Oxidizing Cancer Cells to Death A Simple, Highly-Effective and Low-Cost Universal Cure

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Introduction

The main challenge in treating cancer is killing cancer cells without causing harm to normal cells. Chemotherapy and radiation are highly limited by toxicity to normal cells and tissues. Is modern oncology decades behind the science that has illuminated new targets for cancer therapy? It certainly seems so.

Outlined here is a method for treating cancer that directly targets a key vulnerability within cancer cells. Utilizing this approach has yielded extraordinary responses without any systemic toxicity or adverse side effects. This has been accomplished without pharmaceutical agents, only natural nontoxic nutritional supplements and a unique dietary intervention. This method is highly targeted and directly focused at on exploiting the number one weakness or Achilles heel of cancer.

What if there is a universal way to kill cancer cells that is applicable to all forms and stages of cancer? And what if this method is completely nontoxic and harmless towards normal healthy cells? Have the clues been there for decades but ignored because cancer research is centered on high profit drug development? Has the profit driven pharmaceutical and medical system failed us in the pursuit of safe, effective and cost-effective cancer therapies? It certainly appears to be the case.

A major limitation of drug development is that only one single drug or drug component is evaluated, tested and approved at a time. Development of multi-targeted drugs or combinations of drugs is highly limited. FDA approves one drug at a time and then sometimes approves combinations of drugs. All pharmaceutical drugs are associated with adverse side effects, toxicity and widespread negative consequences.

The most significant roadblock in finding a cure for cancer is the complete dismissal of nutritional interventions. Cancer cells have unique nutritional requirements that can be targeted therapeutically. We know with certainty that diet plays a critical role in cancer prevention. Nutrition is the solution to the prevention and treatment of every degenerative disease. Cancer is no exception.

This paper will explain the science behind an innovative and revolutionary approach to treating cancer that is highly disruptive and could potentially become the standard of care in naturopathic and integrative oncology.

A New Frontier in Cancer Research

Exciting new research is focused on a difference between normal cells and cancer cells that enables selectively killing cancer cells while causing no harm to normal cells. This vulnerability of cancer cells involves oxidative stress (ROS-reactive oxygen species) which is much higher in cancer cells than normal cells. In reality, cancer cells are surviving near the edge of self destruction. Targeting this vulnerability in cancer cells represents a universal treatment approach for all forms and stages of cancer.

Cancer cells exhibit altered energy metabolism which is caused by dysfunctional mitochondria. Altered metabolism leads to the generation of an excess of reactive oxygen species (ROS). In a sense, cancer cells are carrying within a lethal oxidative time bomb. Unleashing the internal time bomb can be accomplished by disabling cancer cell's antioxidant defense systems.

Oxidative stress can be induced directly or through disabling antioxidant defense systems. Since cancer cells have an inherently high level of oxidative stress, simply disabling antioxidant defense systems causes cancer cells to be overloaded and oxidized to death. Apoptosis and ferroptosis are forms of regulated cell death that are fully reliant on oxidative stress overload.

Chemotherapeutic drugs kill cancer cells primarily through oxidation overload. DNA damage is secondary. Cancer cells become resistant to chemotherapy by an upregulation of antioxidant defense. Many studies support enhancing chemotherapy by lowering antioxidant defense. However, chemotherapy and radiation carry a significant risk of triggering a secondary cancer. This is a very important consideration today as cancer is being diagnosed in a much younger population.

Antioxidants - Friend or Foe?

Antioxidants may be important for the prevention of cancer but have no value in cancer treatment. Supplementation with certain antioxidants has been shown to raise cancer risk. Trials involving administration of antioxidants have failed to halt tumor growth or cause any death of cancer cells. Evidence shows that antioxidants actually fuels cancer's spread. This may sound unbelievable or surprising given all the marketing hype for antioxidant supplements and high antioxidant foods.

The definition of antioxidant is a substance that protects cells from the damage caused by free radicals (unstable molecules made by the process of oxidation during normal metabolism). Free radicals are atoms or groups of atoms that have a single unpaired electron. Oxygen molecules form into free radicals and oxidizing agents. Hydrogen peroxide is a well know oxidizing agent. Ozone is a gas which is a powerful oxidizing agent.

Antioxidant Defense Systems

Normal cells and cancer cells depend on antioxidant defense systems for survival and specifically to protect the cell from DNA damage. Cancer cells depend on antioxidant defense much more so than normal cells. Are there methods to weaken or disable antioxidant defense systems so that cancer cells are oxidized to death? Science has revealed that there are simple ways to disable antioxidant defense systems.

There are two major cellular antioxidant defense systems. One is Glutathione (GSH) and has been extensively studied in relation to cancer. Glutathione scavenges free radicals (ROS) maintaining redox balance.

The second major antioxidant defense system is Thioredoxin (TRX). Thioredoxin has also been extensively studied in relation to cancer. Cancer cells are highly dependent on Thioredoxin for survival.

Both antioxidant defense systems have been identified to be involved in chemoresistance meaning the cancer cells adaptive defense against chemotherapy. The same applies to radiotherapy.

Chemotherapeutic drugs kill cancer cells primarily through oxidation overload. DNA damage is secondary. Cancer cells become resistant to chemotherapy by an upregulation of antioxidant defense. Many studies support enhancing chemotherapy by lowering antioxidant defense. Why not fully disable antioxidant defense and allow the endogenous oxidation to reach a cytotoxic threshold killing the cancer cell? This would mean that chemotherapy and radiotherapy are unnecessary. Surgery may become unnecessary in most cases as well.

Disabling Antioxidant Defense

Redox is a term referring to a state of balance between oxidation and reduction. Redox chemistry is an essential part of all living organisms. Redox balance is necessary for normal metabolic functions which does not damage DNA or cellular components.

Drugs are being developed to disable antioxidant defense but there is another way that is much safer and is very inexpensive. One example of a drug being studied is a repurposed rheumatoid arthritis drug called Auranofin which disables the Thioredoxin antioxidant defense system. Another example is Buthionine Sulfoximine which depletes Glutathione.

Here is where nutrition powerfully enters into the solution. A dietary approach is available for disabling the Glutathione antioxidant defense system. This involves

limiting the intake of two amino acids, methionine and cysteine. Glutathione is synthesized from three amino acids, cysteine, glycine and glutamate. Cysteine is a non-essential amino acid. Methionine is essential but at a very low level.

A plant-based diet is significantly lower in methionine and cysteine than a typical animal protein centered diet. Refining a plant-based diet to be sufficiently low in methionine and cysteine is relatively straightforward and practical. It is essentially a diet of strictly fruits and vegetables for short intervals. Nuts, seeds, grains and beans contain significant levels of methionine and cysteine.

There are several different ways to disable the Thioredoxin antioxidant defense system with natural agents. Possibly the most reliable and efficient method is orally administered sodium selenite which is an inorganic selenium salt. Sodium selenite has been studied for over 50 years for its selective anticancer activity.

Building a Redox Based Cancer Treatment Protocol

Oxidizing cancer cells to death can be achieved by sufficiently tipping the redox balance within cancer cells towards excess oxidative stress (ROS). Dual disabling of the Glutathione and Thioredoxin antioxidant systems has been experimentally shown to selectively trigger cancer cell death.

The protocol must eliminate all antioxidant supplements including vitamin C, NAC, Vitamin E Tocopherol and Vitamin A. Antioxidants contained in food are not a limiting factor,. However, it is advised to avoid drinking vegetable juices which may contribute excess exogenous antioxidants.

Dietary restriction of methionine and cysteine is known to impact glutathione synthesis. The limitation is that all foods contain methionine and cysteine. A diet of very low methionine and cysteine has enormous therapeutic value but another element is required to fully deplete cysteine. Vitamin B6 (P5P) comes to the rescue as a way to non-enzymatically deplete cysteine. Iron is incorporated as a catalyst for the reaction between Vitamin B6 (P5P) to proceed at a high rate. Cysteine is broken down into hydrogen sulfide, pyruvate and ammonia. These breakdown products are in very small amounts and of little consequence to normal cells.

Disabling the Thioredoxin antioxidant system can be achieved with the administration of various natural compounds and off-label drugs. The natural compound of choice is sodium selenite because of its ideal pharmacology. Sodium selenite can be orally administered with special formulations that avoid GI upset.

A complete oxidative protocol is therefore based on a methionine/cysteine restricted diet, Vitamin B6 (P5P), Iron and Sodium Selenite. Other elements can be incorporated that induce additional oxidative stress or added support ferroptosis induction. One example for ferroptosis induction is high PUFA oil.

Both Sodium Selenite and Vitamin B6 (P5P) individually exhibit potent anticancer activity. Utilizing these agents within a highly targeted therapeutic system greatly enhances their effects as individual agents.

Additional oxidative stress can be applied through the oral administration of high ozonide ozonated oils. This is a way to implement the equivalent of ozone therapy at home and on a continuous basis.

Conclusion

Presented here is a revolutionary and cutting edge method for treating cancer that leverages the power of diet and inexpensive natural compounds. Small scale human clinical case studies have confirmed that this treatment approach is safe, highly effective and applicable to all forms and stages of cancer. This treatment is highly cost effective because IV injections are unnecessary and the cost of the orally administered nutraceuticals can be as little as \$300 USD per month. No expensive foods or kitchen equipment are necessary. The therapy is 100% home-based except for routine blood tests. The next challenge is disseminating this approach first among naturopathic physicians and integrative oncologists followed by mainstream oncologists.

About the Author

Mark Simon is the founder and director of Nutritional Oncology Research Institute which was establish in 2011. His research for the past 20 years has been focused on developing natural and nontoxic methods for cancer treatment. Mark Simon is involved in developing and manufacturing nutraceutical products for cancer therapy. He can be contacted by email at: msimon20@earthlink.net

Disclaimer: The information presented here is for educational purposes only and is not intended as medical advise. Please consult your healthcare provider prior to embarking on any treatments that are outside the standard of medical care.

References

Cockfield JA, Schafer ZT. Antioxidant Defenses: A Context-Specific Vulnerability of Cancer Cells. Cancers (Basel). 2019 Aug 20;11(8):1208. doi: 10.3390/cancers11081208. PMID: 31434226; PMCID: PMC6721511. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6721511/

Jelic, Marija Dragan1,; Mandic, Aljosa D.1,2; Maricic, Slobodan M.1,2; Srdjenovic, Branislava U.1. Oxidative stress and its role in cancer. Journal of Cancer Research and Therapeutics 17(1):p 22-28, Jan–Mar 2021. | DOI: 10.4103/jcrt.JCRT_862_16 https://journals.lww.com/cancerjournal/fulltext/2021/17010/ oxidative stress and its role in cancer.4.aspx

Nakamura H, Takada K. Reactive oxygen species in cancer: Current findings and future directions. Cancer Sci. 2021 Oct;112(10):3945-3952. doi: 10.1111/cas.15068. Epub 2021 Aug 2. PMID: 34286881; PMCID: PMC8486193. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8486193/

Perillo, B., Di Donato, M., Pezone, A. et al. ROS in cancer therapy: the bright side of the moon. Exp Mol Med 52, 192–203 (2020). https://doi.org/10.1038/s12276-020-0384-2 https://www.nature.com/articles/s12276-020-0384-2

Singh R, Manna PP. Reactive oxygen species in cancer progression and its role in therapeutics. Explor Med. 2022;3:43–57. https://doi.org/10.37349/emed.2022.00073 https://www.explorationpub.com/Journals/em/Article/100173

Yang, H., Villani, R.M., Wang, H. et al. The role of cellular reactive oxygen species in cancer chemotherapy. J Exp Clin Cancer Res 37, 266 (2018). https://doi.org/10.1186/s13046-018-0909-x

https://jeccr.biomedcentral.com/articles/10.1186/s13046-018-0909-x

Manish A. Shah, Harry A. Rogoff, Implications of reactive oxygen species on cancer formation and its treatment, Seminars in Oncology, Volume 48, Issue 3, 2021, Pages 238-245, ISSN 0093-7754, https://doi.org/10.1053/j.seminoncol.2021.05.002. https://www.sciencedirect.com/science/article/pii/S0093775421000518?via%3Dihub

Kennedy L, Sandhu JK, Harper ME, Cuperlovic-Culf M. Role of Glutathione in Cancer: From Mechanisms to Therapies. Biomolecules. 2020 Oct 9;10(10):1429. doi: 10.3390/biom10101429. PMID: 33050144; PMCID: PMC7600400. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7600400/

Boyi Niu, Kaixin Liao, Yixian Zhou, Ting Wen, Guilan Quan, Xin Pan, Chuanbin Wu, Application of glutathione depletion in cancer therapy: Enhanced ROS-based therapy, ferroptosis, and chemotherapy, Biomaterials, Volume 277, 2021, 121110, ISSN 0142-9612, https://doi.org/10.1016/j.biomaterials.2021.121110. https://www.sciencedirect.com/science/article/abs/pii/S014296122100466X

Malin D, Lee Y, Chepikova O, Strekalova E, Carlson A, Cryns VL. Methionine restriction exposes a targetable redox vulnerability of triple-negative breast cancer cells by inducing thioredoxin reductase. Breast Cancer Res Treat. 2021 Dec;190(3):373-387. doi: 10.1007/s10549-021-06398-y. Epub 2021 Sep 22. PMID: 34553295; PMCID: PMC8793942. https://pubmed.ncbi.nlm.nih.gov/34553295/

Wallis KF, Morehead LC, Bird JT, Byrum SD, Miousse IR. Differences in cell death in methionine versus cysteine depletion. Environ Mol Mutagen. 2021 Mar;62(3):216-226. doi: 10.1002/em.22428. Epub 2021 Mar 2. PMID: 33615565; PMCID: PMC8130902. https://pubmed.ncbi.nlm.nih.gov/33615565/

The Role of the Thioredoxin Detoxification System in Cancer Progression and Resistance, Frontiers in Molecular Biosciences Vol.9, 2022 https://www.frontiersin.org/articles/10.3389/fmolb.2022.883297 https://www.frontiersin.org/articles/10.3389/fmolb.2022.883297/full

Jayachandran P, Knox SJ, Garcia-Cremades M, Savić RM. Clinical Pharmacokinetics of Oral Sodium Selenite and Dosing Implications in the Treatment of Patients with Metastatic Cancer. Drugs R D. 2021 Jun;21(2):169-178. doi: 10.1007/s40268-021-00340-9. Epub 2021 Apr 17. PMID: 33866531; PMCID: PMC8206290.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8206290/

Kieliszek M, Lipinski B, Błażejak S. Application of Sodium Selenite in the Prevention and Treatment of Cancers. Cells. 2017 Oct 24;6(4):39. doi: 10.3390/cells6040039. PMID: 29064404; PMCID: PMC5755498. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5755498/

In Vitro Anticancer Activities of B6 Vitamers: A Mini-review TAISUKE MATSUO, YASUYUKI SADZUKA Anticancer Research Jul 2019, 39 (7) 3429-3432; DOI: 10.21873/anticanres.13488 https://ar.iiarjournals.org/content/39/7/3429#