**Alternative Methods of Chemotherapy *(Including Warburg etc.)***

***please read https://medlineplus.gov/cancerchemotherapy.html (MedlinePlus)***

***and other web sites that have explanations of how chemotherapy cures cancer.***

**DNA damage by cyclophosphamide, DNA base analogs, Daunorubicin (antibiotic), X-rays, etc.**

***How do these chemicals kill cancer cells?***

[a] Because **cancer cells expect & depend on fast growth** (are disturbed if slowed?)

[b] Because **slowing copying of DNA creates imbalances** between cell growth versus DNA copying?

[c] Because **more abnormal DNA bases get incorporated** into faster growing cells.

[d] Faster DNA copying creates mistakes, when combined with chemical interference

[e] Because **defective cell cycle checkpoints fail to delay DNA copying** until DNA is repaired?

(For "e" notice that lack of slowing DNA copying is what kills cancer cells!)

Should we think of oncogenes as weakening controls on the mitotic cell cycle?

Would your treatment strategy be different, depending on which of the above ([a] through [e]) is true?

**Mitotic Spindle Damage**: vinblastine, vincristine, taxol (**But not colchicine**, although it does prevent mitosis)

[f] "Expect"

[g] "Imbalances"

[h] "Mitosis distributes chromosomes unequally, creating **aneuploidy**"

[i] "Checkpoint controls fail to delay mitosis until the mitotic spindle is organized perfectly?

What difference in effects of vinblastine versus colchicine results in only the former harming cancer cells selectively?

**Any chemical that inhibits either mitosis or DNA synthesis gets tested as a potential cancer chemotherapy drug.**

**Cancer cells have many other abnormalities** besides rates of division and growth, and some cancers DON'T grow rapidly!) but (until very recently) nobody tested chemicals that inhibit the **Warburg Effect**, or that target **weakened cell contractility**, or selectively harm those cells having **reduced contact inhibition**, or that have **less anchorage dependence.**

**Please invent strategies for selectively harming cells that have one or more of these other abnormalities.**

**http://www.sciencedirect.com/science/article/pii/0014482788900249**

**Fibroblast contractility and actin organization are stimulated by microtubule inhibitors**

B.A. Danowski Journal of Cell Science 1989 93: 255-266; **http://jcs.biologists.org/content/93/2/255.short**

**Please read the American Cancer Society's informative web site**: ***Chemotherapy Drugs: How They Work***

http://www.cancer.org/acs/groups/cid/documents/webcontent/002995-pdf.pdf

Try to find any places in this article where they try to explain how to make chemotherapy chemicals kill cancer cells more than they kill normal body cells. ***Perhaps they assume that greater toxicity automatically results from fast growth***?

**Please imagine inventing a drug** that somehow **protects non-cancerous cells** from being killed by some or all cancer chemotherapy drugs, **but do NOT protect actual cancer cells** from being killed by chemotherapy drugs.

Would such a selectively protective drug be useful for treatment.

**Stimulants, & "de-inhibitors of the immune system? A recently developed monoclonal antibody.**

***Please search on the internet for one or more examples of such a treatment.***