

**Defeating cancer takes more than one treatment method:  
A retrospective case series using multiple nutritional and herbal agents**

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**It is not enough to correct genetic damage and neglect to reverse all other cancer-causing problems. It is not enough to stop angiogenesis and drop the ball on all other cancer-causing problems. It is not enough to attack metastases and leave the primary tumor in a comfortable environment. In order to defeat cancer, it must be attacked at every level and with every method necessary to reverse cancer's multi-pronged assault on the body, even if that means that some of the various treatments have redundant effects. And this all must be accomplished while maintaining the maximum possible wellbeing of the patient, and without sickening or weakening the patient. – Colleen Huber, NMD**

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## **Abstract**

**INTRODUCTION:** Research has shown that for cancer to occur in the body multiple normal functions must break down. Therefore multiple-agent treatments may be the only successful way to treat cancer. We used well-tolerated natural substances to assess their usefulness in combination anti-neoplastic therapy.

**METHODS:** We treated a total of 49 patients for cancer from October 2006, when we opened our practice, until July 2009, when we stopped collecting data and wrote this paper. Data from all 49 patients who came to us with a diagnosis of cancer are included in this paper, excluding only those cancer patients who decided against further treatment after less than two weeks in our care. We treated with natural methods alone, choosing among methods with research-established anti-neoplastic effect, both dietary and supplemented, nutritional and herbal, oral and intravenous, having a preference for those with high patient tolerance and compatibility, and varying with individual needs and tolerance, according to the standard naturopathic principle of “Treat the whole person.”

**FINDINGS:** 27 patients voluntarily left our practice, against our advice, primarily for financial reasons, while still having cancer. Of the remaining 22 patients, 12 went into complete remission, which we define by no evidence of cancer remaining in the body on imaging. Those patients in remission stayed in our care an average of 4.4 months; those who left, 2.0 months. We are still treating 8 patients, and two died while still our patients. Of the 12 who went into remission, none had chosen to have chemotherapy while having our treatments. None of those 12 has come out of remission as of this writing.

**INTERPRETATION:** Our results are an improvement over other known cancer treatment methods, chemotherapeutic (with a 2.5% success rate), radiological or natural. However, the two patients who did not survive treatment as well as four other patients who had an increased tumor load during our treatments make a 12% failure rate. Therefore these treatment strategies are still not adequate to eliminate all patients’ cancers and must be further developed.

## **Introduction**

Cancer treatment, as almost all other areas of medicine, has been constrained by the prevailing view that a single agent must be isolated and tested for its either successful or failing role as the therapeutic agent to eliminate illness. With cancer especially, this viewpoint is especially unlikely to be true, for the following reasons. Many agents are needed to fight cancer primarily because it arises after several normal mechanisms break down, and because cancer preys on the body in numerous ways simultaneously, and because no single agent, whether chemotherapeutic or natural, has yet been found that has enough anti-neoplastic strategic effects to reverse all of those abnormalities in all patients, in effect, to be “the cure” for cancer.

## **Background**

As John Boik has described, cancer becomes possible, has its only opportunity to arise in the body, when seven different events, such as genetic damage, angiogenesis, immune system

evasion, etc. all go wrong,<sup>1</sup> as listed below. Then, once established, cancer is adaptable enough to be able to thrive and grow with the continuation of even one or a few of those deviant events.

Boik describes the seven pro-cancer events as follows:

- 1) genetic instability or vulnerability to mutation, necessarily the first of the variety of events that lead to a tumor;
- 2) abnormal gene expression, in this case that produce proteins that facilitate cancer, or at least do not prevent it;
- 3) abnormal and autonomous cell signal transduction, which allows cancer cells to grow through self-stimulation rather than depending on growth factors from other cells;
- 4) Abnormal cell-to-cell communication, which sets a tumor apart from its neighboring cells metabolically, leaving the tumor in a position to ignore homeostatic mechanisms and, unlike cells throughout the rest of the body, to act in the best interests of the tumor rather than in the best interests of the organism.
- 5) Angiogenesis, the creation of blood vessels and resultant hoarding by the tumor of disproportionately large amounts of nutrients;
- 6) Invasion and metastasis, which not only results from the aggressive nature of the tumor, but also the low integrity and too friable nature of the surrounding normal tissue and basement membranes;
- 7) Evasion of the immune system, which involves both camouflage functions and immune-disabling functions of cancer cells.

The “Dinomit” acronym as a model of cancer development recently proposed by Cedric Garland is a similar recognition of the numerous events necessary to enable the development of cancer.<sup>2</sup> “Dinomit” refers to D: Disjunction, I: Initiation, N: Natural Selection, O: Overgrowth, M: Metastasis, I: Involution, T: Cancer becomes chronic.

Once established in the body, cancer seems to have the ability to thrive and reproduce despite most of the heroic efforts against it by oncologists, and without necessarily requiring all seven of the above pro-cancer events to still be in place. Therefore, without certain knowledge of the precise mechanisms governing any one patient’s cancer, any therapy that targets fewer than those seven major disturbances leaves the body of the cancer patient potentially vulnerable to the disastrous result of allowing continued growth of existing tumors. Shortchanging the patient of a diverse range of available, effective, well-tolerated, well-targeted, compatible, complementary and feasible treatment options also would allow too many of the conditions to persist that gave rise to tumors previously and may do so again, leaving the fertile ground that produced the cancer in the first place. For this reason, successful cancer therapy should be multi-purposed and with multiple agents, many more than are now used with each patient by oncologists.

We have used natural therapies for cancer treatment, because they are well adapted for multi-agent use. Unrefined plant materials have up to thousands of phytochemical components, originally useful for protecting a plant from extreme or adverse conditions in its environment, and ultimately employed as described below by naturopathic physicians in adaptation to the needs of the human patient. Licensed naturopathic physicians, because of thorough medical training and extensive training in the use of natural agents are well suited to choose appropriate combinations of natural therapies for the individual cancer patient. We also take advantage of

the greater compatibility among natural substances than among numerous pharmaceuticals. Just as a meal may contain many different foods without the need for conscious consideration of potential interactions, we have combined many different nutrients and plant materials in each cancer patient's treatment protocol.

## **Materials and Methods**

Dietary interventions are of the utmost importance in cancer therapy, especially keeping blood sugar low. The significant majority of research on the subject establishes a correlation between blood glucose and tumor growth. Using PET imaging preferentially for tumor evaluation, clinicians make use of the fact that tumors take up blood glucose considerably more than does benign tissue, which implies an especially glucose-dependent metabolism in cancer cells.

Research has shown a correlation between blood sugar or glycemic load and cancer growth for pancreatic cancer,<sup>3</sup> breast cancer,<sup>4 5</sup> gastric cancer,<sup>6 7</sup> colon cancer,<sup>8 9</sup> ovarian cancer<sup>10</sup> and prostate cancer.<sup>11</sup> Given all of this evidence, it would be reckless to allow a cancer patient to assume that sugar intake is harmless. We therefore ask all of our cancer patients to avoid sweeteners, such as sugar, honey, maple syrup, corn syrup, as well as fruit juices because such foods tend to have the highest glycemic indices. Use of stevia is encouraged when one wants to sweeten foods. For the same reason, we asked patients to also minimize other refined carbohydrates, specifically flour products. Whole natural foods: vegetables, fruits, whole grains, dairy and meat are encouraged as the entire diet, with the widest available variety in those groups. Use of soy is discouraged because of its phytoestrogenic component, which in some studies has been linked to a possible association with cancer.

Of equal importance with diet are the IV nutrients that we administer three times per week to each cancer patient. These consist of high-dose intravenous vitamin C (ascorbic acid), as well as other nutrients chosen for specific anti-neoplastic effect with regard to the patient's type of cancer. For solid malignant tumors, we address the problem of pH, by infusing both sodium bicarbonate to alkalinize systemically, as well as other specifically anti-cancer nutrients, tailored to the individual patient's tumor load, type of cancer and other health circumstances. B vitamins and minerals are added for synergistic effect with Vitamin C. Naturopathic training emphasizes the treatment of the individual with regard to the entire symptom picture. Therefore, there is no specific formula to be repeated in cookbook fashion from one patient to the next, or even for the same patient from one day to the next. Quantities of the different components of this combination vary for every individual depending on symptoms, signs and type of cancer. Quantities also vary as the patient's needs change. All components are kept far below the LD50 for each component, and are only administered if they have not produced any side effects in our patients.

Research has established that ascorbic acid taken orally cannot attain sufficiently high concentrations in the bloodstream to kill cancer cells.<sup>12 13</sup> However, intravenous use of ascorbic acid has been shown to rise to concentrations that have killed cancer cells in vivo<sup>14 15 16</sup> and in vitro.<sup>17 18 19</sup> The ascorbic acid that we use is in much higher dose than would be tolerated orally, yet at a level where there is sufficient concentration of vitamin C in the bloodstream to create a substantial concentration of the products of vitamin C in the extracellular fluid.<sup>20</sup> Intravenous doses of ascorbic acid have been found to produce from 25 to 70 times as much plasma

concentration as may be attained by oral dosing.<sup>21</sup> Research has confirmed that Vitamin C in such high concentration kills cancer cells while leaving normal tissue unharmed.<sup>22 23</sup> Indeed the cancer patients whom we treat do not have side effects from these treatments, with few exceptions. The two exceptions were allergies to specific B vitamins in two individuals. Both went into remission after we had removed the offending agent early on (Patients #11 and 28).

In addition to this directly and selectively cytotoxic effect on cancer cells, vitamin C has been shown to form collagen<sup>24</sup> and to inhibit hyaluronidase<sup>25</sup> leading to stronger membrane integrity and tensile strength<sup>26</sup> of normal tissue, which inhibits invasion<sup>27</sup> and thus metastases.

Empirical data shows an inverse correlation between vitamin D intake and cancer incidence.<sup>28 29</sup><sup>30</sup> A rapidly growing body of research has confirmed the essential role that Vitamin D plays in cancer prevention and treatment.<sup>31 32 33 34</sup> Vitamin D has been shown to induce differentiation,<sup>35</sup> and apoptosis,<sup>36</sup> to reduce proliferation by effect on signal transduction,<sup>37</sup> to improve intercellular communication by means of gap junction communication preservation,<sup>38</sup> to inhibit angiogenesis,<sup>39 40</sup> and to inhibit metastasis.<sup>41</sup> At our clinic, most cancer patients are prescribed a regular dose of Vitamin D that is compatible with the weight, customary sunlight exposure, current pharmaceuticals if any, as well as the condition of the liver and gallbladder and calcium metabolizing mechanisms.

Vitamin A is a less-widely appreciated but quite crucial part of the treatment protocol for its immune-stimulating<sup>42</sup> and target identifying effects. Another very important quality of Vitamin A with regard to neoplastic cells is its ability to introduce differentiation.<sup>43 44</sup> It has also been shown to induce apoptosis in cancer cells,<sup>45</sup> as well as growth inhibition.<sup>46</sup> Although there have been recent objections made to Vitamin A for an allegedly competitive and detrimental effect to vitamin D,<sup>47</sup> and we have noted lower vitamin D levels after months of concurrent dosing, vitamin A does have the above-mentioned significant anti-cancer properties that make it useful in our work. Older research supports the use of vitamin A and vitamin D dosed together.<sup>48 49 50</sup>

We almost always add the recommendation to take Essiac tea (Resperin Canada Limited, Waterloo, Ontario, Canada), because of its long history in North America, over most of the last century of folk use (outside of conventional medicine) against a wide variety of cancers. Essiac was developed by a Canadian nurse, René Caisse, together with the Ojibwe people of Canada. It is a combination of four herbs, *Arctium lappa*, *Rheum palmatum*, *Rumex acetosella*, and *Ulmus fulva*. Later versions of Essiac, using additional herbs with some pro-estrogenic effect has been linked to breast tissue proliferation,<sup>51</sup> and we do not recommend those altered formulas. Essiac has been found to have in vitro cytotoxic effects specifically against neoplastic cells preferentially, without damage to normal cells.<sup>52</sup> Its main effect seems to be protective against DNA damage.<sup>53</sup> It also seems to have anti-proliferative effect.<sup>54</sup>

For most of our patients, we have also used digestive enzymes apart from meals, for a presumed proteolytic effect against tumors. This use is still speculative and does not appear to be well-supported at this time in the medical literature. However, various digestive enzymes, and bromelain in particular, have been found to heighten immune system response to cancer<sup>55 56</sup> and to inhibit metastasis.<sup>57 58</sup>

For different cancers there are additional appropriate treatments. For example, Kenneth Proefrock NMD has done extensive original work with nebulizers, as well as many other areas of medicine, which he taught us to use with lung cancer patients to good effect.<sup>59</sup> Whereas all of the rest of our treatments arrive to the lungs by way of the bloodstream, Dr. Proefrock has introduced such nebulized botanicals and nutrients as required by the individual patient by way of the airways, thus carrying anti-neoplastic treatments to lung tissue via its other major port of entry.

## Findings

Of the 49 cancer patients whom we have treated long-term, all came to us with a diagnosis of cancer from another physician. All had been diagnosed with cancer by other physicians and none by us. Of those 49, 2 have died of cancer while still our patients under our care, 12 have gone into remission, substantiated by PET/CT or other imaging, and/or biopsy, and all of those have remained in remission; 27 decided to leave our care against our advice while still with cancer, and 8 are still being treated by us. Specific results are shown in Table 1.

**Table 1: Outcomes of naturopathic management of 49 cancer cases**

Patient #s as- assigned for reporting purpose	Stage at start of tx	Type of cancer	Length of treatment in months  Still being treated (S)	Conventional therapies also used during our txs: Chemotx (C) Radiation(R) Surgery (Su)			Final result: Proven total remission (R), Proven reduced tumor load but not remission (Red), Proven increased tumor load (Inc), New metastases (Met) Death (D), Left practice (L) No further information (NFI) Still txing (S)	Quality of Life at end of treatment Improved (Imp) Worsened (Wor) High-functioning (HF) High- functioning with Exercise (HfWE) Same from beginning to end of treatment (Sa) Patient is employed (Job)
				C	R	Su		
1	1	pros	4 (S)	No	No	No	S. PSA from >12 to < 5	Imp/Job
2	3	breast	1	No	No	No	Red, L, NFI	HF/Job
3	2	liver	3 (S)	No	No	No	Red, S	HFwE/Sa
4	1	mesoth	5	Yes	No	No	Inc., L, then 1 mo, then D	Wor
5	3	lung	1	No	No	No	L, then 1 mo, then, D	Sa
6	3	colon	<1	No	No	Yes	L, NFI	Sa
7	2	breast	5	No	No	Yes	Red prior to surg, R	HFwE/S/Job
8	2	lung	3	No	No	No	R	Imp/Job
9	1	breast	4	No	No	Yes	R	HFwE/Sa/Job
10	4	colon	1	No	No	No	L, NFI	HF/Sa

11	4	Lynch col-ov-uterus	2	No, PC	No	No, PS	R	Imp wE
12	1	CLL	2	No	No	No	L with no lymphadenopathy, borderline leukocytosis, NFI	HFwE/Sa/Job 70yo, bikes miles
13	1	lung	4	No	Yes	No	Red, L	Wor from pulm fibrosis not ca
14	3	cerv	<1	No	No	No	L, NFI	HFwE/Sa/Job
15	2	lung	2	No	No	No	Red, Met, then L, then 6 mos, then D	HF/Sa
16	4	breast	2	No	No	Yes	R	HF/Sa
17	2	breast	1 (S)	No	No	Yes	S	HF/Sa/Job
18	1	breast	6	No	No	Yes	R	HFwE/Sa/Job
19	4	panc	5	No	No	No	L, NFI	Sa
20	4, in hospice	pros	<1	No	No	No	Imp, from hospice to outpt, then L, then 1 mo. Then D	Imp. Then Wor
21	3	breast	5 (S)	No, PC	Yes	Yes	Inc, Met, S	Imp/HFwE 68yo walks 2 mi-22 min
22	2	breast	4	Yes	No	Yes	Inc, Met, L	Wor
23	4	pros	<1 (S)	No	No	No	S, PSA from >100 to <6	Imp
24	4	breast	<1	No	No	No, PS	L, NFI	Sa
25	4; 36 bone mets.	lung	4	Yes	Yes	No	D	Wor
26	4	liver and colon	<1 (S)	No	No, PR	No, PS	S	Imp
27	2	breast	5	No	No	Yes	R	Imp HFwE
28	1	breast	2	No	No	No	Inc., L	HF/Sa/Job
29	1	brain	<1	No	No	No	L, NFI	HF/Sa/Job
30	3	breast	6	No	No	No, PS	R	Imp, Hfwe/Sa
31	3	colon	5	Yes	No	Yes	Red by 80%, L then 2 mos D from surg complications	Imp
32	1	pros	2	No	No	No	L, NFI	HFwE/Sa/Job
33	2	mult my	<1	No	No	No	L, NFI	Same
34	4, was sent to hospice by MD	gastric	2	No PC	No	No	L, then 1 mo, then D	Sa
35	4	panc	4	No, PC	No	No	Red, then Inc, D	Imp then Wor
36	3	breast	<1	No	No	No	L to continue tx in home state, NFI	HFwE/Sa
37	1	breast/Paget's	<1 (S)	No	No	Yes	S	HF/Sa
38	2	pros	4	No	No	No	R	HFwE/Sa/Job
39	4	melanoma	6	No	Yes	Yes	R	Imp/HFwE/Job

40	3	giant cell endometrial	7	No, PC	No, PR	Debulking but not resection PS	R	Imp/HFwE/Job
41	4	gastric	1 (S)	No	No	No	S	Imp/ HF
42	3	NHL	3	No, PC	No	No	R	HF/Sa/Job
43	4	lung	<1	No, PC	No	No	L, NFI	Sa
44	3	lung	1	No	No	No	Red, L, NFI	Sa
45	4	aden./palate	1	No	No, PR	No	L, NFI	Same
46	1	pros	2	No	No	No	L, NFI	HF/Sa
47	4	cerv	3	Yes	No, PR	No	L, then 1 month, D of chemotherapy side effects	Sa
48	4	colon	<1	No, PC	No, PR	No, PS	L, then 1 week, D of hepatic coma	Sa
49	2	liver	<1	Yes	No, PR	No	L, NFI	Sa

The results in Table 1 are summarized as follows:

**Table 2 Summarized outcomes of naturopathic management of 49 cancer cases**

<b>Outcome</b>	<b>Number of patients</b>	<b>Avg. no. of months this group of pts stayed for treatments</b>	<b>Number in each group also receiving chemotherapy</b>	<b>Number in each group also receiving surgery</b>
Remission	12	4.4	0	7
Died while still in care	2	4.0	1	0
Left without finishing treatment	27	2.0	5	3
Still being treated	8	2.5	0	3
<b>Total</b>	<b>49</b>		<b>6</b>	<b>13</b>

Of the 14 patients who continued with treatment until remission or death, 12 went into remission, and 2 died. This is an 85% success rate for those completing our treatments. This does not include any of the currently treated patients, because their final data is not yet available. We have even gotten 3 Stage IV cancer patients into remission. Of the patients who went into remission, they have been contacted by us when collecting the data for this paper, in July 2009, and all whom we could reach are still in remission. We could not reach one patient whose phone numbers were disconnected.

The two patients who died were both Stage IV at start of treatment. One had over 36 bone metastases, over 50 total metastases (Patient #25) and chose to have chemotherapy during our treatments. The other chose not to follow our main dietary recommendation during the last month of his treatment, i.e. not to eat sweetened foods (Patient #35). The latter patient's tumors had reduced considerably during our treatments. Of the 2 pancreatic tumors, one disappeared completely, and the other shrank to approximately half the volume. During this time, the patient stayed very physically active, doing construction work in his own house at age 67. Several weeks went by, then new pain arose. The patient then confessed to starting to eat cookies every night after dinner for the last month, which was contrary to our main dietary treatment focus. Within 2 weeks he was dead of pancreatic cancer with new, extensive metastases.

The patients came in to our care starting at the following stages.

**Table 3: Patients now in remission from our treatments and stage at diagnosis**

<b>Stage</b>	<b>Number of patients</b>	<b>Number in each group also receiving chemotherapy concurrently</b>	<b>Number in each group who received previous chemotherapy</b>
I (breast ca)	2	0	0
II (2 br, 1 lu, 1 pros)	4	0	0
III (br, NHL, endometrial)	3	0	2
IV (br, Lynch, melanoma)	3	0	1
<b>Total</b>	<b>12</b>	<b>0</b>	<b>3</b>

Not one of the patients we treated who also had concurrent chemotherapy went into remission. The only patients we had who went into remission had refused current chemotherapy prior to starting our treatments, although some had chosen to have it in the past. However, of the patients who had chemotherapy along with our treatments, all commented on feeling stronger and better able to tolerate their chemotherapy with our treatments. One patient whose tumor volume had reduced by 80% attributed this good result to both our treatments as well as chemotherapy, an evaluation that seems to defy proof or disproof (Patient #30).

7 of our 12 patients to go into remission also had either surgical resection or debulking of their tumors while getting our treatments. This would suggest that surgery was probably a good choice and that the combination of surgical tumor resection and natural treatments was a good strategy for a successful outcome, although not always required for a good outcome.

**Table 4: Results for patients completing our program with all dietary recommendations and choosing not to have chemotherapy**

<b>Outcome</b>	<b>Number of patients</b>
Remission	12
Came out of remission after stopping our treatments	0
Died while still in care	0
<b>Total</b>	<b>12</b>

In fact, one of our patients in remission is the only known survivor of Stage 3 giant cell endometrial carcinoma (Patient #39), at least according to published medical literature.<sup>60</sup> This remission occurred with only natural treatments after all three conventional cancer treatments, chemotherapy, radiation and surgery, were each tried multiple times and failed for this patient.

**Table 5: Summary of interim changes in tumor load with naturopathic care of 49 cancer patients**

<b>Interim changes</b>	<b>Number of patients</b>	<b>Number in each group also receiving chemotherapy</b>
Reduced tumor load, but no remission yet	7	1
Increased tumor load, but no final outcome during treatment	4	2

We have data for change in tumor size for very few patients. By the time a person seeks the help of a naturopathic physician for any ailment, they have often rejected for one reason or another the conventional medical system, leading to a distrust and disdain for conventional imaging. Imaging such as PET/CT fusion is a “hard sell” to such people. (“You want me to have radioactive glucose after telling me not to eat sugar?”) Biopsy was an even harder sell. Many of those patients left our practice for one reason or another, as discussed below, before we had any information about changing tumor size. Because we have so little information on which patients actually had increased or decreased tumor load, we have not yet had the advantage of the best way to determine the success or failure of our treatments. A strong will must be present in a person to ignore the exhortations of oncologists and worried loved ones, and to pursue treatment by a naturopathic physician. This strong will easily enables rebellion against naturopathic physicians and our recommendations as well.

If one considers that the treatment failed for the 2 patients who died, plus the 4 who had an increased tumor load, then of the 49 total patients, the success rate may be seen as 87%, if success means just keeping people alive at the same or better level of wellbeing. Or rather the failure rate is then 13%.

It cannot be assumed that those for whom our treatments failed to reduce cancer are entirely worse off. Most have described a better quality of life since starting the treatments. For example, one of the patients with stage IV breast cancer, and an increased tumor load since starting our treatments, describes herself as more fit than ever since beginning our treatments, at

68 years old, walking 2 miles up and down hills in 22 minutes, gradually improving her time right up to days before this writing (Patient #21). Although we have not yet found the necessary combination of therapies to reduce and eliminate her particular cancer, she is in a strong position to undertake measures to defeat it, now that she will also be employing conventional means.

For the 27 patients who decided to leave before finishing our treatments, it is difficult to assess the degree of success or failure. Reasons for leaving were often not given. There was sometimes a phone message requesting to cancel the future appointments without explanation. However, when we were told reasons for leaving, the following were common:

- 1) Financial reasons: no insurance reimbursement made it hard to continue paying for our treatments out of pocket. This was by far the most common reason given.
- 2) Patient did not feel that anything important was happening with the treatment. There was a strange viewpoint expressed by some patients that cancer is not very frightening, once they saw that they, as well as all of the other non-chemotherapy cancer patients in our IV room maintained their vitality, their hair and their bodily functions. This led some to the dangerously wrong conclusion that cancer was easy to conquer and that our treatments had not accomplished much, and perhaps had not even contributed to their continued wellbeing.
- 3) A related viewpoint was that improvement in the patient's condition should have been faster and more dramatic. If the condition seemingly stayed the same, some patients viewed this as evidence of failure, of not defeating cancer fast enough, and concluded that the treatment was not working, and that they should not waste any more time or money pursuing it, and that it was time to leave and explore other avenues. Other patients, whose tumor size clearly increased, usually left when they perceived our treatments as not working for them.
- 4) Family members or oncologists disapproved of our treatment and persuasively urged chemotherapy exclusively.
- 5) Patient had traveled from another state to receive our treatments, but wanted to return home to be with family, regardless of expected outcome.

**Table 6: Summary of quality of life changes by subjective evaluation of naturopathic physician along with patient self-evaluation during naturopathic care of 49 cancer patients**

<b>Quality of life changes</b>	<b>Number of patients</b>	<b>Number in each group who went into remission</b>	<b>Number in each group also receiving chemotherapy</b>
Came in with high wellbeing / Still the same way	20	8	0
Came in occupationally functional but not physically fit /Ultimately improved vitality	12	4	1
Came in occupationally	11	0	2

functional but not physically fit / Still the same way			
Ultimately worsened vitality	6	0	3
Came in occupationally functional / now dysfunctional	0	0	0
<b>Total number of patients</b>	<b>49</b>	<b>12</b>	<b>6</b>

If one considers quality of life as a criterion for success, then of the patients who stayed well or got better during our treatments, 43 patients out of 49, make a success rate of 87%. If one considers only improvement in wellbeing as a valid marker of success, then that percentage is 24% (12 out of 49 patients.)

It is important to note that only a minority of the patients did all that was recommended by us. For example, although we recommend beginning our treatments immediately after diagnosis, almost all patients delayed naturopathic treatment for months to years after initial diagnosis of cancer, mostly due to lack of information to the public about the effectiveness of natural treatments for cancer. In addition, some chose to disregard the dietary recommendations that we made or to only observe the recommendations partially. Others chose to have fewer in-office treatments than were recommended. Others decided to choose only some of the recommended treatments due to financial constraints or inconvenience.

None of our cancer patients has come out of remission, and all of those in remission are living productive lives, as confirmed by recent contact with them. Even the patient with the disconnected phone called five months ago, which was several months after she went into remission, in order to inform us that she was now physically active and had at last stopped smoking.

## Discussion

Numerous natural agents were simultaneously employed to reduce and eliminate human neoplasms in vivo. We chose to use these together because cancer is a multifactorial disease and has not yet been fought effectively in a majority of patients with a single agent. Specific combinations of natural substances were chosen with regard to the type of cancer and circumstances of each individual cancer patient. Licensed naturopathic physicians are well-qualified to design such treatment programs because of our broad and extensive training with natural and conventional substances and how to combine them. Successful outcomes seemed to be more likely with steadfast patient compliance during the entire duration of the treatment process. Although our results are a strong improvement over all other cancer treatment protocols, both conventional and natural, if measured by either patient remission or survival, these treatment strategies are still not adequate to eliminate all patients' cancers and must be further developed.

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- <sup>1</sup> Boik, John. *Natural Compounds in Cancer Therapy*. Oregon Medical Press. 2001; p.2.
- <sup>2</sup> Garland C, Gorham E, Mohr F. Vitamin D for cancer prevention: global perspective. *Ann Epidemiol* 2009 Jul;19(7):468-83.
- <sup>3</sup> Chan J, Wang F, Holly E. Sweets, sweetened beverages, and risk of pancreatic cancer in a large population based case-control study. *Cancer Causes & Control*. 2009 Aug; 20(6): 835-46. Epub 2009 Mar 11.
- <sup>4</sup> Larsson, S, Bergkvist L, Wolk A. Glycemic load, glycemic index and breast cancer risk in a prospective cohort of Swedish women. *Int J Cancer*. 2009 Jul 1; 125(1): 153-7.
- <sup>5</sup> Wu A, Yu M, Tseng C. et al. Dietary patterns and breast cancer risk in Asian American women. *Am J Clin Nutr*. 2009 Apr; 89(4): 1145-54.
- <sup>6</sup> Ikeda F, Doi Y, Yonemoto K, et al. Hyperglycemia increases risk of gastric cancer posed by *Helicobacter pylori* infection: a population-based cohort study. *Gastroenterology*. 2009 Apr; 126(4): 1234-41.
- <sup>7</sup> Bertuccio P, Praud D, Chatenoud L, et al. Dietary glycemic load and gastric cancer risk in Italy. *Br J Cancer*. 2009 Feb 10; 100(3): 558-61.
- <sup>8</sup> Wang B, Bobe G, La Pres J, Bourquin L. High sucrose diets promote intestinal epithelial cell proliferation and tumorigenesis in APC mice by increasing insulin and IGF-1 levels. *Nutr Cancer*. 2009; 61(1): 81-93.
- <sup>9</sup> Wang B, Bobe G, La Pres, Bourquin L. Dietary carbohydrate source alters gene expression profile of intestinal epithelium in mice. *Nutr Cancer*. 2009; 61(1): 146-55.
- <sup>10</sup> Augustin L, Polesel J, Bosetti C, et al. Dietary glycemic index, glycemic load and ovarian cancer risk: a case-control study in Italy. *Ann Oncol*. 2003 Jan; 14(1): 78-84.
- <sup>11</sup> Freedland S, Aronson, W. Dietary intervention strategies to modulate prostate cancer risk and prognosis. *Curr Opin Urol*. 2009 May; 19(3): 263-7.
- <sup>12</sup> Creagan E, Moertel C, O'Fallon J, et al. Failure of high-dose Vitamin C (ascorbic acid) therapy to benefit patients with advanced cancer. A controlled trial. *New Engl J Med* 1979 Sep 27. 301(13): 687-90.
- <sup>13</sup> Moertel C, Fleming T, Creagan E., et al. High-dose vitamin C versus placebo in the treatment of patients with advanced cancer who have had no prior chemotherapy. A randomized double-blind comparison. *New Engl J Med*. 1985 Jan 17; 312(3): 137-41.
- <sup>14</sup> Cameron E, Campbell A. The Orthomolecular treatment of cancer: II. Clinical trial of high-dose ascorbic acid supplements in advanced human cancer. *Chem Biol Interact*. 1974; 9: 285-315.
- <sup>15</sup> Cameron E, Pauling L. Supplemental ascorbate in the supportive treatment of cancer: prolongation of survival times in terminal human cancer. *Proc Natl Acad Sci*. 1976. 73. 3685-89.
- <sup>16</sup> Cameron E., Pauling L. Supplemental ascorbate in the supportive treatment of cancer: re-evaluation of prolongation of survival times in advanced human cancer. *Proc Natl Acad Sci*. 1978 Sep; 75(9): 4538-42.
- <sup>17</sup> Bram S, Froussard P, Guichard M, et al. Vitamin C preferential toxicity for malignant melanoma cells. *Nature* 1980 Apr 17; 284(57):629-31.
- <sup>18</sup> Leung P, Miyashita K, Young M, et al. Cytotoxic effect of ascorbate and its derivatives on cultured malignant and non-malignant cell lines. *Anticancer Res*. 1993 Mar-Apr; 13(2): 475-80.
- <sup>19</sup> Sakagami H, Satoh K, Hakeda Y, et al. Apoptosis-inducing activity of vitamin C and vitamin K. *Cell Mol. Biol* 2000 Feb; 46(1): 129-43.
- <sup>20</sup> Chen Q, Espey M, Krishna M, et al. Pharmacologic ascorbic acid concentrations selectively kill cancer cells: action as a pro-drug to deliver hydrogen peroxide to tissues. *Proc Natl Acad Sci*. 2005 Sep; 102(38): 13604-09.
- <sup>21</sup> Padayatty S., Sun H, Wang Y, et al. Vitamin C pharmacokinetics: implications for oral and intravenous use. *Ann Intern Med* 2004 Apr 6;140(7): 533-37.
- <sup>22</sup> Chen Q, Espey M, Krishna M, et al. Pharmacologic ascorbic acid concentrations selectively kill cancer cells: action as a pro-drug to deliver hydrogen peroxide to tissues. *Proc Natl Acad Sci*. 2005 Sep. 102(38): 13604-09.
- <sup>23</sup> Padayatty S, Riordan H, Hewitt S, et al. Intravenously administered vitamin C as cancer therapy: three cases. *Canadian Med Assn J*. 2006 Mar 28; 174(7): 937-42.
- <sup>24</sup> Akiyama M, Nakamura M. Bone regeneration and neovascularization processes in a pellet culture system for periosteal cells. *Cell Transplant*. 2009 Apr 15. pii: CT-1917. (Epub ahead of print).
- <sup>25</sup> Yogeeta S, Gnanaprasadam A, Senthilkumar S, et al. Synergistic salutary effect of ferulic acid and ascorbic acid on membrane-bound phosphatases and lysosomal hydrolases during experimental myocardial infarction in rats. *Life Sci*. 2006 Dec.23; 80(3): 258-63.

- 
- <sup>26</sup> Lin Y, Tan F, Marra K, et al. Synthesis and characterization of collagen/hyaluronan/chitosan composite sponges for potential biomedical applications. *Acta Biomater.* 2009 Apr 2. (Epub ahead of print).
- <sup>27</sup> Petrella B. Assessment of local proteolytic milieu as a factor in tumor invasiveness and metastasis formation: in vitro collagen degradation and invasion assays. *Methods Mol Biol* 2009; 511:75-84.
- <sup>28</sup> Penna-Martinez M, Ramos-Lopez E, Stern J, et al. Vitamin D receptor polymorphisms in differentiated thyroid carcinoma. *Thyroid.* 2009 Jun; 19(6): 623-8.
- <sup>29</sup> Robien K, Cutler G, Lazovich, D. Vitamin D intake and breast cancer risk in post-menopausal women: the Iowa Women's Health Study. *Cancer Causes Control.* 2007 Sep; 18(7): 775-82.
- <sup>30</sup> Epstein E, Lindqvist P, Geppert B, et al. A population-based cohort study on sun habits and endometrial cancer. *Br J Cancer.* 2009 Jun 23; [Epub ahead of print].
- <sup>31</sup> Giovannucci E. The epidemiology of vitamin D and cancer incidence and mortality: a review. *Cancer Causes Control.* 2005 Mar; 16(2): 83-95.
- <sup>32</sup> Wei M, Garland C, Gorham E, et al. Vitamin D and prevention of colorectal adenoma: a meta-analysis. *Cancer Epidemiol Biomarkers Prev.* 2008 Nov; 17(11): 2958-69.
- <sup>33</sup> Garland C, Gorham E, Mohr F. Vitamin D for cancer prevention: global perspective. *Ann Epidemiol* 2009 Jul;19(7):468-83.
- <sup>34</sup> Giovannucci E. Vitamin D and cancer incidence in the Harvard cohorts. *Ann Epidemiol.* 2009 Feb 19(2): 84-8.
- <sup>35</sup> Shen M, Yen A. Nicotinamide cooperates with retinoic acid and 1,25 dihydroxyvitamin D(3) to regulate cell differentiation and cell cycle arrest of human myeloblastic leukemia cells. *Oncology* 2009; 76(2): 91-100.
- <sup>36</sup> Kizildag S, Ates H. Treatment of K562 cells with 1,25 dihydroxyvitamin D(3) induces distinct alterations in the expression of apoptosis-related genes BCL-2, BAX, BCL(XL) and p21. *Ann Hematol.* 2009 May 28. (Epub ahead of print.)
- <sup>37</sup> Wu W, Zhang X, Zanello L. 1alpha, 25 dihydroxyvitamin D(3) anti-proliferative actions involving vitamin D receptor-mediated activation of MAPK pathways and AP-1/p21 (waf1) upregulation in human osteosarcoma. *Cancer Lett.* 2007 Aug 28. 254(1): 75-86.
- <sup>38</sup> Fujioka T, Suzuki Y, Okamoto T, et al. Prevention of renal cell carcinoma by active vitamin D(3). *World J Surg.* 2000 Oct; 24(10): 1205-10
- <sup>39</sup> Bao B, Yao J, Lee Y. 1alpha, 25-dihydroxyvitamin D3 suppresses interleukin-8-mediated prostate cancer cell angiogenesis. *Carcinogenesis.* 2006 Sep; 27(9): 1883-93.
- <sup>40</sup> Chung I, Han G, Seshadri M, et al. Role of Vitamin D receptor in the antiproliferative effects of calcitriol in tumor-derived endothelial cells and tumor angiogenesis in vivo. *Cancer Res.* 2009 Feb 1; 69(3):. 967-75.
- <sup>41</sup> Yudoh K, Matsuno H, Kimura T. 1alpha, 25-dihydroxyvitamin D3 inhibits in vitro invasiveness through the extracellular matrix and in vivo pulmonary metastasis of mouse melanoma. *J Lab Clin Med.* 1999 Feb 133(2): 120-8.
- <sup>42</sup> Moro J, Iwata M, von Andriano U. Vitamin effects on the immune system: vitamins A and D take centre stage. *Nat Rev Immunol.* 2008 Sep; 8(9): 685-98.
- <sup>43</sup> Montrone M, Martorelli D, Rosato A, et al. Retinoids as critical modulators of immune functions: new therapeutic perspectives for old compounds. *Endocr Metab Immune Disorder Drug Targets.* 2009 June; 9(2): 133-31.
- <sup>44</sup> Kusmartzev S, Su Z, Heiser A, Dannull J, et al. Reversal of myeloid cell-mediated immunosuppression in patients with metastatic renal cell carcinoma. *Clin Cancer Res.* 2008 Dec 15; 14(24): 8270-8.
- <sup>45</sup> Okuno M, Kojima S, Matsushima-Nishiwaki R, et al. Retinoids in cancer chemoprevention. *Curr Cancer Drug Targets.* 2004 May; 4(3): 285-98.
- <sup>46</sup> Wu Q, Dawon, M, Zheng Y, et al. Inhibition of trans-retinoic acid-resistant human breast cancer cell growth by retinoid X receptor-selective retinoids. *Mol Cell Biol* 1997 Nov; 17(11): 6598-608.
- <sup>47</sup> Cannell J, Vieth R, Willett W, et al. Cod liver oil, vitamin A toxicity, frequent respiratory infections and the vitamin D deficiency epidemic. *Ann Otol, Rhinol, Laryngol.* 2008 Nov; 117(11): 864-70.
- <sup>48</sup> Makishima M, Honma Y, Hozumi M et al. Effects of inhibitors of protein tyrosine kinase activity and/or phosphatidylinositol turnover on differentiation of some leukemia myelomonocytic leukemia cells. *Leukemia Res* 1991; 15(8): 701-08.
- <sup>49</sup> Sokolski J, Sartorelli A. Induction of the differentiation of HL-60 promyelocytic leukemia cells by nonsteroidal anti-inflammatory agents in combination with low levels of vitamin D3. *Leuk Res* 1998 Feb; 22(2): 153-61.
- <sup>50</sup> Shen M, Yen A. Nicotinamide cooperates with retinoic acid and 1,25 dihydroxyvitamin D(3) to regulate cell differentiation and cell cycle arrest of human myeloblastic leukemia cells. *Oncology* 2009; 76(2): 91-100.

- 
- <sup>51</sup> Kulp K, Montgomery J, Nelson D, et al. Essiac and Flor-essence herbal tonics stimulate the in vitro growth of human breast cancer cells. *Breast Cancer Res Treat.* 2006 Aug; 98(3): 249-59.
- <sup>52</sup> Seely D, Kennedy D, Myers S, et al. In vitro analysis of the herbal compound Essiac. *Anticancer Res.* 2007 Nov-Dec; 27(6B): 3875-82.
- <sup>53</sup> Leonard S, Keil D, Mehlman T, et al. Essiac tea: scavenging of reactive oxygen species and effects on DNA damage. *J Ethnopharmacol.* 2006 Jan 16; 103(2): 288-96.
- <sup>54</sup> Ottenweller J, Putt K, Blumenthal E, et al. Inhibition of prostate cancer cell proliferation by Essiac. *J Altern Complement Med.* 2004 Aug; 10(4): 687-91.
- <sup>55</sup> Hale L, Hynes B. Bromelain treatment of human T cells removes CD44, CD45RA, E2/MIC2, CD6, CD7, CD8 and Leu 8/LAM1 surface molecules and markedly enhances CD2-mediated T cell activation. *J Immunol* 1992 Dec 15; 149(12): 3809-16.
- <sup>56</sup> Eckert K, Grabowska E, Strange R, et al. Effects of oral bromelain administration on the impaired immunocytotoxicity of mononuclear cells from mammary tumor patients. *Oncol Rep.* 1999 Nov-Dec; 6(6): 1191-9.
- <sup>57</sup> Batkin S, Taussig S, Szekerezes J. Antimetastatic effect of bromelain with or without its proteolytic and anticoagulant activity. *J cancer Res Clin Oncol.* 1988; 114(5): 507-8.
- <sup>58</sup> Báez R, Lopez T, Salas C, et al. In vivo antitumoral activity of stem pineapple (*Ananas Comosus*) bromelain. *Planta Med* 2007 Oct; 73(13): 1377-83.
- <sup>59</sup> Proefrock K. Botanical considerations for lung cancer patients. (Lecture) Southwest Conference on Botanical Medicine. Tempe, AZ USA. 2001.
- <sup>60</sup> Jones, M, Young, R, Scully R. Endometrial adenocarcinoma with a component of giant cell carcinoma. *Int J Gynecol Pathol.* 1991; 10(3): 260-270.