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Introducing the Sorush Cancer Treatment Protocol (SCTP)

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Abstract

This research introduces Sorush Cancer Treatment Ptotocol (SCTP) based on successful cancer treatment methodology which has done by Dr. Somayeh Zaminpira and Dr. Sorush Niknamian in 54 cancer patients in Violet Cancer Institute (VCI), Combining Specific Ketogenic Diet (SKD)- which has 80% saturated fat, 15% Protein with the lowest glutamine and 5% complex-high fiber carbohydrate- and intravenous ozone therapy which has had marvelous results in the treatment of several cancers in human models. The aim of this protocol is to weaken and starve cancer cells, decreasing acidity, increasing the immune system response, decreasing the possibility of metastasis and decreasing the cachexia without any serious side effects in cancer patients. This protocol introduces mega vitamins and minerals plus several supplements based on some tried and true protocols mainly the Budwig and Bill Henderson Protocol (BHP) with serious revisions. The BHP incorporates principal components of the Budwig Diet which was developed in the early 1950s by German chemist Dr. Johanna Budwig (1908-2003). Budwig's theory is based upon the work of Otto Warburg (1883-1970). Warburg was an earlier Nobel Prize Laureate (1931) for the discovery of the nature and action of the respiratory enzyme, the first of the so-called yellow enzymes, or flavoproteins. Warburg's scientific efforts produced a large body of work and publications in highly respected journals such as Science (1928, 1956). According to Budwig, Warburg theorized that cellular respiration, like many chemical reactions, was dependent upon substrate availability, specifically a sulphydryl group and an unknown saturated fatty acid, which he failed to identify. According to Dr. S. Zaminpira and Dr. S. Niknamian, Cancer is an Evolutionary Metabolic disease (EMHC) and the main cause of cancer is the Butterfly Effect inside normal cells as a result of increasing the amounts of Reactive Oxygen Species (ROS).

Keywords: SCTP, SKD, BHP, Budwig Protocol, EMH, ROS, Ozone Therapy, HBO2T, Cancer Treatment, Cancer Prevention.

Introduction

Evolutionary Metabolic Hypothesis of Cancer (EMHC)

The first living cells on Earth are thought to have arisen more than 3.5 × 109 years ago, when the Earth was not more than about 109 years old. The environment lacked oxygen but was presumably rich in geochemically produced organic molecules, and some of the earliest metabolic pathways for producing ATP may have resembled present-day forms of fermentation. In the process of fermentation, ATP is made by a phosphorylation event that harnesses the energy released when a hydrogen-rich organic molecule, such as glucose, is partly oxidized.

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The electrons lost from the oxidized organic molecules are transferred via NADH or NADPH to a different organic molecule or to a different part of the same molecule, which thereby becomes more reduced. At the end of the fermentation process, one or more of the organic molecules produced are excreted into the medium as metabolic waste products. Others, such as pyruvate, are retained by the cell for biosynthesis. The excreted end-products are different in different organisms, but they tend to be organic acids. Among the most important of such products in bacterial cells are lactic acid which also accumulates in anaerobic mammalian glycolysis, and formic, acetic, propionic, butyric, and succinic acids [1].

The first cell on the earth before the entrance of the bacteria did contain nucleus and used the fermentation process to produce ATP for its energy. Then an aerobic proteo-bacterium enters the eukaryote either as a prey or a parasite and manages to avoid digestion. It then became an endosymbiont. As we observe, the fermentation process used the glucose or even glutamine to produce ATP, but the aerobic process used the glucose, fat and protein to produce more ATP than the previous one. The symbio-genesis of the mitochondria is based on the natural selection of Charles Darwin. Based on Otto Warburg Hypothesis, in nearly all cancer cells, the mitochondrion is shut down or are defected and the cancer cell do not use its mitochondrion to produce ATP [2]. This process of adaptation is based on Lamarckian Hypothesis of Evolution and the normal cells goes back to the most primitive time of evolution to protect itself from apoptosis and uses the fermentation process like the first living cells 1.5 billion years ago. Therefore, cancer is an evolutionary metabolic disease which uses glucose as the main food to produce ATP and Lactic Acid. The prime cause of cancer is the abundance of Reactive Oxygen Species produced by mitochondria that is a threat to the living normal cell and causes mitochondrial damage mainly in its cristae [3].

Hyperbaric Oxygen Therapy (HBOT)

Hyperbaric oxygen therapy (HBO2T) involves administration of 100% oxygen at elevated pressure (greater than sea level, or 1 ATA) in a closed chamber [190].

Ozone therapy and its mechanism of action

Ozone therapy has been utilized and extensively studied for many decades altogether. Its effects are proven, consistent and with minimal side effects. Medical O_3, used to disinfect and treat disease, has been around for over 150 years. Used to treat infections, wounds and multiple diseases, O_3's effectiveness has been well-documented. It has been used to disinfect drinking water before the turn of the last century. Ozone was known to treat as many as 114 diseases [4]. Ozone therapy has been in use since the 1800s and in 1896 the genius Nikola Tesla patented the first O_3 generator in the US, later forming the "Tesla Ozone Company" [5]. During the first world war (1914-18) doctors familiar with O_3's antibacterial properties, and with few other medical resources available to them applied it topically to infected wounds and discovered O_3 not only remedied infection, but also had hemodynamic and anti-inflammatory properties [6]. In the late 1980s, reports had emerged that German physicians were successfully treating HIV patients with 03-AHT (Auto-hemo-therapy). There was then no pharmaceutical treatment for HIV and a pandemic was feared, so Canadian authorities authorized the study to test safety and efficacy of 03-AHT in AIDS patients. Ozone had shown promise in *in vitro* testing. Ozone was seen effective at disinfecting extracorporeal blood samples of HIV; unfortunately for AIDS patients, 03-AHT proved to be an *in vivo* ineffective treatment [7, 8].

Ozone therapy disrupts the integrity of the bacterial cell envelope through oxidation of the phospholipids and lipoproteins. In fungi, O_3 inhibits cell growth at certain stages. With viruses, the O_3 damages the viral capsid and upsets the reproductive cycle by disrupting the virus-to-cell contact with peroxidation. The weak enzyme coatings on cells which make them vulnerable to invasion by viruses make them susceptible to oxidation and elimination from the body, which then replaces them with healthy cells [9].

Ozone therapy causes an increase in the red blood cell glycolysis rate. This leads to the stimulation of 2,3-diphosphoglycerate which leads to an increase in the amount of oxygen released to the tissues. Ozone activates the Krebs cycle by enhancing oxidative carboxylation of pyruvate, stimulating production of ATP. It also causes a significant reduction in NADH and helps to oxidize cytochrome C. There is a stimulation of production of enzymes which act as free radical scavengers and cell-wall protectors: glutathione peroxidase, catalase and superoxide dismutase. Production of prostacyline, a vasodilator, is also induced by O_3 [10].

Ozone administered at a concentration of between 30 and 55 μg/cc causes the greatest increase in the production of interferon and the greatest output of tumor necrosis factor and interleukin-2. The production of interleukin-2 launches an entire cascade of subsequent immunological reactions [11]. Ozone exposure induces a significant mean decrement in vital capacity. It significantly increases mean airway resistance and specific airway resistance but does not change dynamic or static pulmonary compliance or viscous or elastic work. It also significantly reduces maximal transpulmonary pressure. And furthermore, significantly increases respiratory rate and decreased tidal volume [12-20].

Materials and Methods

The Basis for the SCTP

The dietary suggestions, restrictions and supplements of the SATP address the processes Dr. Somayeh Zaminpira and Dr. Sorush Niknamian believe lead to the development of a variety of cancers. They include (1) lack of oxygen to the cells, (2) a weak immune system, (3) excessive acidity, and (4) toxicity in the body as a result of accumulation of tobacco, alcohol and asbestos. (5) High amounts of ROS in cells. (6) Lack of nutritional ketosis [S. Zaminpira, S. Niknamian, EC Cancer, ECRONICON, 2017]

The Supplemental Components of the SCTP:

Chlorella, Bee Pollen and Ascorbic Acid: Strengthening the Immune System and Countering Acidity

Chlorella vulgaris is a green microalgae mainly used as medical treatment in Japan. Alternatively, it has a big potential for biofuel production or as food additive. The proteins content of C. Vulgaris varies from 42 to 58% of its biomass dry weight [21-25]. These proteins are considered as having a good nutritional quality compared to the standard profile for human nutrition of the World Health Organisation and Food and Agricultural Organisation, as the algae synthesise essential and non-essential amino-acids [26]. The algae also contains lipids (5-40% of the dry mass) [27], carbohydrates (12-55% dry weight) [28,29] and pigments with among others chlorophyll, reaching 1-2 % of the dry weight. [30,31] C. vulgaris contains also some minerals and vitamins important for the human nutrition [32]. C. vulgaris is marketed as dietary supplement, additive, [33,34] as colourant or food emulsion [35]. They are all in the form of capsules, extracts, tablets or powder [36,37]. The most part is today consumed in Japan as a medical treatment [38, 39]. Indeed C. vulgaris has demonstrated some antitumor and immune-modulating characteristics [40-44]. However, despite its high nutritious protein content and its potentially health benefits, the incorporation of C. vulgaris in some food products are not yet widely used. The main reason remains its dark green colour and its smell being close to the one of a fish, which are not today well accepted in food [45].

Kubatka P. et al. in 2015 mentioned that there has been considerable interest in both clinical and preclinical research about the role of phytochemicals in the reduction of risk for cancer in humans. The aim of this study was to determine the antineoplastic effects of Chlorella pyrenoidosa in experimental breast cancer in vivo and in vitro. In this experiment, the antineoplastic effects of C. pyrenoidosa in the chemoprevention of N-Methyl-N-nitrosourea-induced mammary carcinogenesis in female rats were evaluated. Chlorella powder was administered through diet at concentrations of 0.3% and 3%. The experiment was terminated 14 wk after carcinogen administration. At autopsy, mammary tumors were removed and prepared for histopathological and immunohistochemical analysis. In vitro cytotoxicity assay, parameters of apoptosis, and proliferation after chlorella treatment in human breast adenocarcinoma (MCF-7) cells were carried out. Basic parameters of experimental carcinogenesis, mechanism of action (biomarkers of apoptosis, proliferation, and angiogenesis), chosen metabolic variables, and side effects after long-term chlorella treatment in animals were assessed. Chlorella at higher concentration suppressed tumor frequency by 61% ($P < 0.02$) and lengthened tumor latency by 12.5 d ($P < 0.02$) in comparison with the controls. Immunohistochemical analysis of rat tumor cells showed caspase-7 expression increase by 73.5% ($P < 0.001$) and vascular endothelial growth factor receptor-2 expression decrease by 19% ($P = 0.07$) after chlorella treatment. In a parallel in vitro study, chlorella significantly decreased survival of MCF-7 cells in a dose-dependent manner. In chlorella-treated MCF-7 cells, a significant increase in cells having sub-G0/G1 DNA content and significant increase of early apoptotic and late apoptotic/necrotic cells after annexin V/PI staining assay were found. Decreases in mitochondrial membrane potential and increasing reactive oxygen species generation were observed in the chlorella-treated MCF-7 cells. This study is the first report on the antineoplastic effects of C. pyrenoidosa in experimental breast cancer in vivo and in vitro.

Panahi Y. et al in 2013 showed that smoking is among the established yet modifiable risk factors for cancers, cardiovascular diseases, and pulmonary disorders. Oxidative stress has been proposed as a key mechanism mediating the deleterious consequences of smoking. The present study evaluated the effect of supplementation with Chlorella vulgaris, some nutrient and bioactive green microalgae with proven antioxidant capacity, on the burden of oxidative stress in Iranian smokers. Thirty-eight smokers (mean age: 37.11 +/- 1.69 years; females: 18.4%) were administered C. vulgaris extract (3600 mg/day) for a period of 6 weeks. Fasted serum samples collected at baseline and after the completion of study were analyzed for the concentrations of vitamin C, vitamin E, glutathione, and malonedialdehyde (MDA) as well as activities of superoxide dismutase, glutathione peroxidase, and catalase. Total antioxidant capacity of serum was also determined by the ability of serum to inhibit the formation of ferryl myoglobin radical species. Six-week supplementation with C. vulgaris extract in smokers was associated with marked elevation of all assessed serum antioxidant measures ($p < 0.001$) and significant reduction of MDA levels ($p = 0.002$). After gender segregation, a similar pattern of changes was observed for both male and female subjects apart from lack of significant change in serum vitamin E status in females. Although the magnitude of change in serum vitamin E was significantly greater in males compared to females ($p = 0.014$), there was no significant change in the magnitude of changes for other assessed parameters between the genders. Supplementation with C. vulgaris extract significantly improves antioxidant status and attenuates lipid peroxidation in chronic cigarette smokers. Hence, C. vulgaris might prevent the disease burden and mortality rate associated with smoking.

Emey Suhana MOHD AZAMAI et al. in 2009 showed that Chlorella Vulgaris (CV) has been reported to have antioxidant and anticancer properties. We evaluated the effect of CV on apoptotic regulator protein expression in liver cancer-induced rats. Male Wistar rats (200~250 g) were divided into eight groups: control group (normal diet), CDE group (choline deficient diet supplemented with ethionine in drinking water to induce hepatocarcinogenesis), CV groups with three different doses of CV (50, 150, and 300 mg/kg body weight), and CDE groups treated with different doses of CV (50, 150, and 300 mg/kg body weight). Rats were sacrificed at various weeks and liver tissues were embedded in paraffin blocks for immunohistochemistry studies. CV, at increasing doses, decreased the expression of anti-apoptotic protein, Bcl-2, but increased the expression

of pro-apoptotic protein, caspase 8, in CDE rats, which was correlated with decreased hepatocytes proliferation and increased apoptosis as determined by Bromo deoxy-uridine (BrdU) labeling and terminal deoxynucleotidyl transferase mediated dUTP nick-end labeling (TUNEL) assay, respectively. Our study shows that CV has definite chemo preventive effect by inducing apoptosis via decreasing the expression of Bcl-2 and increasing the expression of caspase 8 in hepatocarcinogenesis-induced rats.

Tammie J. Mc Quistan et al. in 2012 concluded that Recent pilot studies found natural chlorophyll (Chl) to inhibit carcinogen uptake and tumorigenesis in rodent and fish models, and to alter uptake and biodistribution of trace 14 Caflatoxin B1 in human volunteers. The present study extends these promising findings, using a dose-dose matrix design to examine Chl-mediated effects on dibenzo (def,p) chrysene (DBC)induced DNA adduct formation, tumor incidence, tumor multiplicity, and changes in gene regulation in the trout. The dose-dose matrix design employed an initial 12,360 rainbow trout, which were treated with 0–4000 ppm dietary Chl along with 0 - 225 ppm DBC for up to 4 weeks. Dietary DBC was found to induce dose-responsive changes in gene expression that were abolished by Chl co-treatment, whereas Chl alone had no effect on the same genes. Chl co-treatment provided a dose-responsive reduction in total DBC-DNA adducts without altering relative adduct intensities along the chromatographic profile. In animals receiving DBC alone, liver tumor incidence (as logit) and tumor multiplicity were linear in DBC dose (as log) up to their maximum effect dose, and declined thereafter. Chl co-treatment substantially inhibited incidence and multiplicity at DBC doses up to their maximum-effect dose. These results show that Chl concentrations encountered in Chl-rich green vegetables can provide substantial cancer chemo-protection,and suggest that they do so by reducing carcinogen bioavailability. However, at DBC doses above the optima, Chl co-treatments failed to inhibit tumor incidence and significantly enhanced multiplicity. This finding question the human relevance of chemoprevention studies carried out at high carcinogen doses that are not proven to lie within a linear, or at least monotonic, endpoint dose-response range. The results of the present study demonstrate that increasing dietary doses of Chl-enriched spinach extract provide increasing and potent protection against initial DBC-initiated tumor response in two target organs, that protection by the extract was moderately reduced compared with equivalent doses of CHL or purified Chl, and that protection occurred in the absence of demonstrable changes in gene expression patterns. We also determined that the protective efficacy of dietary Chl co-exposure was strongly dependent on the concentration of DBC in the diet, with good protection in both target organs at low carcinogen dose and tumor response but apparent enhancement in liver tumor response at high carcinogen doses and tumor responses not encountered in human populations. These findings emphasize the necessity in the design of cancer chemoprevention studies to select carcinogen doses within a known linear or monotonic dose-response range. In the absence of this information, results derived at high carcinogen doses and high tumor responses may be irrelevant for human intervention.

Bee Pollen is increasing the immune system response and increasing the blood PH as well as it has a complete dosage of enzymes and anti-parasitic effects. Bee pollen is a valuable apitherapeutic product greatly appreciated by the natural medicine because of its potential medical and nutritional applications. It demonstrates a series of actions such as antifungal, antimicrobial, antiviral, anti-inflammatory, hepato-protective, anticancer immune-stimulating, and local analgesic. Its radical scavenging potential has also been reported. Beneficial properties of bee pollen and the validity for their therapeutic use in various pathological condition have been discussed in this study and with the currently known mechanisms, by which bee pollen modulates burn wound healing process. In adults, 20-40g is applied therapeutically every day. If a teaspoon is 7,5g of pollen, it can be concluded that one dose is 3-5 teaspoons of this product for adults and 1-2 teaspoons for children. Pollen is usually taken 3 times a day before eating. The time of treatment is 1–3 months, but it can be repeated 2-4 times a year. The most appropriate period for treatment is between winter and spring and between summer and autumn. Generally, a smaller dose of pollen is used in the combination therapy, alongside other medicaments and in chronic diseases [46].

Chlorophyll is abundant in Chlorella Vulgaris and it has many anticancer benefits. All green plants also contain chlorophyll, the light-collecting molecule. Chlorophyll and its derivatives are very effective at binding polycyclic aromatic hydrocarbons (carcinogens largely from incomplete combustion of fuels), heterocyclic amines (generated when grilling foods), aflatoxin (a toxin from molds in foods which causes liver cancer), and other hydrophobic molecules. The chlorophyll-carcinogen complex is much harder for the body to absorb, so most of it is swept out with the feces. The chemo-protective effect of chlorophyll and its derivatives has been tested in laboratory cell cultures and animals [47, 48]. There is so much compelling evidence for anti-carcinogenic effects of chlorophyll that a prospective randomized controlled trial is being conducted in Qidong, China to see if chlorophyllin can reduce the amount of liver cancer cases, which arise from aflatoxin exposure in their foods (corn, peanuts, soy sauce, and fermented soy beans). A 55% reduction in aflatoxin-DNA adducts were found in the group that took 100 mg of chlorophyll in three times a day [49]. It was supposed that the chlorophyllin bound up aflatoxins, but there were chlorophyllin derivatives also detected in the sera (which had a green tint to it) of the volunteers who took the supplement, indicating a possible role in the body besides binding carcinogens in the gut [50].

Supplementation with Chlorella early in the morning in fasting state benefits cancer patients by improving the blood PH and increasing the absorption of vitamins and minerals. There have been many studies which approves the supplementation with Chlorella. Chlorella gets its name from the high amount of chlorophyll it possesses. Chlorella contains more chlorophyll per gram than any other plant. Chlorophyll is one of the greatest food substances for cleansing the bowel and other elimination systems, such as the liver and the blood.

Nutrient Mixture Plus Specific Ketogenic Diet: Weakening Cancer Cells and Preventing Metastasis

The SCTP advocates the use of a nutrient mixture of vitamin C, the essential amino acid l-lysine and the non-essential amino acid L-proline. The therapeutic use of this combination was initially described by Dr. Mathias Rath and Dr. Linus Pauling in the 1990s [51, 52] and is purported to counter the mechanisms that lead to the metastasis of cancer [53]. Green tea was added to the nutrient mixture as it was found to improve the effect [53,54]. In Henderson's book, Cancer-Free, Henderson makes the claim that this nutrient mixture will slow down or completely stop the spread of the cancer [1]. The Dr. Rath Research Institute in California conducts research on patho-mechanisms including cancer, and on the beneficial effects of micronutrients in various chronic diseases. They have published over two dozen studies from 2004 to 2009 related to the use of the nutrient mixture for cancer. These studies were performed on human cancer cell lines and mice models. Cancers studied include skin cancer, liposarcoma, glioma, osteosarcoma, testicular cancer, melanoma, lung, renal adenocarcinoma, cervical, mesothelioma, bladder, fibrosarcoma, ovarian, mammary, breast, pancreatic, prostate, colon, Ewing's sarcoma and lymphoma [55-80]. The primary focus of these studies has been to assess the ability of this nutrient mixture to inhibit the expression of enzymes known as Matrix Metalloproteinases (MMPs). MMPs have been found to be up-regulated in nearly every type of human cancer and are correlated with advanced stage, invasive and metastatic cancers [81]. The studies conducted by the Rath Institute showed a dose dependent inhibition of MMP expression [55-80]. Conversely, the only independent study testing this nutrient mixture's effect on cancer is a German study involving of an animal model of neuroblastoma. Results suggested that this nutrient mixture was ineffective when tested on mice with induced tumors and spontaneous liver metastases. A greater amount of objective, independent review of this nutrient mixture would help provide a basis for more firm conclusions to be drawn. Studies involving independent components of the mixture do exist. Demeule, Brossard, Page, Gingras and Beliveau (2000) published a study confirming the use of green tea polyphenols to inhibit MMP activity in rat and human tissue, with catechins epigallocatechin gallate and epicatechin gallate showing the greatest level of MMP inhibition [82].

Ketosis

Ketosis is a metabolic condition in which ketone bodies are the supply of energy in the blood. In contrast to a condition of glycolysis, in which blood glucose provides most of the energy. In nutritional process of ketosis, the serum concentration of ketone bodies goes over 0.5 mM in blood and with low and stable levels of insulin and blood glucose [83, 84]. It is almost always generalized with hyperketonemia, that is, an elevated level of ketone bodies in the blood throughout the body. Ketone bodies are formed by ketogenesis when liver glycogen stores are depleted or from metabolising medium-chain triglycerides [85]. The main ketone bodies used for energy are acetoacetate and β-hydroxybutyrate [86], and the levels of ketone bodies are regulated mainly by insulin and glucagon [87]. Most cells in the body can use both glucose and ketone bodies for fuel, and during ketosis, free fatty acids and glucose synthesis, which is called gluconeogenesis, fuel the remainder. Longer-term ketosis may be the result of staying on a very low-carbohydrate diet, and deliberately induced ketosis serves as a medical intervention for various conditions, such as intractable epilepsy, and the various types of diabetes [88]. In glycolysis, high levels of the hormone insulin promote the storage of body fat and block release of fat from adipose tissues, but in ketosis, fat reserves are released and consumed for energy. Therefore, ketosis is sometimes referred to as the body's fat burning mode [89].

Ketogenic Diet

Ketogenic diet is a kind of regime which uses high fat content and low carbohydrate. This diet changes the metabolic state into the condition called Ketosis. After several days, fat becomes your body's primary energy source which causes an increase in the levels of compounds which is called "ketones" in the blood [90]. In general, a ketogenic diet used for weight loss is about 60-75% of calories as fat, with 15-30% of calories from protein and 5-10% of calories from carbs. However, when a ketogenic diet is being used therapeutically for the treatment of cancer, the fat content may be significantly higher that is up to 90% of calories, and the protein content lower [91].

There are several other mechanisms that may explain how a ketogenic diet can aid in cancer treatment. Firstly, eliminating carbs can quickly lower calorie intake, reducing the energy available to the cells in your body. In turn, this may slow down tumor growth and the cancer's progression. Insulin is an anabolic hormone. That means when it is present in the blood, it makes the growth of cancer cells. Therefore, lower insulin or blood glucose, may slow tumor growth [92,93].

Cancer cells cannot use ketones as fuel. Research shows that ketones may reduce tumor size and growth [94].

Researchers have tested the ketogenic diet as an alternative cancer treatment for more than 50 years and most of these studies were done in animals. A large number of these animal studies have demonstrated that ketogenic diet can reduce tumor growth and improve survival rates [95-98]. One 22-day study in mice looked at the differences between the effects of ketogenic and other diets in treatment of cancer. Interestingly, the researchers found that 60% of mice on a ketogenic diet survived. This increased to 100% in mice that got a ketone supplement in addition to the ketogenic diet. However, none of the mice survived on a regular diet or better told a diet contained carbohydrates [99]. Another study in mice tested a ketogenic diet with or without oxygen therapy [100]. The result is obvious. A ketogenic diet increased survival time by 56%. This number increased to 78% when combined with oxygen therapy. These results prove that cancer cells starve in the lack of glucose and in high amounts of oxygen in blood [101].

Presently, limited researches do seem to show that a ketogenic diet may reduce tumor size and rate of progression in certain cancers. One of the few documented published

case studies was performed on a 65-year-old woman with brain cancer. Following surgery, she received a ketogenic diet. Meanwhile, the tumor's progression slowed. But, 10 weeks after returning to a normal diet, she experienced a significant increase in tumor growth [102].

Similar case reports examined the reactions to a ketogenic diet in two young girls who were undergoing treatment for advanced brain cancer. Researchers observed that glucose uptake was decreased in the tumors of both patients. One of the girls reported that her quality of life had improved and remained on the diet for 12 months. During that time her cancer showed no further progression [103]. One study monitored tumor growth in response to a high-carb versus a ketogenic diet in 27 patients with cancer of the digestive tract. Tumor growth increased by 32.2% in patients who received the high-carb diet, but decreased by 24.3% in the patients on the ketogenic diet [104]. In another study, three out of five patients on a ketogenic diet combined with radiation or chemotherapy experienced complete treatment. More interesting, the other two participants found the disease progressed after they stopped the ketogenic diet [102-104].

In conclusion, the ketogenic diet is a very low carbohydrate, high fat diet. For cancer treatment, fat intake may be as high as 90% of total calorie intake. There are also some mechanisms that suggest a ketogenic diet may help prevent the development of cancer in the first place. As a matter of fact, it may reduce several of the main risk factors for cancer. A few small studies and researches in humans suggest that a ketogenic diet may help slow the progression of cancer. But, more research is needed. Beyond lowering blood sugar, the ketogenic diets may also help treat cancer via other mechanisms. These include lowering calories, reducing insulin and increasing ketones. In animals, the ketogenic diet seems to be a promising alternative treatment for cancer. Ketogenic diet can lower blood sugar levels which in turn helps reduce tumor growth and even starve cancer cells of energy they use to respire.

Cottage Cheese and Flax Oil Mixture (Budwig Diet)

Budwig's theory is based upon the work of Otto Warburg [105]. Warburg was an earlier Nobel Prize Laureate for the discovery of the nature and action of the respiratory enzyme [106], the first of the so-called yellow enzymes, or flavoproteins [107]. Warburg's scientific efforts produced a large body of work and publications in highly respected journals such as Science [108-110]. According to Budwig, Warburg theorized that cellular respiration, like many chemical reactions, was dependent upon substrate availability, specifically a sulphydryl group and an unknown saturated fatty acid, which he failed to identify [105]. Budwig, although supportive of Warburg's work, believed he was looking for the wrong fatty acid. From 1949 to 1952, Budwig and colleague H.P. Kaufmann developed new paper chromatography techniques to identify and quantify fatty acids, the success of which she says initiated widespread research into blood lipids [20,111-122]. Budwig describes how in 1953 she applied these techniques to blood samples of healthy and sick individuals, documenting the differences in fatty acid profiles [13,112], making her one of the first scientists to question the health implications of fat consumption [20]. Modifying the type of dietary fat became the foundation for the development of the Budwig Diet [113]. The modification and reduction of dietary fats in the prevention and treatment of cardiovascular disease and cancer has featured prominently in the last 50 years of dietary research [114,115].

Budwig believed that patients with cancer required highly unsaturated fatty acids, specifically Linoleic Acid (LA) and linolenic acid (LNA) to act as raw materials for cell membrane formation and to drive cellular respiration [105]. Diets lacking these fatty acids offered limited substrate for reactions and resulted in an oxygen poor environment, impeding cellular metabolism. Flaxseed oil, which contains 18–20% LA, 58–60% LNA and lesser amounts of saturated and monounsaturated fat [116], is a key component of Budwig's diet. It was Budwig's theory that when the highly unsaturated fatty acids of flaxseed oil interacted with sulphydryl groups from cottage cheese, the stored energy in the fatty acids would be released remedying the oxygen poor environment [105] (p. 102). Mechanisms of carcinogenesis have evolved since Budwig's time. In the last twenty years, acceptance has grown for endogenous mechanisms of carcinogenesis including Oxygen Free Radicals (OFR). OFR's arise from the cellular oxygen reduction reactions and are highly reactive. They attack cell components such as lipids, damage DNA damage and induce mutations [117,118].

The sulphydryl groups, cysteine and methionine, in cottage cheese, were described by Budwig as substances that facilitated the mobilization of fat by increasing solubility [119]. Through her experiments using paper chromatography Budwig found that blending sulphydryl containing cottage cheese with flaxseed oil would improve the solubility of the flaxseed oil, a reaction that did not occur with the saturated fats derived from pork fat. She reasoned that the sulphydryl groups in the amino acids, hydrogen bonded with the unsaturated fatty acids, forming a lipoprotein [105]. Lipoproteins are the building blocks of the phospholipid bilayer, or as Budwig called them -the external skin of the cell‖ [120-132]. The proper function of cellular membranes is vital as it mediates the flow of materials in and out of the cell. Budwig felt the combination of these two substances (i.e., cottage cheese and flaxseed oil) was important because the bond created by the opposing charges generated the -electromotor force‖ of a lipoprotein [105], which she claimed provided the only path for fast and focused transport of electrons in biological systems [105]. It was not until 1978 that Peter Mitchell received a Nobel Prize for his work on energy production in mitochondria [121-125,132].

In SCTP, the cottage cheese is organic and high fat and the amount is 2/3 Cup (mixed with flaxseed oil). The flaxseed oil is cold press and 6 Tablespoons (mixed with cottage cheese).

The inclusion of this supplement in the SCTP is part of the megavitamin trend popularized in the seventies. The multivitamin/mineral supplement is a combination including 65 vitamins, minerals, essential fatty acids, amino acids, anti-oxidants, digestive enzymes, herbs and superfoods [133] (Table 1 and 2).

Medical Ozone Therapy Applied in SCTP

Medical ozone is produced in varying concentrations in SCTP. The quantity of ozone in comparison with the quantity of oxygen in the gas stream is called percent concentration. It is measured in micro grams (ug) of ozone per milliliter (or cc) of the mixture.

A liter of oxygen weights 1.4 grams. Therefore; 0.5% x 1.4 gm = 7 ug/cc, 1.0% x 1.4 gm = 14 ug/cc, 1.5 x 1.4 gm = 21 ug/cc, 2.0% x 1.4 gm = 28 ug/cc, 2.5% x 1.4 gm = 35 ug/cc, 3.0% x 1.4 gm = 42 ug/cc, 3.5% x 1.4 gm = 49 ug/cc, 4.0% x 1.4 gm = 56 ug/cc, 4.5% x 1.4gm = 63 ug/cc, 5.0% x 1.4 gm = 70 ug/cc.

5% or 70 ug/cc is considered to be the upper limit of the concentration for the internal use of medical ozone. [S. Zaminpira, S. Niknamian, The prime cause and treatment of cancer.

The medical internal ozone therapy in SCTP begins with the lowest concentration in the first week and continues till

Table 1: Supplemental nutrient intake recommended in the Bill Henderson Protocol compared to Institute of Medicine's DRIs.

Nutrient	Total daily amount advised in the BHP [1] (pp. 57–59)	Daily recommended intake (male 51–70 y) [7]	Daily recommended intake (female 51–70 y) [7]	Tolerable upper intake level (adults 19–70 y) [99
Vitamin A	3030 µg/10000 IU	900 µg or2970 IU	700 µg or 2310 IU	3000 µ g/day
Vitamin C	5000 mg	90 mg	75 mg	2000 mg/day
Vitamin D	7000 IU	10 µg or 400 IU	10 µg or 400 IU	50 µ g/day
Vitamin K2	120 µg	120 µg	90 µg	ND
Vitamin E	800 IU/536 mg	15 mg	15 mg	1000 mg/day
Thiamin	300 mg	1.2 mg	1.1 mg	ND
Riboflavin	100 mg	1.3 mg	1.1 mg	ND
Niacin	500 mg	16 mg	14 mg	35 mg/day
Vitamin B6	220 mg	1.7 mg	1.5 mg	100 mg/day
Folate	800 µg	400 µg	400 µg	1000 µg /day
Vitamin B12	1000 µg	2.4 µg	2.4 µg	ND
Biotin	600 µg	30 µg	30 µg	ND
Pantothenic acid	300 mg	5 mg	5 mg	ND
Iodine	200 µg	150 µg	150 µg	1100 µ g/day
Magnesium	1000 mg	420 mg	320 mg	350 mg/day
Zinc	40 mg	11 mg	8 mg	40 mg
Selenium	400 µg	55 µg	55 µg	400 µ g/day
Copper	4000 µg	900 µg	900 µg	10000 µ g/day
Manganese	11 mg	2.3 mg	1.8 mg	11 mg/day
Chromium	400 µg	30 µg	20 µg	ND
Molybdenum	200 µg	45 µg	45 mg	2000 µ g/day
Potassium	200 mg	4.7g	4.7 g	ND
Choline	200 mg	550 mg	425 mg	3.5 g/day
Vanadium	0.300 mg	ND	ND	1.8 mg/day
Boron	2 mg	ND	ND	20 mg
Quercetin	100 mg	ND	ND	ND
N-acetyl cysteine	1200 mg	ND	ND	ND
Trace mineral complex	50 mg	ND	ND	ND
PABA	60 mg	ND	ND	ND
Inositol	200 mg	ND	ND	ND
Silica	52 mg	ND	ND	ND
Rutin	20 mg	ND	ND	ND
Hesperidin	20 mg	ND	ND	ND
Beta Carotene	15000 IU	ND	ND	ND
Tocotrienols	40 mg	ND	ND	ND
Coenzyme Q10	100 mg	ND	ND	ND
Alpha lipoic acid	20 mg	ND	ND	ND
Lutein	12 mg	ND	ND	ND
Lycopene	6 mg	ND	ND	ND
EPA (eicosapentaenoicacid)	200 mg	ND	ND	ND
DHA (docosahexaenoicacid)	300 mg	ND	ND	ND
Fish Oil (Mercury Free)	5000 mg	1600 mg	1100 mg	ND
Gamma linolenic acid	100 mg	ND	ND	ND
Pancreatin	100 mg	ND	ND	ND
Lipase	20 mg	ND	ND	ND
Cellulase	20 mg	ND	ND	ND
Maltase	20 mg	ND	ND	ND
Protease	20 mg	ND	ND	ND
Amylase	20 mg	ND	ND	ND

Table 2: Specific doses for supplements suggested in the SCTP.

Daily advantage herbal	Daily dose suggested in SCTP
Chlorella Vulgaris	8000 mg (Early in the morning in the fasting state with water)
Turmeric	500 mg (Three times per day with meal)
L-Taurine	400 mg
Bee Pollen	5000 mg mixed with VCI protein powder
Acetyl-L-Carnitine	2000 mg
Green tea extract	500 mg 30 minutes before lunch
Panax Ginseng	100 mg with breakfast
Alpha Lipoic Acid (ALA)	**300** /Day

the upper limit concentration. The dosage will go higher every week.

Combination of the Specific Keto-Diet (SKD), Intravenous Ozone Therapy (IOT) and Hyperbaric Oxygen Therapy (HBO2T)

In a research by Dr. Somayeh Zaminpira nad Dr. Sorush Niknamian in 2017, they have done the treatment based on the special model of ketogenic diet and ozone therapy on the 54 cancer patients, including; Liver cancer, Colorectal cancer, kidney cancer, brain metastatic tumors, breast cancer, lung cancer. This was a double blind controlled study which has done at the Violet Cancer Institute (VCI) in Iran/Tehran. 10 patients with the liver cancer, 5 patients with the kidney cancer, 11 with brain metastatic tumors, 18 patients with the breast cancer tumors, 5 patients with the lung cancer and 5 patients with colorectal cancer. The methodology of this study was based on 5 days of water fasting to make the normal cells go to the catabolism state, after 5 days we started the Specific Keto-Diet (SKD) the 80 percent saturated fat including MCT and animal and coconut saturated fats. 15 percent protein powder with the lowest glutamine which we have produced at the Violet laboratory, and 5 percent complex carbohydrates with the highest fiber. After 3 months of this study the average results of the reduction in cancer tumors by MRI device were: 45 Percent decrease in tumor size in lung cancer tumors, 25 percent decrease in tumor size in colorectal cancer tumors. 75 percent decrease in tumor size in breast cancer tumors. 62 percent decrease in tumor size in liver cancer tumors. 54 percent decrease in tumor size in kidney cancer tumors. 87 percent decrease in tumor size in brain cancer tumors [134].

Specific Keto-Diet used by Dr. Somayeh Zaminpira and Dr. Sorush Niknamian contains 80% fat in the form of animal and herbal saturated fat (Butter, Tallow and coconut oil), 15% protein and 5% complex-high fiber carbohydrate. The source of carbohydrate used in this protocol is mainly vegetables with high sulfur compounds.

The fats used by the patients were only organic saturated fats. Organic cow butter, organic cow tallow and coconut oil. Medium Chain Triglyceride (MCT) in this research is not used and they used extra virgin coconut oil.

Almost 50% of the fatty acids in coconut oil is the 12-carbon Lauric-Acid. When lauric-acid is digested, it forms a substance called monolaurin. Both lauric-acid and monolaurin can kill harmful pathogens like bacteria, viruses and fungi. Lauric-acid in extra virgin coconut oil has the protective effect against cancer and heart disease. This important and beneficial compound can be found in human milk that is beneficial to infants in reducing the cancer risk and future heart disease.

HBO2D is done on all cancer patients 3x/week plus ozone therapy. Hyperbaric oxygen therapy (HBO2T) involves administration of 100% oxygen at elevated pressure (greater than sea level, or 1 ATA). HBO2T increases plasma oxygen saturation which facilitates oxygen delivery to the tissue independent of hemoglobin O2 saturation [190]. The potential benefit of using HBO2T to combat the cancer-promoting effects of tumor hypoxia is clear. HBO2T alone has been shown to inhibit tumor growth, reduce tumor blood vessel density, and induce the preferential expression of anti-cancer genes in rat models of mammary tumors [191]. Additionally, radiation and many chemotherapy drugs work by producing free radicals within the tumors, leading to cell death. HBO2T enhances tumor-cell production of reactive oxygen species which contributes to the synergistic effects of HBO2T as an adjuvant treatment to standard care. Indeed, HBO2T enhances the efficacy of both radiation and chemotherapy in animal models [192-196]. In normal tissues, decreased oxygen availability inhibits mitochondrial production of ATP, stimulating an up-regulation of glycolytic enzymes to meet energy needs by substrate level phosphorylation production of ATP. Thus, the cellular response to tumor hypoxia is mediated by several of the same pathways that are overly active in cancer cells with mitochondrial damage and high rates of aerobic glycolysis. This suggests that the ketogenic diet and HBO2T could target several overlapping pathways and tumorigenic behaviors of cancer cells [196].

Dietary Restrictions of SCTP

The SCTP prohibits dairy products (with the exception of cottage cheese), gluten, sugar, processed food, alcohol, vegetable oils, margarine, fruit juices and inorganic meat. Links between cancer and diet have been investigated [135-138].

Inorganic Meat

Conclusions about cancer risk and the consumption of meat are difficult to make given the state of current evidence. In a case control study from Uruguay, 846 cases and 846 controls were followed estimating meat consumption and lung cancer risk. Total meat consumption of red meat and processed meat are reported to be associated with an increased risk of cancer, while total intake of white meat, poultry and fish did not increase risk [139]. Intake of red meat also appears to be associated with an increased risk of breast cancer [140,141] although the variables controlled in studies vary widely such as menopausal status and cooking practices, making it difficult to draw definitive conclusions [142,143]. The intake of saturated fat, found in higher amounts in animal products, has been linked to breast cancer [144]. Colon cancer risk has also been correlated with intakes of red meat, pan fried meat and processed meat mutagens [145]. Fish and specifically the consumption of omega 3 fatty acids, has been associated with a lowered

cancer risk, and more specifically, decreased mortality from prostate cancer [140-145].

Dairy

Dairy products due to their high amounts of lactose is prohibited I cancer patients when ketogenic diet is introduced in SCTP.

There are hypotheses about the risks of milk and milk product consumption associated with an increase in growth hormone/insulin like growth factor-1 that is triggered by the ingestion of milk protein [146]. The recent publication from the European Prospective Investigation into Cancer and Nutrition reported an association between high dairy intake, high serum concentrations of IGF-1 and an increased risk for prostate cancer [147]. Conflicting evidence exists regarding the role of dairy in the development of colorectal cancer. It has been reported that high dairy intakes during childhood resulted in a near tripling in the odds of developing colorectal cancer in adulthood [148]. Conversely, two recent studies suggest that a higher intake of dairy products is associated with a lower risk of colon cancer [149, 150]. High calcium intake has also been correlated with a lower risk of colorectal cancer [151]. Fortified milk and milk products are also a source of vitamin D, which has been associated with a reduced risk of cancer [149-151]. The BHP recommends a supplement containing 40 μg /1600 IU of vitamin D [33], four times the current daily recommended intake of vitamin D.

Gluten

Gluten, found in a variety of grains including wheat, does contain several proteins that are known causes of respiratory and food allergies as well as contact hypersensitivity, in certain individuals [152], but this reaction is not associated with an increased risk of cancer. Celiac disease is described as a small intestine mucosal disorder that is gluten dependent and often characterized by nutrient malabsorption, diarrhea and weight loss [153]. A known complication of celiac disease is the development of bowel cancers [41] (p. 713). A 2009 article reported that if celiac disease was first diagnosed later in life, lymphoma was detected more often than in cases of early diagnosis, the possible culprit being duration of exposure to gluten [154]. The same study also postulated that those with celiac disease may actually be at a lower risk of gastric and colon cancers than previously thought due the celiac characteristic symptoms of impaired absorption and quick excretion of fat, fat soluble substances, hydrocarbons and other potentially carcinogenic substances [153, 154]. In the general population however, an increased risk of cancer from gluten ingestion has not been established.

Sugar and Processed Foods

Foods high in sugar can replace nutrient dense foods, delivering more calories and fewer nutrients. Poor nutrition can lead to numerous health consequences including anemia, delayed immune response and prolonged wound healing. Foods with a high glycemic index have been linked to insulin resistance, which has been correlated with an increased risk for pancreatic cancer [155, 156]. In a 2009 cohort study intended to assess this risk, those with the highest intake of fructose and glucose were found to be at higher risk for pancreatic cancer [157]. Positive associations have also been made between diets with a high glycemic load and both esophageal adenocarcinoma [158] and breast cancer [159-161]. High sugar intake is associated with weight gain and obesity. A 2008 study reported that an estimated 33,966 new cancers (4% of all estimated cancers) in males and 50,535 (7% of all estimated cancers) in females diagnosed in 2007, or 6% of all cancers, may be potentially attributable to obesity [162].

Without the impact of rising obesity rates, incidence rates might have declined (instead of remaining stable) from 1988–1994 to 2001–2004 for uterus, breast and certain other cancers‖ [158]. The following, by the World Cancer Research Fund and The American Institute for Cancer Research, is a list of mechanisms by which obesity can increase cancer risk as reported in their publication —Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perspective‖: Obesity causes an elevation of insulin and leptin levels, which can promote the growth of cancer. Elevated leptin levels are associated with colorectal and prostate cancer. Insulin resistance is increased, causing the pancreas to compensate by increasing insulin production. Hyperinsulinaemia is correlated with an increased risk of cancers of the colon and endometrium, and possibly of the pancreas and kidney. Higher levels of body fat increase the level of estradiol in men and women and may also raise testosterone levels in women. Increased levels of these sex steroid hormones are strongly associated with endometrial cancer and postmenopausal breast cancer and may increase risks for colon and other cancers. Adipose tissue promotes inflammatory factors in the body. Obese individuals have higher concentrations of interleukin-6, C-reactive protein and tumour necrosis factor. Leptin, which again is raised in cases of obesity, also functions as an inflammatory cytokine. Chronic inflammation can increase one's risk of cancer [40].

There is also a potential link between sugar and cancer involving the effect of sugar on the immune system. It has been reported that the daily ingestion of 75-100 g of simple sugars, including glucose, sucrose, fructose and honey has been found to induce a fifty percent drop in the activity of white blood cells for two to five hours [164]. The immune system plays a central role in the elimination of altered and unhealthy cells, including cancer cells [156-162].

Alcohol

Alcohol intake has been associated with an increased risk for certain cancers including breast, rectal and pancreatic cancers [163,164]. There is conflicting data on the effect of alcohol consumption and prostate cancer. While heavy drinking, 50 g or more per day, has been positively associated with prostate cancer [165], 36 g or more per day has been shown to decrease the risk of benign prostatic hyperplasia [166], a marker for prostate cancer risk. A 2009 study evaluated lifetime alcohol exposure and its link to cancer risk in men and concluded that moderate and high alcohol consumption increased risk for cancers of the esophagus, stomach, colon, liver, pancreas, lung and prostate. Cancer association was strongest for beer and to a lesser extent

spirits [167]. Red wine, on the other hand, may offer some protective benefits against a number of degenerative diseases. In a study looking at the effect of alcohol on breast density, a strong intermediate marker for breast cancer risk, red wine showed a consistent inverse association, unlike other types of alcohol [168]. The protective mechanism associated with red wine is often attributed to red wine's content of resveratrol [169]. A recent study suggested that the benefits of red wine include, but are not limited to resveratrol, but may also involve its red pigments [170].

Industrial Vegetable Oils and Margarine

Increased consumption of processed vegetable oils and margarine has led to an epidemic of harmful structural changes in cell membranes. Although it is true that vegetable oils can lower Total and LDL cholesterol levels, they also lower HDL which is a negative effect. Vegetable oils change the structure of the cell membrane which results in an increase in the Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS) in human cells. This increase may be one of the prime drivers behind the transformation of normal cells into cancer cells. The steep increase in consumption of these harmful products is likely to be one of the leading causes of the current epidemic of heart disease and cancer.

Increasing the cancer incidence and consumption of margarine and vegetable oils is highly discussed in Fat & Cholesterol Myths and Obesity & Cholesterol Myths by Sorush Niknamian and Somayeh Zaminpira.

Discussion

SCTP is a protocol introduced by Dr. Somayeh Zaminpira nad Dr. Sorush Niknamian as the treatment for cancer with the lowest side effects. There have been many researches done by the cancer scientists to figure out which diet and what supplements can be used in the treatment of cancer.

We have studied over 700 researches and reviews and done a controlled research in the Violet Cancer Institute (VCI) on 54 seriously ill cancer patients to test our study. After 6 months of this research all the patients cured completely without any serious side effects. This protocol is based on Otto Warburg, Dr. Johanna Budwig and Bill Henderson Protocol (BHP) with a serious revision.

As a large component of his six component diet, Bill Henderson suggests cottage cheese and flaxseed oil recommended by Dr. Johanna Budwig in the 1950s. While it is known that the essential fatty acids found in flaxseed oil, as well as methionine and cysteine, are important in human health, with modern advancements to our understanding in areas of human physiology, lipid metabolism and lipoproteins, Budwig's fervent belief in these combined substances as cure for cancer appears overly simplistic. Although Budwig lived until 2003, and was actively lecturing until the late 1990s, there does not appear to have been any attempts to test her theories using modern technology or different source materials, such as fish oil.

The combination of lysine, proline, ascorbic acid and green tea extract has been studied by The Dr. Rath Research Institute and based on their published work, has shown this nutrient mixture to be a promising anti-cancer proliferation agent [105-132].

The specific Keto-Diet of this protocol is 80% saturated fat, 15% Protein and 5% complex-high fiber carbohydrates which has had marvelous effects in cancer patients. The VCI Protein Powder with the lowest glutamine is a good source of protein taken by the patients to reduce the incidence of Cachexia in cancer patients.

All vitamins and minerals specially Vitamin E and Folate should be natural since these two vitamins in the synthetic form may increase the probability of many types of cancer incidences. Dairy, sugar, fruit juice, vegetable oil and margarine are prohibited in this protocol due to high glucose contents. Supplementation with 5000 mg of fish oil is needed due to reaching the amounts of DHA and EPA mentioned in table 1.

Supplementation with probiotic is a must in cancer patients to increase the rate of vitamins and mineral absorption including vitamin B12. Probiotics are defined as microorganisms that are believed to provide health benefits when consumed. The term probiotic is currently used to name ingested microorganisms associated with benefits for humans and animals. Probiotics may compete against pathogens for the same essential nutrients, leaving less available for the pathogen to utilize (A). They may bind to adhesion sites, preventing pathogen attachment by reducing the surface area available for pathogen colonization (B). Signaling of immune cells by probiotics may result in the secretion of cytokines, targeting the pathogen for destruction (C). Finally, probiotics may attack pathogenic organisms by releasing bacteriocins, killing them directly (D).

Motevaseli et al. in 2017 showed that Probiotics are defined as live bacteria and yeasts that exert beneficial effects for health. Among their various effects, anti-cancer properties have been highlighted in recent years. Such effects include suppression of the growth of microbiota implicated in the production of mutagens and carcinogens, alteration in carcinogen metabolism and protection of DNA from oxidative damage as well as regulation of immune system. We performed a computerized search of the MEDLINE/ PUBMED databases with key words: cancer, probiotics, lactobacilli, metastasis and invasion. Cell line studies as well as animal models and human studies have shown the therapeutic effects of probiotics in reduction of invasion and metastasis in cancer cells. These results support the beneficial effects of probiotics both in vitro and in vivo. However, pre-clinical or clinical studies are not enough to decide about their application [171].

Yu AQ et al. in 2016 mentioned that the human gut microbiota has a significant effect on many aspects of human physiology such as metabolism, nutrient absorption, and immune function. Imbalance of the microbiota has been implicated in many disorders including inflammatory bowel disease, obesity, asthma, psychiatric illnesses, and cancers. As a kind of functional foods, probiotics have been shown to play a protective role against cancer development in animal models. Clinical application of probiotics

indicated that some probiotic strains could diminish the incidence of postoperative inflammation in cancer patients. Chemotherapy or radiotherapy-related diarrhea was relieved in patients who were administered oral probiotics. The present review summarizes the up-to-date studies on probiotic effects and the underlying mechanisms related to cancer. At present, it is commonly accepted that most commercial probiotic products are generally safe and can improve the health of the host. By modulating intestinal microbiota and immune response, some strains of probiotics can be used as an adjuvant for cancer prevention or/and treatment [172].

Some strains of LAB may modulate inflammatory and hypersensitivity responses, an observation thought to be at least in part due to the regulation of cytokine function [173-176]. Clinical studies are assessing whether they can prevent recurrences of inflammatory bowel disease in adults, as well as affect milk allergies [177]. How probiotics may influence the immune system remains unclear [178].

Probiotics are being studied for their potential to influence inflammatory bowel disease. There is some evidence to support their use in conjunction with standard medications in treating ulcerative colitis and no evidence of their efficacy in treating Crohn's disease [179-181].

A live formulation of lyophilized Bifidobacterium breve, Bifidobacterium longum, Bifidobacterium infantis, Lactobacillus acidophilus, Lactobacillus plantarum, Lactobacillus paracasei, Lactobacillus bulgaricus, and Streptococcus thermophilus (VSL3) has shown effectiveness in the small clinical trials, some of which were not randomized nor double-blinded, that had been done as of 2015; more high-quality clinical trials are needed to determine safety and effectiveness [182,183]. Probiotics are under study for their potential to affect irritable bowel syndrome, although uncertainty remains around which type of probiotic works best, and around the size of possible effect [184,185]. Probiotic treatment has been studied as a means of addressing disorders associated with vitamin deficiencies including those of vitamin K, folic acid, and vitamin B12 to increase their absorption [186-188].

Probiotics should be taken two times a day with meal. Also, we recommend eating fermented foods for absorbing vitamin K2 including: Kimchee, Miso and Natto. Consumption of any non-fermented soy is prohibited in the SCTP. There have been many studies relating the consumption of soybeans and increasing the rates of several cancers which is highly discussed in the book "The Effect of Nutrition in Evolution of Cancer" written by Dr. Somayeh Zaminpiraa and Dr. Sorush Niknamian in 2017.

In addition to all above, one important antioxidant which has been shown to be effective in inducing apoptosis and inhibit proliferation of cancer cells relative to normal cells. is Alpha Lipoic Acid (ALA) and ahould be taken with NAC. The antioxidant Alpha-Lipoic Acid (APA) is a naturally occurring compound that has been shown to possess promising anti-cancer activity because of its ability to preferentially induce apoptosis and inhibit proliferation of cancer cells relative to normal cells. Mantovani, et al. (2002) used alpha-lipoic acid (ALA) at a dosage of 300mg/day and N-acetyl-cysteine at 1800 mg/day [89]. Their data showed long term combined maintenance therapy with rIL 2 + medroxyprogesterone acetate (MPA) + antioxidant agents is feasible, has a very low toxicity, and results in the improvement of clinical outcome [134]. The antioxidants N-Acetylcysteine and ALA markedly reduced the effect of the hormone on tumor necrosis factor-induced caspase activation, attest- ing to the involvement of reactive oxygen species (ROS) in the cross-talk between the hormone and the cytokine [135]. Mantovani, et al. [136] tested the ability of different antioxidant agents, used alone or in combination, to reduce the reactive oxygen species (ROS) levels and to increase the Glutathione Peroxidase (GPx) activity. The study included fifty-six advanced stage cancer patients who were mainly stage III (12.5%) and stage IV (82.1%). Single antioxidants were effective in reducing the ROS levels. The results of ALA use in human cancer chemotherapy and as a chemo-preventive agent by a significant inhibition of the formation of the depurating adducts [137] have been reviewed in light of ALA future inclusion into chemotherapeutic protocols [138,139].

Consumption of 500 ml of vegetable juicing is important by cancer patients to improve the acidity, absorption of mineral magnesium and improving the blood electrolyte.

In addition to the specific keto-diet and nutritional supplements mentioned in the materials and methods, we highly propose intravenous ozone therapy once per week starting with the lowest dosage and increase it every week to the maximum dosage to reduce any side effects or allergic reactions. In addition to ozone therapy, with suggest Hyperbaric Oxygen Therapy (HBOT) tow times per week. There are several studies proven the benefits of HBOT in combination with Ketogenic Diet in the treatment of cancer in animal and human models.

Angela Poff et al. in 2013 mentioned that abnormal cancer metabolism creates a glycolytic-dependency which can be exploited by lowering glucose availability to the tumor. The Ketogenic Diet (KD) is a low carbohydrate, high fat diet which decreases blood glucose and elevates blood ketones and has been shown to slow cancer progression in animals and humans. Abnormal tumor vasculature creates hypoxic pockets which promote cancer progression and further increase the glycolytic-dependency of cancers. Hyperbaric Oxygen Therapy (HBO2T) saturates tumors with oxygen, reversing the cancer promoting effects of tumor hypoxia. Since these non-toxic therapies exploit overlapping metabolic deficiencies of cancer, we tested their combined effects on cancer progression in a natural model of metastatic disease. We used the firefly luciferase-tagged VM-M3 mouse model of metastatic cancer to compare tumor progression and survival in mice fed standard or KD ad libitum with or without HBO2T (2.5 ATM absolute, 90 min, 3x/week). Tumor growth was monitored by in vivo bioluminescent imaging. KD alone significantly decreased blood glucose, slowed tumor growth, and increased mean survival time by 56.7% in mice with systemic metastatic cancer. While HBO2T alone did not influence cancer progression, combining the KD with HBO2T elicited a significant decrease

in blood glucose, tumor growth rate, and 77.9% increase in mean survival time compared to controls. KD and HBO2T produce significant anti-cancer effects when combined in a natural model of systemic metastatic cancer. Our evidence suggests that these therapies should be further investigated as potential non-toxic treatments or adjuvant therapies to standard care for patients with systemic metastatic disease [189].

Ozone has been found to be an extremely safe medical therapy, free from side effects. In a 1980 study done by the German medical society for ozone therapy, 644 therapists were pulled regarding their 384,775 patients, comprising a total of 5,979,238 ozone treatments administered. There were only 40 cases of side effects noted out of this number which represents the incredibly low rate of 0.000007% and only 4 fatalities. Ozone has thus proven to be the safest medical therapy ever devised.

The importance of medical ozone therapy for the treatment of cancer in this protocol in combination with the specific keto-diet is to weaken cancer cells, increasing the amounts of ketone bodies in combination with oxygen, increasing the blood flow and uplifting the immune system response. Ozone therapy is the safest cancer treatment therapy which does not have any serious side effects in human models.

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Conclusion

Cancer is an evolutionary metabolic disease which has introduced as EMHC hypothesis in 2017 by Drs. Somayeh Zaminpira and Sorush Niknamian. Based on this hypothesis, the prime cause of cancer is increasing the amounts of Reactive Oxygen Species (ROS) and through the Butterfly Effect, normal eukaryotic cells become cancer cells. For finding the most effective cancer treatment without serious side effects, we have studied the protocols and clinical researches from 1928-2017. The Sorush Cancer Treatment Protocol (SCTP) is introduced by the authors of this paper which is basically uses Specific Ketogenic Diet (SKD), intravenous ozone therapy, Hyperbaric Oxygen Therapy (HBOT) and consumption of nutritional supplements, vitamins, minerals and superfoods which is highly effective in the treatment of almost all cancers. Water fasting and SKD weakens cancer cells and force them to starvation mode. Intravenous ozone therapy in combination with HBOT saturates cancer cells with oxygen, countering acidity, stimulating the immune system response and increasing the amounts of ROS in cancer cells to force them to apoptosis mode without any side effects in normal cells. The most effective vitamins, minerals and superfoods in the treatment of cancer is mentioned in table 1 and table 2. Consumption of Cottage cheese with flaxseed oil has been used by the Budwig and Bill Henderson Protocols (BP and BHP) with good outcomes. Although BP and BHP have some restrictions, we have revised and completed the nutritional supplementation based on tried and true researches in introducing SCTP. This protocol has been used on 54 seriously ill cancer patients with several types of cancer with the 100% positive results and no side effects in 6 months. SKD mentioned in this protocol is 80% saturated fat (including animal fat and coconut oil without the usage of MCTs) and consumption of industrialized vegetable oils and margarines are completely prohibited due to the increased possibility of cancer incidence and inflammation.

References

1. The Evolution of Electron-Transport Chains, Molecular Biology of the Cell. 4th edition. 2002, Bruce Alberts, Alexander Johnson, Julian Lewis, Martin Raff, Keith Roberts, and Peter Walter; Copyright © 1983, 1989, 1994, Bruce Alberts, Dennis Bray, Julian Lewis, Martin Raff, Keith Roberts, and James D. Watson. Bookshelf ID: NBK26849.
2. Keeling PJ, Archibald JM (2008) Organelle evolution: what's in a name?. Current Biology 18: 345-347.
3. Warburg O (1956) On the Origin of Cancer Cells. Science 123: 309-314.
4. Shoemaker JM (2010) Ozone therapy: History, physiology, indications, results.
5. McLean L (2009) The miracle of ozone therapy.
6. Stoker G (1902) Ozone in chronic middle ear deafness. Lancet 160: 1187-1188.
7. Wells KH, Latino J, Gavalchin J, Poiesz BJ (1991) Inactivation of human immunodeficiency virus type 1 by ozone in vitro. Blood: 78: 1882-1890.
8. Carpendale MT, Freeberg JK (1991) Ozone inactivates HIV at noncytotoxic concentrations. Antiviral Res 16: 281-292.
9. Haug KF, Heidelberg, Rilling S, Viebahn R (1987) The use of Ozone in Medicine. classical medical ozone textbook. 11 edition.
10. Sunnen GV (1988) Ozone in medicine: Overview and future directions. J Adv Med 1: 159-174.
11. Washutti J, Viebahn R, Steiner I (1988) The influence of ozone on tumor tissue in comparison with healthy tissue. Ozone Sci Engg 12: 65-72.
12. Washutti J, Viebahn R, Steiner I (1989) Immunological examinations in patients with chronic conditions under administration of ozone/oxygen mixtures. Ozone Sci Engg 11: 411-417.
13. Zänker KS, Kroczek R (1990) In vitro synergistic activity of 5-fluorouracil with low-dose ozone against a chemoresistant tumor cell line and fresh human tumor cells. Int J Exp Clin Chemother 36: 147-154.
14. Bocci V, Paulesu L (1990) Studies on the biological effects of ozone 1: Induction of interferon on human leucocytes. Haematologica 75: 510-515.
15. Viebahn-Hansler R (1991) Ozone therapy-the underlying therapeutical concept and models of efficacy. Erfahrungs Heilkunde 4: 40.
16. Kawalski H, Sondej J, Cierpiol TE (1992) The use of ozonotherapy in nose correction operations. Acta Chirurgiae Plasticae 34: 182-184.
17. Ozmen V, Thomas WO, Healy JT, Fish JM, Chambers R (1993) Irrigation of the abdominal cavity in the treatment of experimentally induced microbial peritonitis: efficacy of ozonated saline. Am Surg J 59: 297-303.
18. Gérard V, Sunnen (2003) SARS and ozone therapy: Theoretical considerations.
19. Why consider ozone therapy/oxygen Spa as alternative treatment dallas Fort Worth?.
20. Viebahn-Hänsler R (2003) The use of ozone in medicine: Mechanisms of action. Munich 23-25.
21. Chlorella vulgaris (HTML). NCBI taxonomy. Bethesda, MD: National Center for Biotechnology Information. Retrieved 5 December 2017. Other

names: synonym: Chlorella vulgaris var. viridis Chodat includes: Chlorella vulgaris Beijerink IAM C-27 formerly Chlorella ellipsoidea Gerneck IAM C-27.

22. Safi C, Zebib B, Merah O, Pontalier P Y, Vaca-Garcia C (2014) Morphology, composition, production, processing and applications of Chlorella vulgaris: A review. Renewable & Sustainable Energy Reviews 35: 265-278.

23. Beijerinck M W (1890) Culturversuche mit Zoochlorellen, Lichenengonidien und anderen niederen Algen. Bot. Zeitung 48: 781-785.

24. Kiwa Kitada, Siti Machmudah, Mitsuru Sasaki, Motonobu Goto, Yuya Nakashima, et al. (2009) Supercritical CO2 extraction of pigment components with pharmaceutical importance from Chlorella vulgaris. Journal of chemical technology and biotechnology 84: 657-661.

25. Přibyl P, Cepák V, Zachleder V (2012) Production of lipids in 10 strains of Chlorella and Parachlorella, and enhanced lipid productivity in Chlorella vulgaris. Appl Microbiol Biotechnol 94: 549-561.

26. Chisti Y (2007) Biodiesel from microalgae. Biotechnol Adv 25: 294-306.

27. Lee DJ, Liao GY, Chang YR, Chang JS (2012) Coagulation-membrane filtration of Chlorella vulgaris. Bioresour Technol 108: 84-189.

28. Cheng YL, Juang YC, Liao GY, Ho SH, Yeh KL et al. (2010) Dispersed ozone flotation of Chlorella vulgaris. Bioresour Technol 101: 9092-9096.

29. Singh A, Nigam PS, Murphy JD (2011) Renewable fuels from algae: An answer to debatable land based fuels. Bioresource Technology 102: 10-16.

30. Demirbas M F (2011) Biofuels from algae for sustainable development. Applied Energy 88: 3473-3480.

31. Wang K, Brown RC, Homsy S, Martinez L, Sidhu SS (2013) Fast pyrolysis of microalgae remnants in a fluidized bed reactor for bio-oil and biochar production. Bioresource Technology 127: 494-499.

32. Becker E W (1994) Microalgae: biotechnology and microbiology. Volume 10: Cambridge University Press.

33. Morris HJ, Almarales A, Carrillo O, Bermúdez RC (2008) Utilisation of Chlorella vulgaris cell biomass for the production of enzymatic protein hydrolysates. Bioresource technology 99: 7723-7729.

34. Safi C, Charton M, Pignolet O, Silvestre F, Vaca-Garcia C, et al. (2013) Influence of microalgae cell wall characteristics on protein extractability and determination of nitrogen-to-protein conversion factors. Journal of applied phycology 25: 523-529.

35. Servaites JC, Faeth JL, Sidhu SS (2012) A dye binding method for measurement of total protein in microalgae. Analytical biochemistry 421: 75-80.

36. Jafar Seyfabadi, Zohreh Ramezanpour, Zahra Amini Khoeyi (2011) Protein, fatty acid, and pigment content of Chlorella vulgaris under different light regimes. Journal of applied phycology 23: 721-726.

37. Bránykiová I, Maršálková B, Doucha J, Brányik T, Bišová K et al. (2011) Microalgae—novel highly efficient starch producers. Biotechnology and bioengineering 108: 766-776.

38. Choix FJ, de-Bashan LE, Bashan Y (2012) Enhanced accumulation of starch and total carbohydrates in alginate-immobilized Chlorella spp. induced by Azospirillum brasilense: II. Heterotrophic conditions. Enzyme and microbial technology 51: 300-309.

39. de-Bashan LE, Bashan Y, Moreno M, Lebsky VK, Bustillos JJ (2002) Increased pigment and lipid content, lipid variety, and cell and population size of the microalgae Chlorella spp. when co-immobilized in alginate beads with the microalgae-growth-promoting bacterium Azospirillum brasilense. Canadian Journal of Microbiology 48: 514-521.

40. Gonzalez L E, Bashan Y (2000) Increased growth of the microalga chlorella vulgariswhen coimmobilized and cocultured in alginate beads with the plant-growth-promoting bacterium Azospirillum brasilense. Applied and Environmental Microbiology 66: 1527-1531.

41. Fradique M, Batista AP, Nunes MC, Gouveia L, Bandarra NM, et al. (2010). Incorporation of Chlorella vulgaris and Spirulina maxima biomass in pasta products. Part 1: Preparation and evaluation. Journal of the Science of Food and Agriculture 90: 1656-1664.

42. Li HB, Jiang Y, Chen F (2002) Isolation and purification of lutein from the microalga Chlorella vulgaris by extraction after saponification. Journal of agricultural and food chemistry 50: 1070-1072.

43. Fernandes B, Dragone G, Abreu A P, Geada P, Teixeira J, et al. (2012) Starch determination in Chlorella vulgaris—a comparison between acid and enzymatic methods. Journal of applied phycology 24: 1203-1208.

44. Shizhong Liang, Xueming Liu, Feng Chen, Zijian Chen (2004) Current microalgal health food R & D activities in China. Asian Pacific Phycology in the 21st Century: Prospects and Challenges 45-48.

45. Yamaguchi K (1996) Recent advances in microalgal bioscience in Japan, with special reference to utilization of biomass and metabolites: a review. Journal of applied phycology 8: 487-502.

46. S. Bogdanov (2014) Pollen: Production, Nutrition and Health: A Review. Bee Product Science.

47.

48. Chernomorsky S, Segelman A, Poretz RD (1999) Effect of dietary chlorophyll derivatives on mutagenesis and tumor cell growth. Teratog Carcinog Mutagen 19: 313-322.

49. Sarkar D, Sharma A, Talukder G (1994) Chlorophyll and chlorophyllin as modifiers of genotoxic effects. Mutat Res 318: 239-247.

50. Egner PA, Wang JB, Zhu YR, Zhang BC, Wu Y, et al. (2001) Chlorophyllin intervention reduces aflatoxin-DNA adducts in individuals at high risk for liver cancer. Proc Natl Acad Sci U S A 98:14601-14606.

51. Egner PA, Stansbury KH, Snyder EP, Rogers ME, et al (2000) Identification and characterization of chlorin e(4) ethyl ester in sera of individuals participating in the chlorophyllin chemoprevention trial. Chem Res Toxicol 13: 900-906.

52. Rath M, Pauling L (1992) Plasmin-Induced Proteolysis and the Role of Apoprotein(a), Lysine, and Synthetic Lysine Analogs. J Orthomol Med 7: 17-23.

53. Rath M (1992) Reducing the Risk for Cardiovascular Disease with Nutritional Supplements. J. Orthomol Med 7: 153-162.

54. Rath Institute Research, 2009. Dr. Mathias Rath Institute Web site.

55. Demeule M, Brossard M, Pagé M, Gingras D, Béliveau R (2000) Matrix metalloproteinase inhibition by green tea catechins. Biochim Biophys 1478: 51-60.

56. Ivanov V, Ivanova S, Roomi MW, Kalinovsky T, Niedzwiecki A (2007) Naturally produced extracellular matrix inhibits growth rate and invasiveness of human osteosarcoma cancer cells. Med Oncol 24: 209-217.

57. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2004) Anti-tumor effect of ascorbic acid, lysine, proline, arginine, and epigallocatechin gallate on prostate cancer cell lines PC-3, LNCaP, and DU145. Res. Commun Mol Pathol Pharmacol 115-116: 251-264.

58. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2005) Anti-angiogenic effects of a nutrient mixture on human umbilical vein endothelial cells. Oncol Rep 14: 1399-1404.

59. Roomi MW, Roomi N, Ivanov V, Kalinovsky T, Niedzwiecki A, et al. (2005) Inhibitory effect of a mixture containing ascorbic acid, lysine, proline and green tea extract on critical parameters in angiogenesis. Oncol Rep 14: 807-815.

60. M Waheed Roomi, Nusrath W Roomi, Vadim Ivanov, Tatiana Kalinovsky, Aleksandra Niedzwiecki et al. (2005) Modulation of N-methyl-N-nitrosourea induced mammary tumors in Sprague-Dawley rats by combination of lysine, proline, arginine, ascorbic acid and green tea extract. Breast Cancer Res 7: R291-R295.

61. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2005) In vitro and in vivo antitumorigenic activity of a mixture of lysine, proline, ascorbic acid, and green tea extract on human breast cancer lines MDA-MB-231 and MCF-7. Med Oncol 22: 129-138.

62. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2005)

Antitumor effect of a combination of lysine, proline, arginine, ascorbic acid, and green tea extract on pancreatic cancer cell line MIA PaCa-2. Int J Gastrointest Cancer 35: 97-102.

63. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2005) In vivo antitumor effect of ascorbic acid, lysine, proline and green tea extract on human prostate cancer PC-3 xenografts in nude mice: evaluation of tumor growth and immunohistochemistry. In Vivo 19: 179-183.

64. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2005) In vivo antitumor effect of ascorbic acid, lysine, proline and green tea extract on human colon cancer cell HCT 116 xenografts in nude mice: Evaluation of tumor growth and immunohistochemistry. Oncol Rep 13: 421-425.

65. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2005) Antitumor effect of nutrient synergy on human osteosarcoma cells U-2OS, MNNG-HOS and Ewing's sarcoma SK-ES.1. Oncol Rep 13: 253-257.

66. Roomi MW, Roomi N, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Inhibition of pulmonary metastasis of melanoma b16fo cells in C57BL/6 mice by a nutrient mixture consisting of ascorbic Acid, lysine, proline, arginine, and green tea extract. Exp Lung Res 32: 517-530.

67. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Effect of Ascorbic Acid, Lysine, Proline, and Green Tea Extract on Human Osteosarcoma Cell Line MNNG-HOS Xenografts in Nude Mice: Evaluation of Tumor Growth and Immunohistochemistry. Med Oncol 23: 411-418.

68. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Anticancer effect of lysine, proline, arginine, ascorbic acid and green tea extract on human renal adenocarcinoma line 786-0. Oncol Rep 16: 943-947.

69. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Suppression of human cervical cancer cell lines Hela and DoTc2 4510 by a mixture of lysine, proline, ascorbic acid, and green tea extract. Int J Gynecol Cancer 16: 1241-1247.

70. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Inhibition of malignant mesothelioma cell matrix metalloproteinase production and invasion by a novel nutrient mixture. Exp Lung Res 32: 69-79.

71. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Antitumor effect of ascorbic acid, lysine, proline, arginine, and green tea extract on bladder cancer cell line T-24. Int J Urol 13: 415-419.

72. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) In vivo and in vitroantitumor effect of ascorbic acid, lysine, proline, arginine, and green tea extract on human fibrosarcoma cells HT-1080. Med Oncol 23: 105-111.

73. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) Inhibition of matrix metalloproteinase-2 secretion and invasion by human ovarian cancer cell line SK-OV-3 with lysine, proline, arginine, ascorbic acid and green tea extract. J Obstet Gynaecol Res 32: 148-154.

74. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2006) In vivo and in vitro antitumor effect of ascorbic acid, lysine, proline and green tea extract on human melanoma cell line A2058. In Vivo 20: 25-32.

75. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2007) Inhibition of cell invasion and MMP production by a nutrient mixture in malignant liposarcoma cell line SW-872. Med Oncol 24: 394-401.

76. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2007) Inhibition of glioma cell line A-172 MMP activity and cell invasion in vitro by a nutrient mixture. Med Oncol 24: 231-238.

77. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M (2007) Inhibitory effects of a nutrient mixture on human testicular cancer cell line NT 2/DT matrigel invasion and MMP activity. Med Oncol 24: 183-188.

78. Roomi MW, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M, et al. (2008) Inhibition of 7,12-dimethylbenzanthracene-induced skin tumors by a nutrient mixture. Med Oncol 25: 333-340.

79. Roomi MW, Bhanap BA, Roomi NW, Rath M, Niedzwiecki A (2009) Antineoplastic effects of nutrient mixture on raji and jurkat t cells: the two highly aggressive non-Hodgkin's lymphoma cell lines. Exp Oncol 31: 149-155.

80. Roomi MW, Roomi NW, Kalinovsky T, Rath M, Niedzwiecki A (2009) Marked inhibition of growth and invasive parameters of head and neck squamous carcinoma FaDu by a nutrient mixture. Integr Cancer Ther 8: 168-176.

81. Waheed Roomi M, Ivanov V, Kalinovsky T, Niedzwiecki A, Rath M. (2006) In vivo and in vitro antitumor effect of a unique nutrient mixture on lung cancer cell line A-549. Exp. Lung Res 32: 441-453.

82. Lockhart AC, Braun RD, Yu D, Ross JR, Dewhirst MW, et al. (2003) Reduction of Wound Angiogenesis in Patients Treated with BMS-275291, a Broad Spectrum Matrix Metalloproteinase Inhibitor. Clin Cancer Res 9: 551-554.

83. Lode, H.N.; Huebener, N.; Strandsby, A.; Gaedicke, G. (2008) Nutrient mixture including vitamin C, L-lysine, L-proline, and epigallocatechin is ineffective against tumor growth and metastasis in a syngeneic neuroblastoma model. Pediatr Blood Cancer 50: 284-288.

84. Volek&Phinney, page 91.

85. The American Heritage Medical Dictionary Copyright (2007). Mosby's Medical Dictionary, 8th edition. 2009. Dorland's Medical Dictionary for Health Consumers.

86. Bach A, Schirardin H, Weryha A, Bauer M (1977) Ketogenic response to medium-chain triglyceride load in the rat. J Nutr 107: 1863-1870.

87. Pamela C. Champe, Richard A Harvey (2005) Lippincott's Illustrated Reviews: Biochemistry. Lippincott Williams & Wilkins.

88. Johnston DG, Pernet A, McCulloch A, Blesa-Malpica G, Burrin JM, et al. (1982) Some hormonal influences on glucose and ketone body metabolism in normal human subjects. Ciba Foundation symposium 87: 168-191.

89. Kossoff Eric H, Freeman John M, Turner Zahava, Rubenstein James E (2011) Ketogenic Diets: Treatments for Epilepsy and Other Diseases. Demos Health.

90. Manninen Anssi H (2004) Metabolic Effects of the Very-Low-Carbohydrate Diets: Misunderstood "Villains" of Human Metabolism. J IntSoc Sports Nutr 1: 7-11.

91. Volek JS, Fernandez ML, Feinman RD, Phinney SD (2008) Dietary carbohydrate restriction induces a unique metabolic state positively affecting atherogenic dyslipidemia, fatty acid partitioning, and metabolic syndrome. Prog Lipid Res 47: 307-318.

92. Allen BG, Bhatia SK, Anderson CM, EichenbergerGilmore JM, Sibenaller ZA, et al. (2014) Ketogenic diets as an adjuvant cancer therapy: History and potential mechanism. Redox Biol 2: 963-970.

93. Otto Warburg, Franz Wind, Erwin Negelein (1927) The Metabolism of Tumors in the Body. J Gen Physiol 8: 519-530.

94. Klement RJ (2013) Calorie or carbohydrate restriction? The ketogenic diet as another option for supportive cancer treatment. Oncologist 18: 1056.

95. Rainer J Klement, Ulrike Kämmerer (2011) Is there a role for carbohydrate restriction in the treatment and prevention of cancer? Nutr Metab (Lond) 8: 75.

96. AM Poff, C Ari, P Arnold, TN Seyfried, DP D'Agostino (2014) Ketone supplementation decreases tumor cell viability and prolongs survival of mice with metastatic cancer. Int J Cancer 135: 1711-1720.

97. Weihua Zhou, Purna Mukherjee, Michael A Kiebish, William T Markis, John G Mantis, et al. (2007) The calorically restricted ketogenic diet, an effective alternative therapy for malignant brain cancer. Nutr Metab (Lond) 4: 5.

98. Poff AM, Ari C, Seyfried TN, D'Agostino DP (2013) The ketogenic diet and hyperbaric oxygen therapy prolong survival in mice with systemic metastatic cancer. PLoS One 8: e65522.

99. Mavropoulos JC, Buschemeyer WC 3rd, Tewari AK, Rokhfeld D, Pollak M, et al. (2009) The effects of varying dietary carbohydrate and fat content

on survival in a murine LNCaP prostate cancer xenograft model. Cancer Prev Res (Phila) 2: 557-565.

100. Otto C, Kaemmerer U, Illert B, Muehling B, Pfetzer N, et al. (2008) Growth of human gastric cancer cells in nude mice is delayed by a ketogenic diet supplemented with omega-3 fatty acids and medium-chain triglycerides. BMC Cancer 8:122.

101. Zuccoli G, Marcello N, Pisanello A, Servadei F, Vaccaro S, et al. (2010) Metabolic management of glioblastoma multiforme using standard therapy together with a restricted ketogenic diet: Case Report. Nutr Metab (Lond) 7: 33.

102. Nebeling LC, Miraldi F, Shurin SB, Lerner E (1995) Effects of a ketogenic diet on tumor metabolism and nutritional status in pediatric oncology patients: two case reports. J Am Coll Nutr 14: 202-208.

103. Schmidt M, Pfetzer N, Schwab M, Strauss I, Kämmerer U (2011) Effects of a ketogenic diet on the quality of life in 16 patients with advanced cancer: A pilot trial. Nutr Metab (Lond) 8: 54.

104. Rossi-Fanelli F, Franchi F, Mulieri M, Cangiano C, Cascino A, et al. (1991) Effect of energy substrate manipulation on tumour cell proliferation in parenterally fed cancer patients. Clin Nutr 10: 22832.

105. Kenneth Schwartz, Howard T Chang, Michele Nikolai, Joseph Pernicone, Sherman Rhee, et al. (2015) Treatment of glioma patients with ketogenic diets: report of two cases treated with an IRB-approved energy-restricted ketogenic diet protocol and review of the literature. Cancer Metab 3: 3.

106. Budwig J (1956) Cytostatic or cytodynamic control of cancer? Hippokrates 27: 605-612.

107. Budwig J (1971) Photo-elements of life as an anti-carcinoma factor, successful as a preventive and in the progressive state of the illness. Minerva Ginecol 23: 115-117.

108. Budwig J (2005) Cancer: The Problem and the Solution. Nexus: Kernen, Germany, 2005.

109. Hans Krebs (2010) Nobel Prize Web site. https://www.nobelprize.org/prizes/medicine/1953/krebs/biographical/

110. Otto Warburg, 2009. https://www.nobelprize.org/prizes/medicine/1931/warburg/biographical/ (accessed on 15 September 2009).

111. Otto Warburg (2009) Yellow enzyme. Encyclopedia Britannica. Available online: http://www.britannica.com/EBchecked/topic/635734/Otto-Warburg#ref=ref139725 (accessed on 15 September 2009).

112. Warburg O (1928) The Chemical Constitution of Respiration Ferment. Science 68: 437-443.

113. Warburg O (1956) On respiratory impairment in cancer cells. Science 124: 269-270.

114. Warburg O (1956) On the origin of cancer cells. Science 123: 309-314.

115. Budwig J (1994) Flax Oil as a True Aid against Arthritis, Heart Infarction, Cancer and Other Diseases. Apple Publishing Company: Vancouver, BC, Canada.

116. Kaufmann H, Budwig J, Duddek E (1951) Die Papier-Chromatographie auf dem Fettgebiet VI: Anwendung auf Seifen. Fette und Seifen 53: 285-288.

117. Kaufmann H, Budwig J (1951) Die Papier-Chromatographie auf dem Fettgebiet IX: Anwendung auf Lackrohstoffe. Fette und Seifen 53: 408-412.

118. Kaufmann H, Budwig J, Szakall A (1951) Die Papier-Chromatographie auf dem Fettgebiet VIII: Der Lipoid-Nachschub in der lebenden menschlichen Haut und seine papier-chromatographische Bestimmung. Fette und Seifen 53: 406-408.

119. Kaufmann H, Budwig J (1952) Zur Biologie der Fette V: Die Papier-Chromatographie der Blutlipoide, Geschwulstproblem und Fettforschung. Fette und Seifen 54: 156-165.

120. Kaufmann H, Budwig J, Schmidt C (1952) Die Papier-Chromatographie auf dem Fettgebiet XI: Nachweis und Trennung von Konjuen-Fettsäuren. Fette und Seifen 54: 10-12.

121. Kaufmann H, Budwig J, Schmidt C (1952) Die Papier-Chromatographie auf dem Fettgebiet XII: Nachweis und Trennung von Konjuen-Fet säuren, 2. Teil. Fette und Seifen 54: 73-76.

122. Kaufmann H, Budwig J (1950) Der «Schaumtest» in der Papier-Chromatographie. Fette und Seifen 52: 555-556.

123. Kaufmann H, Budwig J (1951) Die Papier-Chromatographie auf dem Fettgebiet IV: Die Radiometrie der Ölsäure. Fette und Seifen 53: 69-73.

124. Kaufmann H, Budwig J (1951) Die Papier-Chromatographie auf dem Fettgebiet VII: Nachweis und Trennung von Fettsäuren. Fette und Seifen 53: 390-399.

125. Kaufmann H, Budwig J (1951) Dei Papier-Chromatographie auf dem Fettgebiet V: Die radiometrische Jodzahl der Fette. Fette und Seifen 53: 253-259.

126. Kaufmann H, Budwig J (1952) Die Papier-Chromatographie auf dem Fettgebiet XIV: Untersuchung von ›Poly-Ölen‹. Fette und Seifen 54: 348-356.

127. Kaufmann H, Budwig J (1952) Die Papier-Chromatographie auf dem Fettgebiet X: Fluoreszenz-Farben als Indikatoren bei der papyrographischen Analyse von Fettsäuren und Fetten. Fette und Seifen 54: 7-10.

128. Gonzalez CA, Riboli E (2010) Diet and cancer prevention: Contributions from the European Prospective Investigation into Cancer and Nutrition (EPIC) study. Eur J Cancer 46: 2555-2562.

129. Hooper L, Summerbell CD, Higgins JP, Thompson RL, Clements G, et al. (2001) Reduced or modified dietary fat for preventing cardiovascular disease. Cochrane Database Syst Rev.

130. Flaxseed oil (2010) Natural Standard. (accessed on 21 June 2010).

131. Marnett LJ (2000) Oxyradicals and DNA damage. Carcinogenesis 21: 361-370.

132. Valko M, Izakovic M, Mazur M, Rhodes CJ, Telser J (2004) Role of oxygen radicals in DNA damage and cancer incidence. Mol Cell Biochem 266: 37-56.

133. Peter Mitchell (2010) Nobel Prize Web site.

134. Institute of Medicine. Daily Recommended Intakes (2005).

135. Somayeh Zaminpira, Sorush Niknamian (2017) Evolutionary Metabolic Hypothesis of Cancer (EMHC). EC Pulmonology and Respiratory Medicine 5: 167-179

136. Gonzalez CA, Riboli E (2010) Diet and cancer prevention: Contributions from the European Prospective Investigation into Cancer and Nutrition (EPIC) study. Eur J Cancer 46: 2555-2562.

137. Lee JE, Mannisto S, Spiegelman D, Hunter DJ, Bernstein, et al. (2009) Intakes of fruit, vegetables, and carotenoids and renal cell cancer risk: A pooled analysis of 13 prospective studies. Cancer Epidemiol Biomarkers Prev 18: 1730-1739.

138. Lin J, Kamat A, Gu J, Chen M, Dinney CP, et al. (2009) Dietary intake of vegetables and fruits and the modification effects of GSTM1 and NAT2 genotypes on bladder cancer risk. Cancer Epidemiol Biomarkers Prev 18: 2090-2097.

139. Satia JA, Tseng M, Galanko JA, Martin C, Sandler RS (2009) Dietary patterns and colon cancer risk in Whites and African Americans in the North Carolina Colon Cancer Study. Nutr Cancer 61: 179-193.

140. De SE, Boffetta P, Deneo-Pellegrini H, Ronco AL, Aune D, et al. (2009) Meat intake, meat mutagens and risk of lung cancer in Uruguayan men. Cancer Causes Control 20: 1635-1643.

141. Ferrucci LM1, Cross AJ, Graubard BI, Brinton LA, McCarty CA, et al. (2009) Intake of meat, meat mutagens, and iron and the risk of breast cancer in the Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial. Br J Cancer 101: 178-184.

142. Taylor VH, Misra M, Mukherjee SD (2009) Is red meat intake a risk factor for breast cancer among premenopausal women? Breast Cancer Res Treat 117: 1-8.

143. Pala V, Krogh V, Berrino F, Sieri S, Grioni S, et al. (2009) Meat, eggs, dairy products, and risk of breast cancer in the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort. Am J Clin Nutr 90: 602-612.

144. Ferrucci LM, Sinha R, Graubard BI, Mayne ST, Ma X, et al. (2009) Dietary meat intake in relation to colorectal adenoma in asymptomatic women. Am J Gastroenterol 104: 1231-1240.

145. Pauwels EK, Kairemo K (2008) Fatty acid facts, part II: role in the prevention of carcinogenesis, or, more fish on the dish? Drug News Perspect 21: 504-510.

146. Chavarro JE, Stampfer MJ, Hall MN, Sesso HD, Ma J (208) A 22-y prospective study of fish intake in relation to prostate cancer incidence and mortality. Am J Clin Nutr 88: 1297-1303.

147. Melnik BC (2009) Milk-the promoter of chronic Western diseases. Med Hypotheses 72: 631-639.

148. Van der Pols JC, Bain C, Gunnell D, Smith GD, Frobisher C, Martin RM (2007) Childhood dairy intake and adult cancer risk: 65-y follow-up of the Boyd Orr cohort. Am J Clin Nutr 86: 1722-1729.

149. Huncharek M, Muscat J, Kupelnick B (2009) Colorectal cancer risk and dietary intake of calcium, vitamin D, and dairy products: a meta-analysis of 26,335 cases from 60 observational studies. Nutr Cancer 61: 47-69.

150. Lee SA, Shu XO, Yang G, Li H, Gao YT, et al. (2009) Animal origin foods and colorectal cancer risk: A report from the Shanghai Women's Health Study. Nutr Cancer 61: 194-205.

151. Lappe JM1, Travers-Gustafson D, Davies KM, Recker RR, Heaney RP (2008) Vitamin D and calcium supplementation reduces cancer risk: results of a randomized trial. Am J Clin Nutr 85: 1586-1591.

152. Pilz S, Tomaschitz A, Obermayer-Pietsch B, Dobnig H, Pieber TR (2009) Epidemiology of vitamin D insufficiency and cancer es. 29: 3699-3704.

153. Battais F, Richard C, Jacquenet S, Denery-Papini S, Moneret-Vautrin DA (2008) Wheat grain allergies: An update on wheat allergens. Eur. Ann. Allergy Clin. Immunol. 40: 67-76.

154. Freeman HJ (2010) Celiac disease (gluten-sensitive enteropathy). Minerva Gastroenterol Dietol. 56: 245-249.

155. Freeman HJ (2009) Adult celiac disease and its malignant complications. Gut Liver. 3: 237-246.

156. Jiao L, Flood A, Subar AF, Hollenbeck AR, Schatzkin A, et al. (2009) Glycemic index, carbohydrates, glycemic load, and the risk of pancreatic cancer in a prospective cohort study. Cancer Epidemiol. Biomarkers Prev. 18: 1144-1151.

157. Polesel J, Talamini R, Negri E, Bosetti C, Boz G, et al. (2010) Dietary habits and risk of pancreatic cancer: an Italian case-control study. Cancer Causes Control 21: 493-500.

158. Mulholland HG, Cantwell MM, Anderson LA, Johnston BT, Watson RG, et al. (2009) Glycemic index, carbohydrate and fiber intakes and risk of reflux esophagitis, Barrett's esophagus, and esophageal adenocarcinoma. Cancer Causes Control 20: 279-288.

159. Lajous M, Boutron-Ruault MC, Fabre A, Clavel-Chapelon F, Romieu I (2008) Carbohydrate intake, glycemic index, glycemic load, and risk of postmenopausal breast cancer in a prospective study of French women. Am J Clin Nutr 87: 1384-1391.

160. Sieri S, Pala V, Brighenti F, Pellegrini N, Muti P, et al. (2007) Dietary glycemic index, glycemic load, and the risk of breast cancer in an Italian prospective cohort study. Am J Clin Nutr 86: 1160-1166.

161. Wen W, Shu XO, Li H, Yang G, Ji BT, et al. (2009) Dietary carbohydrates, fiber, and breast cancer risk in Chinese women. Am J Clin Nutr 89: 283-289.

162. Polednak AP (2008) Estimating the number of U.S. incident cancers attributable to obesity and the impact on temporal trends in incidence rates for obesity-related cancers. Cancer Detect Prev 32: 190-199.

163. Sears W. Sugar and immunity, 2009.

164. Johansen D, Borgstrom A, Lindkvist B, Manjer J (2009) Different Markers of Alcohol Consumption, Smoking and Body Mass Index in Relation to Risk of Pancreatic Cancer. A Prospective Cohort Study within the Malmo Preventive Project. Pancreatology 9: 677-686.

165. Platek ME, Shields PG, Marian C, McCann SE, Bonner MR, et al. (2009) Alcohol consumption and genetic variation in methylenetetrahydrofolate reductase and 5-methyltetrahydrofolate-homocysteine methyltransferase in relation to breast cancer risk. Cancer Epidemiol Biomarkers Prev 18: 2453-2459.

166. Poynter JN, Haile RW, Siegmund KD, Campbell PT, Figueiredo JC, et al. (2009) Associations between Smoking, Alcohol Consumption, and Colorectal Cancer, Overall and by Tumor Microsatellite Instability Status. Cancer Epidemiol. Biomarkers Prev 18: 2745-2750.

167. Gong Z, Kristal AR, Schenk JM, Tangen CM, et al. (2009) Alcohol consumption, finasteride, and prostate cancer risk: results from the Prostate Cancer Prevention Trial. Cancer 115: 3661-3669.

168. Parsons JK, Im R (2009) Alcohol consumption is associated with a decreased risk of benign prostatic hyperplasia. J Urol 182: 1463-1468.

169. Benedetti A, Parent ME, Siemiatycki J (2009) Lifetime consumption of alcoholic beverages and risk of 13 types of cancer in men: Results from a case-control study in Montreal. Cancer Detect Prev 32: 352-362.

170. Flom JD, Ferris JS, Tehranifar P, Terry MB (2009) Alcohol intake over the life course and mammographic density. Breast Cancer Res Treat 117: 643-651.

171. Guerrero RF, Garcia-Parrilla MC, Puertas B, Cantos-Villar E. (2009) Wine, resveratrol and health: A review. Nat Prod Commun 4: 635-658.

172. Motevaseli E, Dianatpour A, Ghafouri-Fard S (2017) The Role of Probiotics in Cancer Treatment: Emphasis on their In Vivo and In Vitro Anti-metastatic Effects. Int J Mol Cell Med 6: 66-76.

173. Yu AQ, Li L (2016) The Potential Role of Probiotics in Cancer Prevention and Treatment. Nutr Cancer. 68: 535-544.

174. Reid G, Jass J, Sebulsky MT, McCormick JK (2003) Potential uses of probiotics in clinical practice. Clin Microbiol Rev 16: 658-672.

175. Ouwehand AC, Salminen S, Isolauri E (2002) Probiotics: an overview of beneficial effects (PDF). Antonie Van Leeuwenhoek 82: 279-289.

176. Hatakka K, Savilahti E, Pönkä A, Meurman JH, Poussa T, et al. (2001) Effect of long-term consumption of probiotic milk on infections in children attending day care centres: double blind, randomised trial. BMJ 322: 1327.

177. Näse L, Hatakka K, Savilahti E, Saxelin M, Pönkä A, et al. (2001) Effect of long-term consumption of a probiotic bacterium, Lactobacillus rhamnosus GG, in milk on dental caries and caries risk in children. Caries Research 35: 412-420.

178. Kirjavainen PV, Salminen SJ, Isolauri E (2003) Probiotic bacteria in the management of atopic disease: underscoring the importance of viability. J Pediatr Gastroenterol Nutr 36: 223-227.

179. Braat H, van den Brande J, van Tol E, Hommes D, Peppelenbosch M, et al. (2004) Lactobacillus rhamnosus induces peripheral hyporesponsiveness in stimulated CD4+ T cells via modulation of dendritic cell function. The American Journal of Clinical Nutrition 80: 161825.

180. Maria Jose Saez-Lara, Carolina Gomez-Llorente, Julio Plaza-Diaz, Angel Gil (2015) The Role of Probiotic Lactic Acid Bacteria and Bifidobacteria in the Prevention and Treatment of Inflammatory Bowel Disease and Other Related Diseases: A Systematic Review of Randomized Human Clinical Trials. Biomed Res Int (Systematic review) 2015: 15.

181. Probiotics and prebiotics. World Gastroenterology Organisation Global Guidelines". World Gastroenterology Organisation. October 2011.

182. Ghouri YA, Richards DM, Rahimi EF, Krill JT, Jelinek KA, et al. (2014). Systematic review of randomized controlled trials of probiotics, prebiotics, and synbiotics in inflammatory bowel disease. Clin Exp Gastroenterol (Review). 7: 473-487.

183. Moayyedi P, Ford AC, Talley NJ, Cremonini F, Foxx-Orenstein AE, et al. (2010) The efficacy of probiotics in the treatment of irritable bowel syndrome: a systematic review. Gut (Systematic review) 59: 325-332.

184. AlFaleh K, Anabrees J (2014) Probiotics for prevention of necrotizing enterocolitis in preterm infants. Cochrane Database of Systematic Reviews 4.

185. Newlove-Delgado TV, Martin AE, Abbott RA, Bethel A, Thompson-Coon J, et al. (2017) Dietary interventions for recurrent abdominal pain in childhood. Cochrane Database Syst Rev 3: CD010972.

186. Shortliffe LMD (2013). Wein AJ, ed. Chapter 116: Infection and Inflammation of the Pediatric Genitourinary Tract. Urology. 4 (10th ed.). Saunders Elsevier 3121.

187. Cooke G, Behan J, Costello M (2006) Newly identified vitamin K-producing bacteria isolated from the neonatal faecal flora. Microbial Ecology in Health and Disease 18: 133-138.

188. Strozzi GP, Mogna L (2008) Quantification of Folic Acid in Human Feces After Administration of Bifidobacterium Probiotic Strains. Journal of Clinical Gastroenterology 42: S179-S184.

189. Molina VC, Médici M, Taranto MP, Font de Valdez G (2009) Lactobacillus reuteriCRL 1098 prevents side effects produced by a nutritional vitamin B12deficiency. Journal of Applied Microbiology 106: 467-473.

190. Angela M Poff1, Csilla Ari, Thomas N Seyfried, Dominic P. D'Agostino1 (2003) The Ketogenic Diet and Hyperbaric Oxygen Therapy Prolong Survival in Mice with Systemic Metastatic Cancer. PLOS ONE 8: e65522.

191. Gill AL, Bell CNA (2004) Hyperbaric oxygen: its uses, mechanisms of action and outcomes. QJM 97.

192. Stuhr L, Raa A, Oyan A, Kalland K, Sakariassen P, et al. (2007) Hyperoxia retards growth and induces apoptosis, changes in vascular density and gene expression in transplanted gliomas in nude rats. Journal of neuro-oncology 85: 191-393.

193. Moen I, Øyan A, Kalland K-H, Tronstad K, Akslen L, et al. (2009) Hyperoxic treatment induces mesenchymal-to-epithelial transition in a rat adenocarcinoma model. PloS one 4.

194. Stuhr L, Iversen V, Straume O, Maehle B, Reed R (2004) Hyperbaric oxygen alone or combined with 5-FU attenuates growth of DMBA-induced rat mammary tumors. Cancer letters 210: 35-75.

195. Bennett M, Feldmeier J, Smee R, Milross C (2008) Hyperbaric oxygenation for tumour sensitisation to radiotherapy: a systematic review of randomised controlled trials. Cancer treatment reviews 34: 577-591.

196. Takiguchi N, Saito N, Nunomura M, Kouda K, Oda K, et al. (2001) Use of 5FU plus hyperbaric oxygen for treating malignant tumors: evaluation of antitumor effect and measurement of 5-FU in individual organs. Cancer chemotherapy and pharmacology 47: 11-14.

197. Petre P, Baciewicz F, Tigan S, Spears J (2003) Hyperbaric oxygen as a chemotherapy adjuvant in the treatment of metastatic lung tumors in a rat model. The Journal of thoracic and cardiovascular surgery 125: 85-91.