A Potent Oxidative Nutraceutical Cocktail for Cancer Treatment

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February 2021

Researching and developing natural compounds for the treatment of cancer is the primary focus of NORI. Natural compounds carry many advantages including very low toxicity, low-cost, few, if any side effects and simple in-home treatment. An enormous body of scientific evidence has uncovered exciting opportunities in developing natural compounds as powerful cancer therapeutic agents. Combining several natural compounds together to form a synergistic cocktail is an optimal approach because of limitations of one single agent. By choosing the most optimal combination of compounds, efficacy, safety, universality and minimization of off target effects will be greatly maximized. This paper discusses a combination of three natural and selective chemotherapeutic agents that have proven safety and efficacy in human clinical studies. Thousands of natural compounds have been discovered that selectively trigger apoptosis in cancer in vitro. Bioavailability is the key limiting factor preventing the vast majority of natural compounds from becoming viable therapeutic agents. NORI focuses on compounds that exhibit sufficiently high bioavailability without resorting to nano particle technology or other means to increase absorption.

It is well established that cancer cells struggle with redox regulation or in other words, managing oxidative stress. This is due, in part, to altered/reprogrammed metabolism and defects within the mitochondrial respiratory chain. More specifically, dysfunction in the electron transport chain (ETC) may be why mitochondria are unable to support the bulk of ATP synthesis compared to a normal cell. Complexes I-IV exhibit defects in malignant cells and these defects cause electron leakage causing an elevation in ROS. Mutations within mitochondrial DNA are seen in cancer cells and are directly implicated in malfunctions in the ETC. Cleary, exploitation of redox imbalance that is on the edge of catastrophic cellular breakdown is an excellent target for cancer treatment. Most chemotherapy agents trigger cancer cell death through elevation of ROS. The approach of creating redox imbalance is nonspecific and independent of cell signaling pathways. It is a broad and universal approach that is unlikely to cause drug resistance.

Upsetting redox balance towards increased oxidative stress in cancer cells is a reliable means to trigger apoptosis. Blocking glutathione synthesis with BSO triggers apoptosis by causing an abrupt and severe elevation in ROS within the cancer cell. Various agents are known to elevate ROS through a variety of different mechanisms. Some agents are redox recycling inducing (quinones), some are lipid peroxidation catalysts and others simply react with intracellular molecules generating H2O2 and superoxide. Both oxygen and nitrogen reactive species may be involved in tipping the redox balance leading to apoptosis. Mitochondria are intimately involved in apoptosis through initiating the cascade of apoptotic signals beginning with the release of cytochrome c. Other mitochondrial events include depolarization of the mitochondrial membrane. Collapse of mitochondrial function and structures is the key event leading to the demise of cancer cells. One way to view this is inducing further damage to already damaged mitochondria.

Nutraceutical Cocktail

This paper discusses the application and potential synergistic behavior of sodium selenite, molecular iodine and vitamin E delta-tocotrienol as a pro-oxidant cocktail. Each component individually possess potent and selective cytotoxicity across a wide array of cancer cell lines. Each agent demonstrates dose and time dependent selective cytotoxicity. Combining these agents together as a cocktail may allow for lower dosages, oral administration only, virtually zero adverse side effects and very high therapeutic potential. There is no single agent that will be consistently reliable and effective in triggering apoptosis or other forms of cancer cell death. A cocktail broadens out the mode of action and interacts with multiple targets. The term "cocktail" does imply that the agents are mixed together or must be administered simultaneously. Each agent may be administered through different routes, time schedule and frequency. The plasma half-life of each agent is sufficient for overlap over a sufficiently long time interval.

Sodium Selenite

Sodium selenite is an inorganic salt of selenium. Sodium selenite is the key pro-oxidant agent in the cocktail. it is a small water soluble molecule and easily crosses the blood-brain barrier. Sodium selenite has a wide therapeutic index. Dosage range for sodium selenite is 8-24 mg/day. Sodium selenite is available as a sublingual tablet, slow release oral tablet and as an IV solution. NORI has incorporated sodium selenite as the primary cytotoxic agent for 10 years. It has demonstrated the ability to stabilize and fully regress a wide array of cancers. Sodium selenite has been very effective in treating metastatic papillary thyroid cancer. It has shown efficacy for prostate, breast lung, colon, brain and blood cancers. Adverse reactions are extremely rare. Sodium selenite will induce nausea if the stomach lining is exposed to an excess. This is why a sublingual or very slow release tablet must be incorporated for oral administration.

Molecular Iodine

Molecular iodine (I2) behaves very differently than iodide. Molecular iodine is inherently very unstable but stable formulations have been manufactured that can be administered orally. Molecular iodine is a very powerful antimicrobial agent and far more powerful than ionic iodine. Dosage is typically 5 mg/day. Human clinical studies have verified the safety and efficacy of molecular iodine as an adjunctive therapy for breast cancer. It is interesting to note that iodine is right next to selenium on the periodic chart of the elements. Studies show that molecular iodine triggers apoptosis through identical mechanisms as sodium selenite.

Vitamin E Delta-Tocotrienol

Vitamin E delta-tocotrienol is a form of vitamin E that behaves primarily as a pro-oxidant. The tail portion of the VEDT molecule has three unsaturated bonds that are subject to the initiation of lipid peroxidation chain reactions. The bioavalibility of VEDT is sufficient to achieve pharmacologically effective plasma concentration. VEDT appears to localize within the mitochondrial membrane. Dosage is between 900-1200 mg/day. VEDT must be pure and free of any vitamin E tocopherol which will interfere with the oxidative effects of VEDT and the other agents of the cocktail. VEDT has demonstrated promising results in the treatment of pancreatic cancer in human clinical studies.

Supportive Nutraceuticals and Therapies

There are nutraceuticals that may potentially support and enhance the effects of the cocktail. These include high dose zinc, high dose melatonin and high dose glucosamine. Ozone therapy is likely a supportive adjunctive therapeutic modality. Any treatment that increases oxidative stress or depletes antioxidant defenses will cooperate and enhance the effects of the cocktail.

NORI recommends a low-fat plant-based diet as a foundation for any form of cancer therapy. One can take a step further and implement a low-methionine diet which may enhance the effects of the cocktail. Methionine deprivation is believed to cause an elevation of ROS in cancer cells by limiting glutathione synthesis. A well designed plant-based diet may potentially help suppress angiogenesis, lower inflammation, lower IGF-1, lower insulin and create favorable changes to the tumor microenvironment.

Availability of Cocktail

NORI Nutraceuticals manufactures and supplies all three components of the cocktail to both clinicians and patients. Close supervision is highly recommended. It is the intention of NORI to provide this research and nutraceuticals to naturopathic oncologists, Integrative oncologists and other health professional licensed to practice alternative methods of healing. Since the cocktail is composed of non-patentable components, there is no incentive for further research and development by the pharmaceutical industry or other mainstream entities.

Disclaimer: The statements made regarding these products have not been evaluated by the United States Food and Drug Administration. The efficacy of these products has not been confirmed by FDA-approved research. These products are not intended to diagnose, treat, cure or prevent any disease.

References Available Upon Request