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Supportive Use of High Dose Antioxidants with Probiotics Would Enhance Effectiveness of Chemo/Radiation Therapy While Protecting Normal Cells

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Abstract

Despite treatments with Chemo/radiation therapy, the number of cancer death continues to increase. The number of deaths increased from 569,000 in 2010 to 611,720 in 2024. These treatments produce acute adverse effects during treatment and late adverse side-effects after treatment. Therefore, a new approach to improve survival rate and reduce toxicity is needed. In 2014, FLASH radiation therapy delivers radiation dose to tumor at a dose rate of 40 Gy/sec compared to dose rate of 0.01 Gy/sec of conventional radiation therapy protected normal tissue against acute injury, while the tumor response did not change. Therefore, another new approach is needed. Individual antioxidants such as vitamin C and vitamin E succinate at high doses inhibit the growth of cancer cells and enhance the effects of chemotherapeutic agents and x-irradiation on cancer cells, while protecting or no significant effects on normal cells in culture and in animal model.

While searching for mechanisms of high dose antioxidants, it was found that cancer cells require glucose and glutamine for their survival and growth. This led us to propose that high doses of antioxidants kill cancer cells by inhibiting the uptake and metabolism of these nutrients. Indeed, literature search revealed that some individual antioxidants prevent uptake of glucose, while others prevent the uptake of glutamine in cancer cells. Therefore, we developed of a formulation of high doses of multiple antioxidants which may kill tumor cells by inhibiting the uptake of glucose and glutamine and enhance the effectiveness of chemo/radiation therapy while reducing their toxicity Since intestinal dysbiosis participates in the development and progression of cancer and reduces the effectiveness of chemo/radiation therapy, combining probiotics and prebiotics with high doses of multiple antioxidants before, during, and after treatment may markedly enhance survival rate of patients while protecting normal cells.

Keywords: Antioxidants; Probiotics; Cancer Treatment; Normal Cells; Toxicity

Abbreviations: CONV-RT: Conventional Radiation Therapy; NB: Neuroblastoma; COX-: Cyclooxygenase; IEC-6: Epithelial Cell Line; GLUT-1: glucose transporter-1; GLUT-3: Glucose Transporter-3; CRC: colorectal cancer; VEGF: vascular endothelial growth factor; MMP-2: Marker Matrix Metalloproteinase 2; ER: Estrogen Receptor; EGCG: Epigallocatechin Gallate

Introduction

As of 2024, the 5-year relative survival rate for all cancers combined has improved to 69% (diagnosed 2013-2019), a significant rise from 49% in the mid-1970s. This form of therapy is highly effective for localized and regional cancers, such as for early-stage anal cancer (86.3% 5-year survival), but rates vary widely based on cancer type and stage (American Cancer Society.2024). The extent of increase depends upon the type of tumor, the stage of tumor, and the location of tumor. Despite these treatments the number of cancer death continues to increase. In 2010, the number of deaths was 569,000, in 2020, it was 602,000 and in 2024, it was approximately 611,720. In addition, chemo/radiation therapy produces adverse side-effects which include loss of hairs, depressed immune response, loss of appetites, fatigues, and nausea. The late adverse effects of these cancer

treatments include recurrence of primary tumor and appearance of second primary cancers. In addition, damage to the brain, bone, endocrine system, eyes, hearing, heart, lung, joints, and mouth, if occur these organs are involved in the treatment area [1,2]. To improve radiation treatment, in 2014, a new technology referred to as FLASH radiation therapy which delivers radiation dose to tumor at a dose rate of 40 Gy/sec compared to dose rate of 0.01 Gy/sec of conventional radiation therapy (CONV-RT) was developed. This therapy protected normal tissue against acute radiation injury, but the tumor repones rate was like CONV-RT [3]. Therefore, another novel approach which can enhance the survival rate irrespective of the type, stage and treatment sensitivity and reduce acute and late adverse effects of Their toxicity is needed.

Primary mechanisms of damage by Chemo/Radiation therapy include free radicals and free radical induced inflammation. This form of therapy damages both cancer cells and normal cells. Commonly used antioxidants are known to scavenge free radicals. Therefore, use of low dose antioxidants during chemo/radiation therapy would reduce the effectiveness of therapy by protecting both cancer cells and normal cells.

Use of High Doses of Individual Antioxidants on Cancer and Normal Cells

Vitamin C on Human Cancer: In1976, Dr. Linus Pauling and his colleague were first to demonstrate that high doses of vitamin C alone inhibited the growth of cancer cells in humans [4,5]. This observation created a lot of controversy among oncologists. The above observation could not be confirmed in another clinical study on human [6].

Vitamin C on Cancer Cells in Culture: We decided to test Dr. Pauling observation on murine neuroblastoma (NB) cells in culture and found that high doses of vitamin C as sodium ascorbate killed NB cells in culture, however, the growth of murine fibroblasts was minimally affected. In addition, we showed that vitamin C at high doses enhances the growth-inhibitory effects of x-irradiation and chemotherapeutic agents on neuroblastoma cells in culture without significantly affecting normal fibroblasts. Vitamin C at high doses also inhibited the growth of human tumorigenic parotid acinar cells in culture but had no effect on the growth of non-tumorigenic parotid acinar cells [7].

Vitamin E Succinate on Cancer Cells: In 1982, we discovered that d-alpha-tocopheryl succinate (vitamin E succinate) at high doses induced differentiation and growth inhibition in murine melanoma cells in culture [8]. Vitamin E succinate at a high dose reduced the expression of oncogenes c-myc and H-ras in melanoma cells in culture [9]. Several studies using other cancer cell lines in culture and in animal models of cancer confirmed this observation [10-13]. A recent review has further documented the role of vitamin E succinate in inhibiting the growth of tumor cells [14].

Beta-carotene or retinol on cancer cells in culture: Treatment with high doses of beta-carotene or retinol induced differentiation in murine neuroblastoma cells and B-16 murine melanoma cells in culture, respectively, and inhibited their growth [15].

Quercetin on Cancer cells in Culture: Overexpression of cyclooxygenase (COX-2) plays a significant role in the development and progression of cancer. Treatment with high doses of quercetin increased apoptosis and inhibited the growth of human colon cancer cells (HT29 cells) over-expressing Cox-2 enzyme without significant effect on the growth of normal epithelial line (IEC-6) [16].

Resveratrol on Cancer Cells In Vivo and In Vitro: Treatment with high doses of resveratrol reduced the growth of human leiomyoma cells in culture and in rat-derived uterine leiomyoma transplanted in athymic mice [17].

Coenzyme Q10 on human Cancer: Daily supplementation of coenzyme Q10 at a high dose of 390 mg or more daily may increase the survival time of breast cancer patients receiving standard cancer therapy [18-20]. On the other hand, another clinical study on breast cancer revealed that administration of 300 mg coenzyme Q10 did not improve fatigue or the quality of life during treatment [21].

Curcumin on Cancer Cells in Culture: Treatment with high doses of curcumin reduced the growth and caused apoptosis in various cancer cells in culture including gastric carcinoma and colon cancer cells [22-25].

Antioxidant Mixture on Cancer Cells in Vivo and In Vitro: Treatment with a mixture of antioxidants containing high doses of ascorbic acid, quercetin, green tea extract, lysine, and proline inhibited the growth of human ovarian cells in culture and in athymic mice [26]. The elimination of quercetin from this mixture did not affect the potency of this mixture in inhibiting the growth of human retinoblastoma cells in culture and in athymic mice [27], human leukemic cell line (U-93,) and head and neck squamous cell carcinoma cell line (FaDu) in culture and in athymic mice [28]. Treatment with another antioxidant mixture containing high doses of quercetin, curcumin, resveratrol, green tea extract, and cruciferex also reduced the growth of human head and neck squamous cell carcinoma in culture (OHSU-974 cell line) and in athymic mice as well as in fibrosarcoma (HT-180 cell line) and melanoma (A2058 cell line) [29]. A mixture of antioxidants containing vitamin C, vitamin E, and beta-carotene enhanced the cytotoxic effects of combined treatment with paclitaxel and carboplatin on human lung squamous cell carcinoma cell line H520 [30]. Another mixture of antioxidants containing vitamin C, retinoic acid, polar carotenoids, and d-alpha-tocopheryl succinate even at non-growth-inhibitory dose reduced the growth of human melanoma cells in culture by about 50%. This suggests that antioxidants in the mixture interact with each other to reduce

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the growth of cancer cells. In addition, increasing the dose of vitamin C in the same mixture dramatically reduced the growth of melanoma cells in culture [31]. This suggests that high doses of all four antioxidants in combination might have produced more dramatic inhibition of growth of these cells. The same antioxidant mixture reduced the growth of tumorigenic parotid acinar cells (2HP1G) without significantly affecting that of growth of non-tumorigenic parotid acinar cells (2HPC8) in culture [7]. These studies suggest that high doses of antioxidants kill cancer cells but not normal cells [32-34].

The above studies of high dose antioxidants on cancer cells although appeared impressive drew no interest from oncologists and cancer researchers. The main reason could have been that no mechanism of action of antioxidant other than scavenging free radical was known. The mechanisms of action of high dose of antioxidants were totally unknown.

This review proposes a novel mechanism of action of high dose antioxidants which selectively kill cancer cells irrespective of their type, stage, location, and treatment sensitivity without affecting normal cells. Such mechanism of action would also enhance the effectiveness of chemo/radiation therapy. Use of such high dose antioxidants may reduce the acute and late adverse effects of these therapies.

Searching for Mechanisms of Action of High Dose Antioxidants

A few years ago, while I was searching for mechanisms of action of high dose antioxidants, the question arose in my mind, do cancer cells require specific nutrients for their survival and growth? Literature research revealed that cancer cells require glucose and glutamine for their survival and growth. Glucose is needed to produce energy, while glutamine is essential for making vital molecules such as protein and nucleic acid.

Requirements of glucose: All cancer cells require excessive amounts of glucose, because they utilize glycolysis as an inefficient energy-producing system, which converts one molecule of glucose to 2 molecules of ATP (adenosine triphosphate), whereas normal cells use oxidative phosphorylation pathways which generate 36 ATP from one molecule of glucose. Cancer cells exhibit increased expression of glucose transporter-1 (GLUT-1) and glucose transporter-3 (GLUT-3) that enhance the uptake of glucose causing accumulation of glucose in the cells. Excess glucose in cancer cells is oxidized to produce extensive amounts free radicals which are required for the proliferation and metastasis of cancer cells [35]. In addition, increased accumulation of glucose leads to more production of lactic acid making cellular environment acidic which favors growth of cancer cells [36]. Inhibitors of glucose transporters GLUT-1 and GLUT-3 increase the death of cancer cell by inhibiting the uptake of glucose [37].

Requirement of Glutamine: All cancer cells require glutamine uptake and metabolism for their survival and growth [38], because it provides source of energy, and nitrogen for protein and nucleic acid synthesis that are essential for their survival and growth [39]. Therefore, preventing the uptake of glutamine may cause death of cancer cells. It has been demonstrated that reducing the availability of glutamine promotes radiosensitivity of prostate cancer [40]. It has been shown that inhibition of glutamine metabolism increases the sensitivity of Kras positive pancreatic ductal adenocarcinoma to radiation therapy [41]. These studies indicate that the inhibition of uptake or metabolism of glutamine can cause death of tumor cells. Based on the above studies, the question arose whether high dose antioxidants inhibit glucose and glutamine in cancer cells. Literature search revealed that they do. Some data are presented here.

High Dose Individual Antioxidant Kills Cancer Cells by inhabiting Uptake and Metabolism of Glucose

Vitamin C: Treatment with high dose of vitamin C inhibited glycolysis creating energy crisis causing death of several types of cancer cells. These include human colorectal cancer (CRC cell line) carrying Kras mutation or Braf mutation that makes them resistance to standard therapies [42], non-small cell lung cancer cells [43], and colon cancer [44].

Alpha-Lipoic Acid: Treatment with alpha-lipoic acid at high doses reduced glucose uptake and reduced the growth of neuroblastoma cells and breast cancer cells (SKBr3 cell line) in culture and on transplanted tumor in a thymic mouse [45].

Quercetin and Epigallocatechin Gallate (EGCG): Treatment with quercetin blocked the uptake of glucose leading to inhibition of glycolysis causing growth inhibition of tumor cells in culture. This also reduced growth and metastasis in animal models by decreasing the levels of its marker matrix metalloproteinase 2 (MMP-2), MMP-9, and vascular endothelial growth factor (VEGF). Quercetin also inhibited tumor growth and metastasis by inhibiting glycolysis in vivo [46]. Both quercetin and EGCG inhibited the uptake of glucose and reduced the growth of estrogen receptor (ER)-positive cell line (MCF7) and ER-negative cell line (MDA-MB-231) breast cancer cells in culture [47].

Resveratrol: Treatment with resveratrol-loaded polymeric nanoparticles reduced glucose metabolism and inhibited the growth of colon cancer cells in culture (CT26 cell line) and in CT26 transplanted mice [48]. Resveratrol induced apoptosis in ovarian cancer cells in culture by inhibiting the uptake of glucose [49] Treatment with resveratrol reduced glucose uptake and glycolysis in breast cancer cells in culture and reduced the growth of tumor in mice carrying Lewis Lung carcinoma, HT-colon cancer, and breast cancer cells (T47D) [50].

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Curcumin: Treatment with curcumin at high doses inhibited glucose uptake in variety of cancer cells and reduced their growth [51]. It also prevented glucose-induced chemoresistance by blocking the uptake of glucose in hepatic carcinoma cells [52].

High Dose Individual Antioxidants Kills Cancer Cells by Inhibiting Glutamine Uptake and Metabolism

Resveratrol: Treatment with resveratrol enhanced cisplatin-induced apoptosis in hepatoma cells in culture by inhibiting glutamine metabolism [53].

Curcumin: Curcumin treatment in combination with cisplatin suppressed proliferation of colon cancer cells in a synergistic manner. In addition, curcumin treatment overcame cisplatin resistance in colon cancer cells by inhibiting the uptake of glutamine [54].

Vitamin D3: Treatment with vitamin D3 at a high dose inhibited glutamine uptake and reduced the growth of H-ras transformed human breast epithelial cells. This effect of vitamin D3 was mediated by reducing the level of a major glutamine transporter protein (SLC1A5). Vitamin D3 treatment inhibited glutamine uptake and reduced the growth of human breast cancer in culture [55].

Causes of Cancer Cells to Develop Resistant to Therapies

A nuclear transcriptional factor, Nrf2 is constitutively expressed in cancer cells [56]. Activation of Nrf2 increases the level of antioxidant enzymes, which protect cancer cells from oxidative damage, allowing rapid progression and metastasis and making them resistant to therapeutic agents. In addition, glutathione peroxidase-2, which is highly expressed in cancer cells, enhances level of glutathione that protects cancer cells from oxidative damage and make them resistant to therapeutic agents [57].

Proposed High Doses of Multiple Antioxidants as Supportive Care in Combination with Chemo/Radiation Therapy

Since some individual antioxidants inhibit glucose uptake and metabolism while others inhibit glutamine uptake and metabolism, we developed a formulation high dose of multiple antioxidants for use as supportive care in combination with chemo/radiation therapy, this formulation has been patented (US patent number 11,938,152, B2). We propose that a formulation of high doses of multiple antioxidants would kill cancer cells irrespective of their type, stage, location, and sensitivity of treatment.

Role of Intestinal Dysbiosis in the Development and Treatment of Cancer

The role of intestinal dysbiosis (increase in the number of harmful bacteria and decline in the number of beneficial bacteria) in the treatment of cancer has not drawn significant attention from oncologists. Intestinal dysbiosis contributes to the initiation and progression of colorecta cancer [58], lung cancer [59], breast cancer [60], prostate cancer [61], and brain tumors [62] and reduces the effectiveness of chemotherapy and radiation therapy [63-65]. Therefore, we propose that supplementation with probiotics with prebiotics together with high doses multiple antioxidants before, during, and after Chemo/Radiation therapy may reverse the harmful effects of intestinal dysbiosis.

Proposed Interim Cancer Treatment Protocol Using High Doses of Multiple Antioxidants Together with Probiotics with Prebiotics

In the absence of patented high dose antioxidant preparation, I developed an interim cancer treatment protocol which can be used in consultation with oncologists. This protocol involves using commercially available multivitamins 3 times of normal dose after breakfast, after lunch, and after dinner. Each patient will receive about 17 gm of multiple antioxidants before, during, and after chemo/radiation therapy. This total dose of multiple antioxidants is like the patented "high dose antioxidants in cancer treatment" US Patent No 11,938,152, B2 which has 16.8 gm of multiple antioxidants. This protocol also includes taking commercially sold probiotics with prebiotics (one capsule in the morning and one in the evening). This interim cancer treatment protocol was not approved by any state, Federal, or any company. Adopting the interim protocol depended upon patients and their oncologists. At least over 50 patients with different type, stages, and locations of cancer have used this interim cancer protocol. Most have used it in combination of chemo/radiation therapy, while a few of them used only interim protocol. Preliminary data have been published [66].

Conclusion

At present, Chemo/radiation therapy has increased 5-year of survival; however, the extent of increase in survival rate depends upon the type, stage, and location of cancer. In addition, acute damage to normal tissue during treatment and late adverse effects of these therapies remain a major concern of oncologists. Therefore, a new approach to improve clinical outcome is needed. In 2014, a new technology referred to as FLASH radiation therapy which delivers radiation dose to tumor at a dose rate of 40 Gy/sec compared to dose rate of 0.01 Gy/sec of conventional radiation therapy was developed. This form of therapy protected normal tissue against acute injury, but the tumor repose rate was like conventional radiation therapy. Therefore, another approach is needed. Several experimental studies to support the value of high doses of multiple antioxidants in cancer treatment are available, but the mechanisms of action are unknown. While searching for mechanisms of action of high dose antioxidants, it was found that all cancer cells need glucose and glutamine for survival and growth and high doses of antioxidants block

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them in cancer cells causing their death. Based on this idea, we developed an interim cancer protocol of high doses of multiple antioxidants in combination with probiotics which can be used in consultation with oncologists. This protocol involves taking a commercially available multivitamins 3 times of normal dose after breakfast, after lunch, and after dinner together with one capsule of commercial sold Probiotics with prebiotics in the morning and one in the evening. Each patient will receive about 17 gm of multiple antioxidants before, during, and after chemo/radiation therapy. The total dose of multiple antioxidants is like patented "high dose antioxidants in cancer treatment" US Patent No 11,938,152, B2 which has 16.8 gm of multiple antioxidants.

Ethical Statement

Since it is a review manuscript, ethical statement is not needed. Any ethical statement related to a review paper has been met.

Conflict

The author is Chief Scientific Officer of Engage Global of Utah. This company sells nutritional products to consumers.

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