed paper on IPT found out of 20 million abstracts in Pubmed.)

What makes cancer cells different

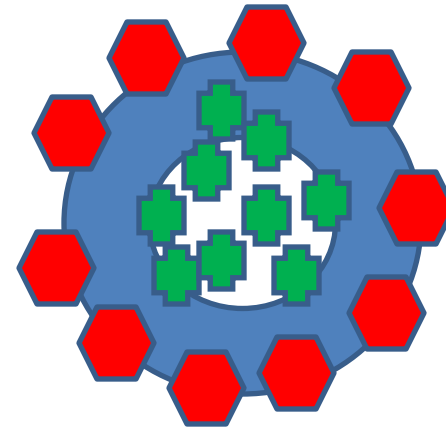
<http://www.camaa.co.za/index.html>

NORMAL CELL



CANCER CELL

- Increased glucose receptors (red) on membrane (proven). Increased uptake of anti-cancer drugs (not proven).



CANSA reacts



MEDIA RELEASE FROM THE CANCER ASSOCIATION OF SOUTH AFRICA

Tuesday 14 February 2006

Att: News Editors

For immediate release

CANSA warns against Insulin Potentiation Therapy (IPT) for cancer patients

The Cancer Association of South Africa (CANSA) is warning cancer patients about false promises that have been made about so-called Insulin Potentiation Therapy (IPT). During the past few weeks there has been a campaign, especially using the radio, to present IPT as a breakthrough in cancer therapy. Some desperate cancer patients have grasped at this treatment, especially when conventional treatment has failed to arrest the growth of their cancers.

“CANSA researchers have looked into IPT and discovered that it is not a new breakthrough but was developed in Mexico in the 1930s,” said Dr Carl Albrecht, CANSA Research Advocate. “Since then there has not been a single published report in a peer-reviewed journal of a clinical trial showing any tumour regression or complete response (disappearance). The best that was found was a reported 7 percent decrease in tumour growth or 4 millimeters less growth over 49 days relative to a tumour of 61 millimeters in diameter compared to controls. The bottomline is that the tumours continued to grow in this study and no cures, or even partial responses (50 percent decrease) have ever been published in reputable journals. **These results are of no clinical significance and IPT has not been proven as a treatment for cancer**”, Dr Albrecht said.

“The treatment is based on the theory that cancer cells need and absorb glucose more than normal cells and that by injecting a cancer patient with sufficient insulin, cancer cells could be made far more sensitive to chemotherapy. Consequently far less chemotherapy would be required and cancers could be eradicated more effectively. If this were true, there would be world-wide excitement around this approach, however, it is reported in only one publication in 1981 and the idea has not progressed since. It is certainly no breakthrough”, Dr Albrecht said.

“Furthermore it was reported that 10 percent of the IPT-treated patients showed low blood sugar symptoms within 20 minutes of treatment and had to receive glucose injections. Doctors have warned that this treatment is potentially lethal because insulin can cause a rapidly falling blood sugar level, which can produce coma, shock, stroke, and even death”, Dr Albrecht said.

The Mayo Clinic in the United States has referred to IPT as being a dangerous alternative cancer treatment and has stressed that there is absolutely no scientific evidence that it works or is safe. The world famous Memorial Sloan-Kettering Cancer Center has referred to IPT as being unproven and questionable with potentially lethal side-effects. A South African cancer patient enquired about the IPT treatment and was told that he would need to go to Mexico for treatment which would cost R36 000 a month and in his case could last up to three years. The patient remarked that he would “ have to sell a farm to cover these costs “. After being shown the facts concerning IPT he opted for conventional therapy in South Africa.

The representative association of oncologists in South Africa has expressed support for CANSA and the data presented.

CANSA is warning all cancer patients to stay away from Insulin Potentiation Therapy.

Literature search September 2009:

Literature search shows almost complete lack of IPT publications but an explosion of publications on Insulin-like Growth Factor (IGF) receptor inhibition.

What is going on?

Modern version of IPT

Clinical Development of Inhibitors of the Insulin-like Growth Factor Receptor in Oncology.

[Gualberto A.](#), [Pollak M.](#)

Pfizer Oncology, 50 Pequot Ave. MS6025-A3266, New London, CT 06320, USA. antonio.gualberto@pfizer.com.

Curr Drug Targets. 2009 Oct 1. [Epub ahead of print]

The insulin-like growth factor I receptor (IGF-IR) pathway plays a major role in cancer growth, tumor cell survival and resistance to therapy. Ancillary evidence that targeting the IGF-IR may be useful in the treatment of cancer has been accumulating for almost two decades. Today, more than two dozen compounds have been developed and clinical trials are underway for at least 12 of those. The ability to pharmacologically control the IGF-IR pathway holds not only promising therapeutic implications but also the possibility to gather a better understanding of the role of the IGF axis in tumor initiation and progression. This review focuses on the preclinical rationale for targeting the IGF-IR and other components of the IGF-I system, early clinical results observed to date, biomarker approaches employed and the lessons from these early results for future study design. Early clinical trials reveal an acceptable safety profile together with pharmacodynamic evidence of receptor targeting. Instances of single-agent activity during phase I evaluations have been well documented and a recently reported randomized phase II study indicates that co-administration of an anti-IGF-IR antibody with chemotherapy improves objective response rate and progression-free survival in non-small cell lung cancer patients. These early results support ongoing research across a broad range of cancer indications.

This work shows that by blocking the receptors of IGF, cancer cell growth can be inhibited.

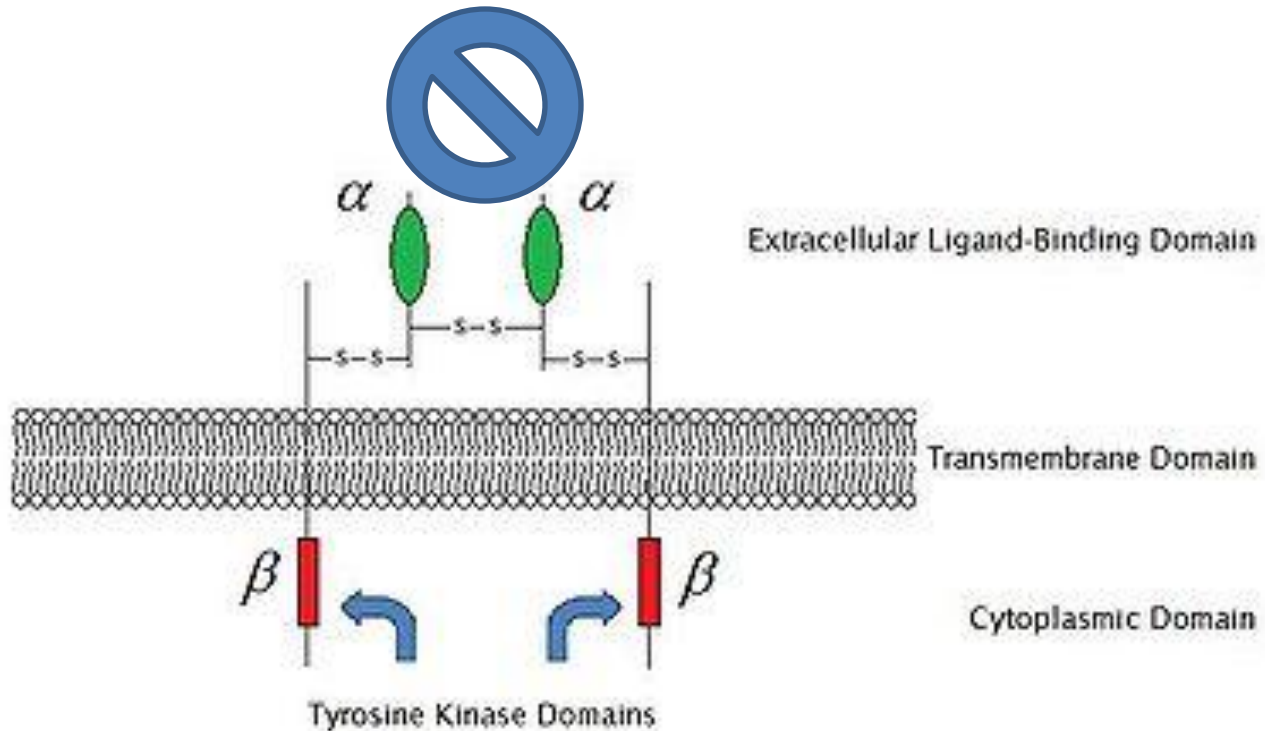
IGF production is stimulated by insulin

IGF protects cancer cells against the effects of anti-cancer drugs

Thus insulin could stimulate cancer indirectly by stimulating IGF

Thus IPT could stimulate cancer . This would be paradoxical!

AIM IS TO BLOCK IGF-IR



Emerging role of insulin-like growth factor receptor inhibitors in oncology: early clinical trial results and future directions.

[Gualberto A](#), [Pollak M](#).

Oncogene. 2009 Aug 27;28(34):3009-21. Epub 2009 Jul 6. Review

Clinical Department, Pfizer Oncology, New London, CT, USA.

Preclinical evidence that targeting the insulin-like growth factor receptor (IGF-IR) is effective in cancer treatment has been accumulating for almost two decades. Efforts to develop drugs began in the late 1990s, and initial data from clinical trials were reported in 2006. The biological rationale for IGF-IR targeting has potential relevance to many tumor types, and early results have justified expanded programs to evaluate IGF-IR-targeting agents in many areas of clinical need. More than two dozen drug candidates have been developed and clinical trials are underway for at least 12 of these. Early clinical trials reveal an acceptable safety profile together with pharmacodynamic evidence that the receptor can be successfully targeted. It is premature to draw conclusions regarding efficacy, but well-documented instances of single-agent activity were noted during phase I evaluations, and recent evidence from a phase II study suggests that co-administration of an anti-IGF-IR antibody with chemotherapy for non-small-cell lung cancer improves objective response rate and progression-free survival. With more than 70 trials involving a variety of drug candidates underway, the IGF-IR is becoming one of the most intensively investigated molecular targets in oncology. Early results justify the continuation of ongoing research across a broad range of cancer indications.

Modern clinical trials

: J Thorac Oncol. 2009 Sep 10. [Epub ahead of print

Safety, Pharmacokinetics, and Pharmacodynamics of the Insulin-Like Growth Factor Type 1 Receptor Inhibitor Figitumumab (CP-751,871) in Combination with Paclitaxel and Carboplatin.

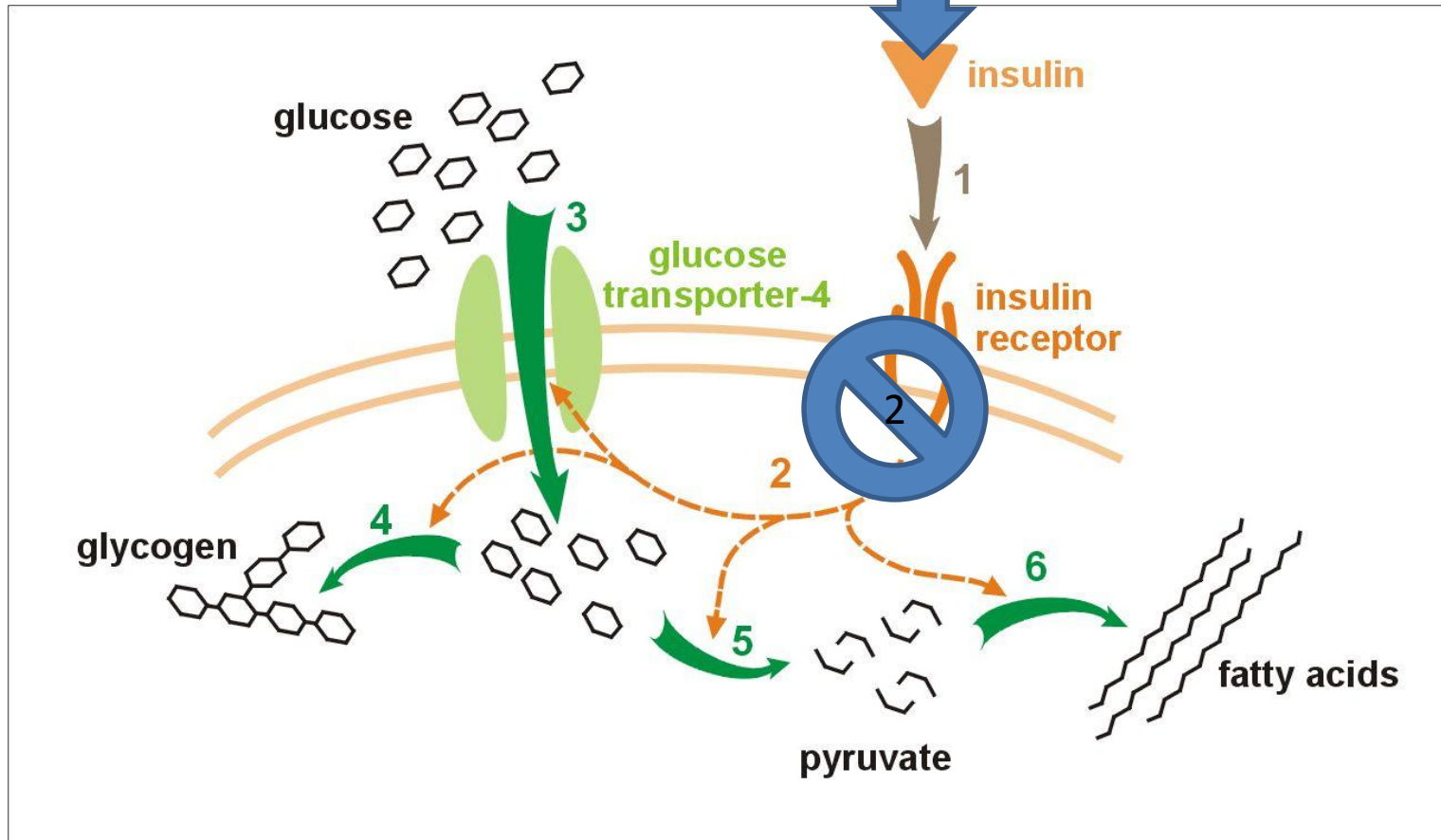
[Karp DD](#), [Pollak MN](#), [Cohen RB](#), [Eisenberg PD](#), [Haluska P](#), [Yin D](#), [Lipton A](#), [Demers L](#), [Leitzel K](#), [Hixon ML](#), [Terstappen LW](#), [Garland L](#), [Paz-Ares LG](#), [Cardenal F](#), [Langer CJ](#), [Gualberto A](#).

*The University of Texas M. D. Anderson Cancer Center, Houston, Texas;

INTRODUCTION:: This phase 1 study was conducted to determine the recommended phase 2 dose of the selective insulin-like growth factor type 1 receptor (IGF-1R) inhibitor figitumumab (F, CP-751,871) given in combination with paclitaxel and carboplatin in patients with advanced solid tumors. **METHODS::** Patients received paclitaxel 200 mg/m², carboplatin (area under the curve of 6), and F (0.05-20 mg/kg) q3 weeks for up to six cycles. Patients with objective response or stable disease were eligible to receive additional cycles of single agent F until disease progression. Safety, efficacy, pharmacokinetic, and pharmacodynamic endpoints were investigated. **RESULTS::** Forty-two patients, including 35 with stages IIIB and IV non-small cell lung cancer (NSCLC), were enrolled in eight dose escalation cohorts. A maximum tolerated dose was not identified. Severe adverse events possibly related to F included fatigue, diarrhea, hyperglycemia, gamma glutamyl transpeptidase elevation, and thrombocytopenia (one case each). F plasma exposure parameters increased with dose. Fifteen objective responses (RECIST) were reported, including two complete responses in NSCLC and ovarian carcinoma. Notably, levels of bioactive IGF-1 seemed to influence response to treatment with objective responses in patients with a high baseline-free IGF-1 to IGF binding protein-3 ratio seen only in the 10 and 20 mg/kg dosing cohorts. **CONCLUSIONS::** F was well tolerated in combination with paclitaxel and carboplatin. Based on its favorable safety, pharmacokinetic, and pharmacodynamic properties, the maximal feasible dose of 20 mg/kg has been selected for further investigation

OPPOSING THERAPIES:

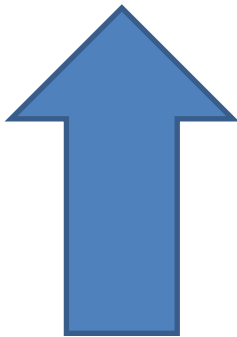
1. IPT INCREASES INSULIN
2. ANTI-IGF-IR INHIBITS INSULIN-LIKE EFFECTS.



Difference between IPT and IGF-Inhibition

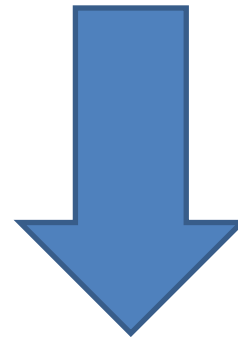
IPT

- Increases insulin systemically



IGF-IR

- Inhibits insulin-like growth factor signals going into cancer cells



IPT is Outdated

IPT

- Insulin injected
- IGF not involved
- IGF – IR not involved
- IGF- IR inhibitors not involved
- No information of any clinical trials
- Diluted chemotherapy used
- No positive clinical results published

Modern Version

- Insulin not used
- IGF involved
- IGF-IR involved
- IGF-IR inhibitors involved
- 70 clinical trials in progress
- IGF-IR is most investigated target
- Anti-IGF-IR monoclonal antibodies are the drug and potentiate chemotherapy
- Positive clinical results published

Reasons for Rejecting IPT

- 1. Lack of peer-reviewed, published, proof that it works in humans. No academic status.
- 2. Lack of a cohesive foundation of peer-reviewed publications on mode of action, efficacy and toxicology. Not recognised as a tried and tested medical procedure.
- 3. Has exactly opposite mode of action compared to highly promising modern anti-IGF-IR antagonists, i.e. stimulates production of IGF via insulin as opposed to inhibiting its action.
- 4. Could inhibit the working of anti-cancer drugs by stimulating the production of IGF which makes cancer cells resistant to chemotherapeutic drugs, i.e. tumour promotion rather than tumour eradication. This means that IPT could cause patients to end up worse off.
- 4. Unsubstantiated promises
- 5. Potentially dangerous (insulin-induced hypoglycemic coma)
- 6. Not recognised by Medical Aids
- 7. Not recognised by oncologists
- 8. Obvious lack of support by healed patients in the media. Surely cured patients would step forward to proclaim a cancer-curing treatment? Why not?
- 9. Expensive
- 10. Shows the hallmarks of cancer quackery.

Insulin potentiation therapy: A dangerous alternative cancer treatment

Insulin potentiation therapy: A dangerous alternative cancer treatment

Question

What does Mayo Clinic think about insulin potentiation therapy as a treatment for cancer?

Answer

Insulin potentiation therapy (IPT) is an unproven and dangerous alternative cancer treatment. It's based on the theory that injected insulin increases the therapeutic effects of chemotherapy drugs so that lower doses are needed. Proponents claim that IPT is an effective cancer treatment that also dramatically reduces the adverse side effects of chemotherapy. **However, no clinical trials have been done to validate those claims.**

Advocates of insulin potentiation therapy believe that cancer cells consume more sugar (glucose) than healthy cells do and as a result are more sensitive to insulin. IPT is performed by injecting insulin in the arm, followed by an injection of chemotherapy drugs. The insulin causes low blood sugar (hypoglycemia), which supposedly makes cancer cells more susceptible to chemotherapy drugs. **However, this is unproved.** Also, if too much insulin is given, it can cause dangerously low levels of blood sugar, which can result in seizures, coma, shock, stroke and even death. While insulin potentiation therapy has been used for decades in Mexico and some other countries, there **is absolutely no scientific evidence that it works or is safe.**