Lesson 11
Meeting Life: Cancer, Immunodeficiency, and Botanical Medicine

Even though there can be a very bright shining feeling for people who treat and are treated by Herbal Medicine for cancer, there are some initial clouds and shadows to look at. For every person you treat successfully there is a great feeling. It is important to realize from the on start that a Herbalist will not be successful with every patient, in fact not even the majority. Among the different diseases a Herbalist is likely to encounter, cancer is perhaps the most challenging to treat successfully. This is not because botanical and natural therapies are not efficacious in the treatment of cancer, but is due in part to the fact that cancer represents a malignant change in a long-standing condition, and like all long-standing, chronic conditions, it is difficult to overcome the spiral of degeneration once this pattern sets it. The difficulty also stems from an emotional and cultural fear of cancer. Many practitioners have said that the word CANCER kills more people than the disease itself does.

Botanical treatment of cancer is compounded by a number of other issues as often a cancer patient will only visit a herbalist when the prognosis is poor and/or conventional oncological therapies are considered ineffective. This behavior essentially decreases the likelihood of successful treatment and to some extent, facilitates a bias against using herbal treatments, which are probably not going to be effective in such circumstances. Furthermore, cancer patients are often counseled against using herbal and nutritional therapies during conventional treatment. Such counsel discounts the benefits of synergism between natural products, radiation and chemotherapy, and the benefits of using natural therapies to protect the body.
against the harmful effects of chemotherapeutic drugs and radiation. This antagonism to holistic interventions can present ethical problems for the herbalist, especially when the patient wants to use these therapies regardless of the advice of their oncologist.

Lastly, the holistic perspective suggests that cancer should not be viewed as a disease of simple biomechanics, but a confluence of physical, psychological and spiritual factors, which must be addressed simultaneously. Thus cancer is not simply a physical disease, but is also a disease of the mind and spirit, and to this extent, especially when the prognosis is poor and the patient is dying, efforts must not only be made to make the patient as comfortable as possible, but to help them die with dignity and clarity. Our culture has an enormous fear of death, and it is important that dying patients make this transition feeling empowered and at peace. Often the Herbalist has to be comfortable with the mere possibly of extending the person’s life, with a better quality of life.

It might be hard to see at first, but one of the cures for cancer is death. The patient stops feeling the pain, (physical, emotional and mental). From a simple biomechanical point of view this does not make sense, but from a large spiritual point of view it often does. One complication always present with any terminal illness is the question of family, close friends and support people. Often the patient tries to hold on for the sake of these people, instead of following their own impulses. It is often said that we are very alone at the end. This is always a time of reflection and of coming to peace with their emotional, mental and spiritual visions.

Role of Herbalist in Cancer Treatment

The figures say 33–83% of people with cancer use complementary and alternative medicines (CAM) after diagnosis, depending on which country they live in. The most likely users in Canada are educated women with breast cancer. Patients using CAM are more optimistic than others and the most common reason they chose to use CAM was the increase of hope. In Europe, 48% of people with the diagnosis of cancer use herbal medicines. Patients combine
conventional medicine and complementary therapies, with approximately 80% of people with cancer who use CAM also undergoing conventional treatment.

Part One: Biomechanical Mechanisms of Neoplasia

There are many types of Cancer, or neoplasia, an uncontrolled proliferation of anaplastic cells, with the most common one being a tumor, which will have a tendency to invade surrounding tissue and may metastasize through the blood or lymphatic systems to other tissues. Tumors are classified into two basic types, benign and malignant.

Benign tumors

Benign tumors do not penetrate to adjacent tissues and nor do they metastasize. They tend to be more highly differentiated than malignant tumors, more closely resembling the tissue of origin. Benign tumors are generally denoted by the suffix −oma, the term preceding it indicating the cell type. Thus a benign tumor of the squamous epithelium is termed an epithelioma, and when branched and growing outward is called a papilloma. A benign tumor that arises from a gland is called an adenoma, or adenomatous tumor. (Rubin 2001, 92-93)

Malignant tumors

Malignant tumors are tumors that actively invade and penetrate adjacent tissues and/or metastasize to sites distant to the tumor, spread by the blood or lymphatic system. Unlike benign tumors, malignant tumors are described as anaplastic, referring to a loss of cell differentiation within
the affected tissue such that it assumes a more primitive form. There are many different ways of describing malignant tumors, based on the location of the tumor, its morphology, and/or the cell type(s) involved. Thus there are a great variety of terms used to describe a malignant tumor. (Rubin 2001, 93-95)

The term **carcinoma**, refers to malignant cancers of the epithelium and are the most common type of cancer. They are usually differentiated based upon their location and the type of epithelial tissue involved. Thus a *gastric adenocarcinoma* refers to a malignant tumor of glandular epithelial tissues of the stomach. A *colorectal mucoid carcinoma* refers to malignant epithelial cells in the colon and rectum that secrete mucin. The term *esophageal squamous cell carcinoma* refers to a malignant growth in the thickened epithelium of the esophagus. (Rubin 2001, 93-95)

The term **sarcoma** refers to relatively rare malignant tumors of connective tissues such as cartilage, muscle, bone, fibrous connective tissue and blood vessels. The term *fibrosarcoma* refers to cancer of the fibrous sheath of a muscle whereas a *chondrosarcoma* refers to the growth of malignant chondrocytes in bone tissue. (Rubin 2001, 93-95)

Sometimes the term used to describe a malignant tumor only describes its location, such as a *hepatoma* (neoplasm of the liver), *lymphoma* (neoplasm of the lymphatic system) and *seminoma* (neoplasm of the testis).¹ Malignant neoplasms of the blood are designated with the suffix –*emia*, as in *leukemia*. Malignant tumors of skin cells that produce melanin are called *melanomas*. Cancers that affect the nervous system have various descriptors depending on the cell type or location involved, most often described as *gliomas*, comprised of malignant glial cells, including astrocytoma and glioblastoma (affecting astrocytes), and

¹ Note that hepatoma, lymphoma and seminoma use the suffix –*oma*, which technically speaking, describes benign tumors. Nonetheless, these are all highly malignant tumors, and thus exemplify the confusion in the terminology used to classify various cancers. These terms can be seen to be a hold out from an earlier time in medicine, before the meaning and definition of the various suffixes used to describe tumors was firmly established.
medulloblastoma (tumor of the cerebellum). (Rubin 2001, 93-95)

Malignant cancers can vary to a large degree in their size and shape, and are also classified according to their morphological and functional characteristics. Such terms include papillary, which refers to a frond-like structure in a tumor; medullary, referring to a soft cellular tumor with little connective tissue; scirrhous or desmoplastic, referring to a tumor with dense fibrous stroma; colloid carcinoma, which secretes mucus around islands of tumor cells; and comedo carcinoma, which refers to an intraductal neoplasm in which necrotic material can be expressed from the ducts, most commonly seen affecting the mammary glands of the breast. It is the use of such secondary terms from which more complex terms are derived, such as papillary serous cystadenocarcinoma of the ovary, or polypoid adenocarcinoma of the stomach. In cases where the histology is poorly understood an eponym is usually retained, such as Hodgkin’s disease or Ewing sarcoma. (Rubin 2001, 93-95)

Cancer growth and development

A tissue is comprised of thousands of different cells, all in constant communication, either directly through gap junctions, or indirectly through the release of hormones and cytokines. Each of the cells within a tissue performs a specific function, contributing to the greater good of that tissue and the body as a whole. In normalcy, when a cell becomes damaged or is no longer required, it undergoes apoptosis, a term derived from a Greek word that refers to the falling of leaves at the end of the summer. Unlike the other form of cell death called necrosis, apoptosis occurs as a scattered event in a given tissue, involving only a few cells. Further, apoptosis does not involve the rupture of the plasma membrane and the release of inflammatory compounds that characterizes necrosis, thus sparing adjacent cells from damage. As a cell undergoes apoptosis phagocytic cells locate it and engulf it before the plasma membrane is damaged. (Rubin 2001, 93-121; Boik 2001, 1-2)
It appears that cells are programmed to undergo apoptosis by default, and survive only from the release of factors that essentially tell it to keep living. These “keep living” signals come from two sources: from genes within the cell itself such as bcl-2 (which appear to be over expressed in cancer cells), and from the release of cytokines and cell-to-cell communication in gap junctions. The messages that tell a cell to undergo apoptosis come from three sources. The first is mediated by cellular damage, and specifically, damage that involves the DNA. The gene p53 will attempt to repair the damage, but if the condition is irreparable this same gene will induce apoptosis. The second mechanism that induces apoptosis is from the release of growth-inhibiting cytokines such as transforming growth factor beta (TGF-β). TGF-β regulates cell differentiation and proliferation, and can either stimulate or inhibit cell development. In early cancer cells TGF-β induces apoptosis: in later stage cancer cells however TGF-β can work in an opposite fashion, suppressing immune vigilance (so the cancer cells escape detection) and promoting cellular proliferation. The third mechanism that induces apoptosis comes from within the cell itself through the expression of genes such as Bax. When stimulated these genes express proteins that promote apoptosis; in cancer cells however these genes can be mutated and/or can be under-expressed, thereby inhibiting apoptosis. (Rubin 2001, 93-121; Boik 2001, 31-33).

From the above, it can be seen that cancer is a fundamental disruption in the regulation of cell growth and differentiation, and a failure to undergo apoptosis. A variety of mechanisms have been described that interfere with this event, and will be discussed in more detail. When a cell no longer responds to the default mechanisms that cause it to undergo apoptosis (essentially “forgetting” who it is, or failing to respond to the signals released by the community of cells in which it resides, “isolating” itself from these cells), this cell is at the mercy of its environment. This stage is called initiation. If this cell is under the influence of a proliferating agent, such as a foreign chemical, a virus or inflammatory processes, this precancerous cell divides to form daughter cells, which in turn, divide into more daughter cells, all of which respond to the same promoting agent. This stage is called
promotion. At some point these cells undergo a mutation that makes them capable of self-stimulation, marking the formation of a true cancer cell. Such a cell no longer requires the stimulus of the promoting agent, and manufactures its own cytokines and hormones to ensure its continued, unchecked, growth and development. As this clump of cells grows it becomes capable of invasion and metastasis, evading immune activity, mutating under adverse conditions, and inducing the growth of a network of blood supply to it in a process called angiogenesis. This final stage of carcinogenesis is called progression. (Rubin 2001, 93-121; Boik 1995, 5-7)

Events identified in the process of carcinogenesis

Researchers have identified a number of events that can be seen to occur in cancer growth and development. These include:

1. The induction of genetic instability and the resultant abnormal expression of genes;
2. Abnormal signal transduction;
3. Abnormal cell-to-cell communication;
4. Evasion of immune detection;
5. Induction of angiogenesis;
6. Invasion and metastasis. (Boik 2001, 2-4)

The following diagram illustrates these events:

1. Gene mutations, abnormal gene expression
2. Abnormal signal transduction
3. Abnormal cell-to-cell communication
4. Production of immunosuppressive compounds, immune evasion
5. Induction of angiogenesis
6. Invasion and metastasis

Cellular events in carcinogenesis (Boik 2001, 3)
Genetic instability and abnormal gene expression

Cellular functions are guided and maintained by the expression of genes that encode for the synthesis of certain proteins, found within the DNA. Each DNA strand can be thought of as a kind of “library” of different “cookbooks” (i.e., genes) that describe the creation of certain “recipes” (i.e., proteins). In order for the recipes to be used, however, the DNA must be transcribed by a “chef,” called RNA (ribonucleic acid). In a reaction catalyzed by RNA polymerase, paired nucleotides within a certain portion of the DNA are broken apart. The RNA then slips in between the DNA strands that have been broken apart (i.e., a specific gene), and copies the genetic information onto itself. When the “chef” (i.e., the RNA) has completely transcribed the “recipe” (i.e., the gene), the “chef” leaves the library (i.e., the DNA, in the nucleus) and goes into his “kitchen” (i.e., the cytosol) to “cook the meal” (i.e., initiate protein synthesis). In these two processes of transcription and translation, or gene expression, the result is the creation of a specific protein with a biological function that in some way affects the cell, or if released as a hormone or cytokine, the other cells around it. (Boik 2001, 14-15) (For a review of cell biology please refer to the Wild Rose College course Introductory Biology and Biochemistry.)

In a normal, healthy cell, genes are constantly being expressed to maintain the normal function of the cell. In contrast, the unregulated growth of cancer cells results from the acquisition of mutations in genes that control cell growth and differentiation. These mutations can be the result of several mechanisms, the most common of which

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<th>ONCOGENES</th>
<th>ACTION</th>
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| Bcl-2     | • inhibits apoptosis  
|           | • prevents damage by free-radicals |
| c- myc    | • affects cell proliferation, differentiation and apoptosis |
| fos and jun | • act as transcription factor  
|           | • facilitates proliferation |
| HER-2/neu | • facilitates signal transduction |
| MDM2      | • inhibits p53 activity |
| ras       | • promotes DNA synthesis  
|           | • promotes chromosomal abnormalities  
|           | • facilitates tumor invasion |

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<th>SUPPRESSOR GENES</th>
<th>ACTION</th>
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| Bax              | • competes with Bcl-2  
|                  | • promotes tumor death  
|                  | • induces apoptosis |
| Genes that produce connexion proteins, e.g. Cx32 and Cx43 | • form gap junctions to enable cell-to-cell communication |
| p53              | • normal (“wild”) type suppresses tumor growth  
|                  | • initiates tumor repair  
|                  | • induces apoptosis  
|                  | • mutant p53 allows proliferation of cells with DNA damage |

Table 1: Selected oncogenes and tumor suppressor genes (Boik 2001, 18)
relate to apparently spontaneous errors in DNA replication and repair. The genes that are involved in the pathogenesis of cancer are oncogenes, altered versions of normal genes that code for malignant transformation. One of the causes for this alteration is thought to lie in the activity of oncogenic viruses that infect a cell and alter the host DNA to produce mutant neoplastic cells. Human cells however have also been shown to naturally contain proto-oncogenes that, when activated through genetic errors, result in DNA mutation independent of viral stimulation. One important component in oncogenesis is the tumor-suppressor genes that are involved in normal cellular differentiation and inhibit the proliferative activities of the oncogenes. In this case the evolution of a mutant neoplastic cell is thought to be a failure or loss of tumor suppressor gene activity. (Boik 2001, 18-19)

The root cause of oncogene over-expression and tumor suppressor gene under-expression is gene mutation. Mutations can also affect the cell in other ways, such as the abnormal production of transcription factors or abnormal signal transduction. DNA mutation occurs on two basic levels, called classical mutation and epigenetic mutation. (Boik 2001, 18-19)

There are many theories of why these mutations happen. As mentioned above viruses are thought to be involved. In some cases environmental factors, such as chemicals, seem to be involved. Emotional stress and more important, long standing emotional blocks are theorized to be the major cause of the mutation. With more research it might be found to be a combination of the above.

Classical mutation describes a process by which mutation results in a change in the DNA base sequences, or in other words, changes to the order in which bases occur within a gene, altering the structure of the gene (i.e. the genotype). The result of this mutation is an altered phenotype, or an alteration in how the gene expresses itself through protein synthesis. Such mutations occur with great frequency and most are detected and repaired. If a mutation escapes notice by the DNA repair system however, and does not have toxic effects in the cell, it will be passed along (i.e. inherited) to subsequent daughter cells. Such mutations are
considered to be an important component of biological evolution, inducing phenotypic changes in response to environmental stimuli. Thus most species are programmed for some degree of mutation, but if the damage is significant, or if it occurs in oncogenes or tumor suppressor genes, the result can be carcinogenesis. (Boik 2001, 19)

The factors that promote classical mutation are numerous. Some mutations occur spontaneously, resulting in the transformation of one base into another; a feat easily understood when one appreciates the similarity in their chemical structure, only involving the addition or removal of a few atoms. Some mutations occur as a result of external agents that similarly alter a base in some way. For example, ultraviolet light can fuse bases together; alkylating compounds such as epoxides and aziridines can add a methyl group to a base; free radicals such as the hydroxyl radical can add an oxygen group to a base. The net result of such chemical reactions is an alteration of the base sequence and gene expression, and in some cases, the promotion of carcinogenesis. (Boik 2001, 19-20)

**Epigenetic mutations** are different from classical mutations in that they do not involve a change in the sequence of bases, and are reversible. Within DNA, epigenetic changes are characterized by the attachment of a methyl group to a specific location in a cytosine base. Such a mutation plays an important role in determining which genes are activated for transcription, and as a result, functions as a kind of switch to determine which proteins are synthesized. The degree of cytosine methylation determines the degree to which that gene is expressed: the greater the methylation, the more strongly the gene is inhibited. Unlike classical mutations, the body does not perceive epigenetic mutation as a DNA error, and thus the pattern of this kind of methylation is passed along from the parent to the daughter cells. Nonetheless, abnormalities in cytosine methylation play an important role in carcinogenesis. Most genes in cancer cells tend to be hypomethylated and thus over-expressed. Or, in other cases, genes such as the tumor suppressor gene p53 that inspects and repairs damage to DNA can be hypermethylated and thus under-expressed. For example, the mutation and inhibition of p53 allows for classical
mutations to occur with greater frequency. Hypermethylation also silences the genes involved in adhesion proteins that allow for cell-to-cell communication, facilitating cancer progression. It has also been reported that when a methyl group is added to cytosine, the entire combination can be easily switched to another base, thereby promoting classical mutations. It is also thought that epigenetic changes play a role in switching metastasis on and off. (Boik 2001, 20-21)

Both classical and epigenetic mutations play a role in promoting genetic instability. It is the instability that allows the cancer cell to survive adverse conditions, including immunovigilance, chemotherapeutic drugs, and impaired nutrient and oxygen supply. These mutations give a cancer cell a distinct advantage over other cells, in that it is no longer working for the collective good, and can “selfishly” serve its own priorities, with a single-minded determination to out-compete neighbouring cells that still cling to a collective vision. (Boik 2001, 21-22)

Abnormal signal transduction

**Signal transduction** is a term that describes how a message from outside is relayed into the nucleus of a cell, where it acts to modify gene expression. One of the most important sources of these messages are the variety of **hormones** and **cytokines** (also called **growth factors**) that either pass through the plasma membrane, or bind to receptors on the surface of the plasma membrane, to alter the activity of a cell. (Boik 2001, 37-38)

**Protein phosphorylation signaling**

Unlike fat-soluble hormones (e.g. estradiol, testosterone), water-soluble hormones (e.g. epinephrine) and cytokines (e.g. TGF-β) must bind to a receptor on the surface of a plasma membrane, called a **first messenger**, to alter the cell’s function. Once the receptor has been stimulated, the **second messenger** then relays messages inside cell. (Boik 2001, 40)

The most common secondary messenger is **cyclic AMP (cAMP)**, synthesized from ATP by **adenylate cyclase**. The activities of cAMP are regulated by the activities of an
enzyme called phosphodiesterase. Hormones and cytokines that up-regulate the function of a cell’s activity involve the increased synthesis of cAMP. Receptors are attached to molecules called G-proteins, and when a G-protein is activated, it in turn activates adenylate cyclase to synthesize cAMP. Special enzymes called protein kinases are activated by cAMP, which may be free or bound to the plasma membrane. Protein kinases are called phosphorylating enzymes, because they remove a phosphate group from ATP and add it to a protein, usually another enzyme. Phosphorylation affects the function of other enzymes that act upon substrate molecules, and can activate or deactivate them, working much like an on/off switch. The process of phosphorylation regulates the activities of the cell, such as the initiation of protein or glycogen synthesis, or changing the permeability of the plasma membrane. (Boik 2001, 40-41)

An amplification of the effect of the hormone occurs as the first messenger activates 100 or so G-proteins, each of which then activates an adenylate cyclase molecule. If each adenylate cyclase then activates about a thousand cAMP molecules, this would result in the activation of over 100,000 secondary messengers in-cell through the act of phosphorylation. It is the millions of phosphorylated enzymes that catalyze these chemical reactions that produce the physiological changes induced by the hormone or cytokine.

As has been previously discussed, the various cytokines play an important role in regulating cell function. Cytokines include:
- epidermal growth factor (EGF)
- fibroblast growth factor (FGF)
- Insulin-like growth factor (IGF)
- platelet derived growth factor (PDGF)
- transforming growth factor alpha and beta (TGF-α and TGF-β)
- vascular endothelial growth factor (VEGF) (Boik 2001, 38-39)

In the case of the cytokines mentioned above, all

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<th>CYTOKINE</th>
<th>ACTION IN CANCER</th>
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| EGF      | • activated by EGF and TGF-α  
• increases cell proliferation, cell motility, invasion and metastasis |
| FGF      | • stored in the extracellular matrix  
• stimulates cell proliferation and angiogenesis |
| IGF      | • shares similar functions to insulin, but does not stimulate glucose utilization specifically |
| PDGF     | • stimulates proliferation of epithelial cells as well as other cells  
• stimulates wound healing |
| TGF-α    | • binds to EGF receptors  
• induces angiogenesis |
| TGF-β    | • can either increase or decrease cell proliferation, depending on cell type and environment  
• in early stages, acts as an inhibitory agent  
• advanced cancers become resistant to it  
• promotes invasion and metastasis  
• immunosuppressive |
| VEGF     | • induces endothelial proliferation and vascular permeability  
• plays a role in angiogenesis |

Cytokines and their actions in cancer cells (Boik 2001, 39)
(except TGF-β, which uses a receptor type comprised of three distinct proteins) utilize **protein tyrosine kinases (PTK)**. The manufacture of these PTK receptors can be produced by oncogenes, and by producing an excessive amount or by making them hypersensitive to stimulation, a cancer cell can upregulate its cellular functions. Although cytokines are usually produced to modify the activity of a neighboring cell, cancerous cells are unique in that they can produce their own cytokines (allowing self-stimulation), as well as significantly enhance the number of receptors for cytokines. Cancer cells can also produce free-radicals, which make cytokine receptors more responsive to stimulation. (Boik 2001, 39-41)

Another mediator of signal transduction is **protein kinase C (PKC)**, a family of at least 12 enzymes, many of which are over-expressed in cancer cells. It is specifically required in angiogenesis and metastasis in some types of cancer. It has also been reported to play a role in telomerase, an enzyme that increases the longevity of cancer cells, and plays a role in tumor invasion and drug resistance. (Boik 2001, 39-41)

**Cyclin-dependent kinases (CdKs)** are not involved with signal transduction but help control signals that drive the cell cycle, namely, by activating cell proteins called **cyclins** that help instruct the cell to begin mitosis. The ability to stimulate cyclins by CdKs is in part inhibited by a family of CdK inhibitors such as p21 and p27. In healthy cells, these CdK inhibitors halt cell proliferation and promote DNA repair, as well as mediate the activity of TGF-β. In cancer cells CdK inhibitors such as p21 and p27 are usually under-expressed, leading to unchecked cell replication. (Boik 2001, 42-43)

**Ras proteins** are expressed by ras oncogenes, and also play a role in abnormal signal transduction and are usually over produced by cancer cells. Specifically, ras proteins play a role in **mitogen-activated protein kinase (MAPK)** signal transduction, which plays an important role in cell proliferation by interacting with nuclear transcripting factors. Ras proteins also activate PKC. (Boik 2001, 43-44)

**Reduction-oxidation signaling**
Apart from protein phosphorylation, the balance between pro-oxidants and antioxidants also plays a role in the transcription of genes. The fluctuating concentration of free-radicals can act as a kind of switch, turning on and off a variety of cellular processes, including enzyme activation, gene transcription, and gene expression. As in protein phosphorylation, reduction-oxidation (redox) signaling is often abnormal in cancer cells. (Boik 2001, 53-54)

Free radicals are in large part derived from normal cellular processes, from molecular oxygen that is converted to reactive oxygen species (ROS) during cell respiration. ROS are also generated with immune activity, when macrophages release superoxide, or in redox reactions involving trace metal ions such as copper and iron. Other sources of ROS include the over-expression of ras oncogenes, and the activation of transcription factors such as nuclear factor kappa-beta (NF-κβ), which leads to the over-production of TNF and other proinflammatory cytokines. (Boik 2001, 56-57)

To protect against these oxidants the body maintains a number of antioxidant compounds, some of which are directly obtained from the diet (e.g. vitamin A, C and E), and others that are manufactured from components of the diet (e.g. catalase, glutathione peroxidase and super oxide dismutase). These compounds donate electrons to ROS species to make them more stable; they then either become less harmful free radicals, inert substances, or useful compounds such as water. Cancer cells are often found to be deficient in the enzymes catalase and SOD, the net result of which is an increased production of hydrogen peroxide and superoxide, which can produce changes within the cancer cell that favor its growth and development. (Boik 2001, 52-54)

The excessive production of ROS and free radicals assist in cancer cell proliferation and survival in a variety of ways. This includes damaging DNA, which promotes mutations, as well as increasing some types of redox signaling that promotes cell proliferation (e.g. NF-κβ), or inactivating certain transcription factors that limit cell proliferation (e.g. p53). Cancer cells typically rely upon abnormally low
levels of p53 and abnormally high levels of NF-κB. (Boik 55-58)

Abnormal cell-to-cell communication

All cells in the body are in constant communication with adjacent cells through cell-to-cell communications mediated by cell adhesion molecules and gap junctions. **Cell adhesion molecules (CAMs)** are specialized proteins (e.g. integrins, cadherins, selectins and immunoglobulins) located on the surface of the plasma membrane that bind cells to one another. **Gap junctions** are structures that exist between cells that act as portals through which there is a free exchange of ions and small molecules, allowing for instantaneous communication. Unlike normal, healthy cells, the cytokines produced by cancer cells cause them to detach from the surrounding healthy cells and grow in isolation, thereby limiting the flow of messages that would tell the cancer cell to undergo apoptosis. Since these same communications also provide cytokines that signal the message to “keep living,” cancer cells also evolve the production of their own cytokines to ensure continued growth and development. (Boik 2001, 67-68)

Immunosuppression and immune evasion

Immune cells are an important component of inhibiting carcinogenesis, when the cancer cell’s DNA is damaged and no longer has the capacity to “listen” to its neighbors and undergo apoptosis. Immune cells are active in searching for and destroying tumor cells include T cells, natural killer (NK) cells, and macrophages. **T cells** rely upon the activity of antigen presenting cells (APCs) to engulf and process the tumor cell, and present the altered antigens together with MHC-I self-antigens on its plasma membrane. Tumor cells however often disrupt how antigens are presented on the surface of the plasma membrane due to their altered genetic structure, and thus escape T cell recognition. This modulation of surface proteins includes receptors for the cytokine TNF, which cannot bind to the tumor cell and therefore cannot destroy it. **Natural Killer (NK) cells** are an important mechanism against the metastasis of tumor cells, and although they normally constitute 15% of the total lymphocyte
population, are often found deficient in cancer patients. Like NK cells, macrophages are an important component of tumor cell destruction, and function independently of antigen recognition. A depression in the number of circulating NK cells and macrophages is a feature of the immunosuppression that occurs with cancer, and in turn, adversely affects T cell activity, and promotes immune evasion. (Boik 2001, 60-61; 123-25)

Immune cells destroy tumor cells by releasing large volumes of ROS, which disrupts the integrity of the plasma membrane and causes cell lysis. Tumor cells however are able to suppress immune activity by releasing immunosuppressive cytokines such as interleukin (IL)-10, TGF-β, and prostaglandin (PG) E2. The inhibitory effects of such factors upon immune activity can result in a mild release of ROS that can actually promote tumor growth by modulating transcription factors and signal transduction. (Boik 2001, 60-61)

**Induction of Angiogenesis**

Angiogenesis is the growth of new blood vessels, a process that occurs in healthy tissues (such as during the development of the endometrium or in wound healing), but also in pathologies such as atherosclerosis, rheumatoid arthritis, and cancerous tissues. In many respects the mechanisms between normal and cancer-induced angiogenesis are identical. In order for a tumor to grow it must enhance its vascular supply, and to this end, many tumors will secrete cytokines such as PDGF, FGB and TGF to stimulate the growth of new blood vessels in the host tissue. VEGF in particular promotes vascular permeability, which appears to be an important rate-limiting step in angiogenesis. Another important component in inducing angiogenesis is prostaglandin and leukotriene synthesis. Cancer cells appear to synthesize excessive
levels of PGE$_2$ and leukotrienes, in most part derived from the consumption of vegetable oils, increasing cellular proliferation. (Boik 2001, 79-88)

Cells deprived of oxygen release lactic acid while metabolizing glucose for energy, which like the cytokines mentioned above, acts as an angiogenic cytokine. Despite a rapacious appetite for glucose and oxygen, tumor cells often experience hypoxic conditions, through the process of local inflammation (which inhibits blood flow), and the chaotic development of blood vessels in tumors. Within tumors, this leads to large masses of cells that exist in a hypoxic state, impairing access to it by immune cells and other anti-tumor substances. These hypoxic conditions also cause the central portions of the tumor to become necrotic. These prolonged states of hypoxia combined with the increased growth and development of the tumor greatly enhance glycolysis (up to five times greater than normal), and causes an excess of lactic acid to be produced, facilitating angiogenesis. Local macrophages, also under the influence of hypoxic conditions, release cytokines that enhance angiogenesis. As angiogenesis occurs and tumors become larger, they retain a high capacity for glucose, often robbing the body of vital energy. This can be seen in a positron emission tomography (PET) scan, in which a cancer patient is given a small intravenous dosage of radiolabelled glucose and then surveyed by the scanning equipment. After administration the marker compound, being glucose, can be seen to be rapidly taken up by the tumor, and identifies the tumor location, and also its appetite for glucose (which has implications for dietary strategies). (Boik 2001, 79-88)

Insulin is another cytokine that enhances glycolysis and promotes angiogenesis. In cancer patients, high insulin levels are often found generally throughout the body, and specifically in cancerous tissues. Non-insulin dependent diabetes mellitus (NIDDM) itself has been implicated as a risk factor in various cancers. (Boik 2001, 79-88)

**Invasion and Metastasis**

Most malignant tumors grow within the tissue of origin where they enlarge and begin to invade surrounding tissues
and organs in a process called invasion. The process of invasion by tumor cells involves the secretion of compounds that dissolve the extracellular matrix (ECM). The ECM mesh of interlacing glycosaminoglycans (GAGs), such as hyaluronidase, that lies between cells, serves as a conduit for the transfer of electrolytes, metabolites, dissolved gases, nutrients, hormones, vitamins, cytokines and enzymes. The ECM also functions as a barrier against tumor invasion. Tumor cells however secrete enzymes such as glycosidases, proteases, and hyaluronidase that break down the ECM, facilitating invasion. Examples of tumor invasion include squamous carcinoma of the cervix, which may grow and extend into the vagina to produce fistulas and obstruct the ureters. (Boik 2001, 105-12)

Metastasis refers to the transfer of malignant cells from one site to another site that is not directly connected to it. In much the same way that a malignant tumor can penetrate the parenchyma of surrounding tissues, neoplastic cells may come into contact with the blood and lymphatic system through which they are disseminated to distant locations. In hematogenous metastasis malignant cancer cells invade local capillaries and venous tissue, forming a thrombus on the vessel surface. As the thrombus grows particles of the thrombus tend to break off and form neoplastic emboli which then travel through the blood. Malignant abdominal tumors spread via the hepatic portal vein and thus are usually responsible for liver metastasis. The invasion of other systemic veins frequently leads to neoplastic emboli being carried to the vena cava, and through the pulmonary vein to the lungs. In lymphatic metastasis neoplastic cells invade local lymphatic channels, forming microscopic neoplastic emboli in the lymphatic stream. These neoplastic cells are then taken to the nearest lymphatic node, where it invades the tissue, causing it to become enlarged and hard. Lymphatic metastasis frequently involves the occlusion of the affected neoplastic tissue, causing the lymphatic flow and the neoplastic cells contained within it to be directed or reversed to other parts of the lymphatic system. This accounts for the often irregular spread of lymphatic metastasis. As the cancer develops, progressively more and more lymphatic tissue is affected, spreading the cancer to
Causes of cancer

In the previous section we reviewed the basic mechanisms that to lead to carcinogenesis, such as genetic mutation, abnormal gene expression, abnormal cellular communication, evasion of immune detection, the role of angiogenesis, and invasion and metastasis. The following is an overview of a variety of factors that are known to promote the mutations that underlie these processes.

Cancer is a multifactorial disease that modern medicine still knows very little about. Many theories exist as to what may cause cancer and only a few cancers have definitive risk factors (for example, cigarette smoking is felt to cause lung cancer, or human papilloma (HPV) might have a connection to cervical cancer). However, even with these connections, not everyone who smokes gets lung cancer and not everyone with HPV gets cervical cancer. As cancer is a multifactorial disease, we cannot say that one issue is the causal factor for a particular type of cancer. That being said, the following table shows some of the current ideas on possible general theories incorporating medical and naturopathic causes of cancer.

<table>
<thead>
<tr>
<th>POSSIBLE CAUSE</th>
<th>EXPLANATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetics</td>
<td>Certain cancers, such as breast, prostate and colon cancer, have been found to have some hereditary links. There are certain genetic markers that the medical fraternity can test for that have been linked to certain cancers, e.g. the BRCA gene for breast cancer. It is important to remember, just because a person has a ‘gene’, does not mean it is turned on. Epigenetics plays a large role here.</td>
</tr>
<tr>
<td>Viruses and infection</td>
<td>Viruses such as HPV have been linked with the development of certain cancers. Examples of these include human papillomavirus (cervical cancer); Epstein-Barr virus (non-Hodgkin’s lymphoma); HIV (Kaposi’s sarcoma, primary central nervous system lymphoma, invasive cervical cancer and non-Hodgkin’s lymphoma); hepatitis B virus (hepatocellular carcinomas); retroviruses (adult T-cell leukaemia); Helicobacter pylori (gastric cancer in people with certain polymorphisms); simian virus 40 (non-Hodgkin’s lymphoma). Again</td>
</tr>
</tbody>
</table>

other organs and tissues. (Rubin 2001, 98-100; Boik 2001, 113-16)
it is important to note that not all, in fact very few, people who come in contact with these viruses end up with the associated cancer. The overall terrain and health of the individual has to be taken into consideration (physically, emotionally, mentally and spiritually).

<table>
<thead>
<tr>
<th>Topic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitochondrial dysfunction</td>
<td>Cancer progression has been linked with mitochondrial dysfunction. This can be a result of mitochondrial DNA (mtDNA) mutations and/or depletion. These mutations or mitochondrial depletions have also been linked with an increase of drug resistance within cells. This mitochondrial dysfunction has been found in certain cancers such as prostate cancer.</td>
</tr>
<tr>
<td>Environmental influences</td>
<td>There has been a significant increase in cancer in the Western world over the last 50 years. Epidemiological studies have associated this increase with exposure to environmental toxins such as organochlorides and synthetic pesticides, with fetuses, infants, children and young adults being the most at risk. Mesothelioma has a direct link with exposure to asbestos.</td>
</tr>
<tr>
<td>Immune system</td>
<td>A weak immune system may increase the chance of cancer developing. The natural killer cells (NK) are the main surveillance system of the body, protecting against cancer formation and infection. If NK cells are not functioning correctly, tumors are more likely to arise through abnormal cell development.</td>
</tr>
<tr>
<td>Cell cycle mitosis malfunction</td>
<td>In cell replication certain checkpoints and cellular activities maintain the health of the cell. If these checkpoints, such as p53, have mutations or there are abnormal centrosomes in a cell, cancer cells may develop. The elderly in particular are prone to malignant tumors that are related to the accumulation of damaged DNA from malfunctioning cell mitosis.</td>
</tr>
<tr>
<td>Oxidative damage</td>
<td>Oxidative damage, especially to DNA and cellular components, can be attributed to the development of cancer. Even though reactive species such as ROS (reactive oxygen species) and RNS (reactive nitrogen species) are a natural by-product of normal biochemical and physiological reactions in the body, they can cause damage to the DNA, cellular membrane and cellular organelles, and can interfere with cellular regulators if there are not enough antioxidants to counteract the reactive species’ damaging effect.</td>
</tr>
<tr>
<td>Mind–body connection</td>
<td>Some feel there is a strong connection between the mind and the development of certain cancers, but this has not been confirmed. However, there is good evidence that mind–body medicine should be taken into consideration when addressing cancer. Some people have ‘spontaneously healed’ with a change of emotional and spiritual point of view.</td>
</tr>
</tbody>
</table>
| Polymorphisms                 | There are numerous studies on polymorphisms and genetic mutations increasing the risk of nearly all cancers. As this is a relatively new area of research, more studies will emerge on new polymorphisms that can
be linked with certain cancers, and testing procedures will become more accessible and less expensive.

**Epigenetics**

The increase of scientific knowledge on how chromatin organization modulates gene transcriptions has highlighted how epigenetic mechanisms are involved in the initiation and progression of human cancer. These epigenetic changes have been found to affect nearly every step in tumor progression, especially the aberrant promoter hypermethylation that is associated with gene silencing.

There are a few types of cancer that seem to have a strong link to specific dietary or lifestyle issues. In the following table I will list a few of these.

<table>
<thead>
<tr>
<th>FOOD OR BEVERAGE</th>
<th>IMPLICATED CANCER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>• Lung cancer</td>
</tr>
<tr>
<td></td>
<td>• Cervical cancer in people with HPV2</td>
</tr>
<tr>
<td></td>
<td>• Vulvar squamous cell carcinoma (SCC)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>• Total cancer risk</td>
</tr>
<tr>
<td></td>
<td>• Liver cancer</td>
</tr>
<tr>
<td></td>
<td>• Breast cancer—especially estrogen-dependent cancers</td>
</tr>
<tr>
<td></td>
<td>• Colon cancer</td>
</tr>
<tr>
<td>Low fruit and vegetable, fiber intake, high intake of processed meats, nitrosamines, highly salted foods</td>
<td>• Gastric cancer</td>
</tr>
<tr>
<td>Obesity</td>
<td>• Gastric cancer</td>
</tr>
<tr>
<td></td>
<td>• Breast cancer—link with leptin regulation</td>
</tr>
</tbody>
</table>

It should be noted that even if a person has all of these areas covered perfectly they could still end up with cancer. With such a multifactorial disease, people can only do the best they can to prevent its initiation and growth in their body. In fact there is suitable evidence that shows if a person spends too much time and energy concerned with preventing cancer, they can increase their odds of getting some forms of it.

A basic list of areas to consider in the prevention of cancer includes:
• diet—eat a balanced diet with lots of fresh fruit and vegetables (refer to the section on diet) and ensure adequate nutrition
• exercise
• maintenance of an optimal body weight
• decrease of negative stress
• adequate sleep
• having fun and enjoyment in life
• reducing alcohol intake, reducing or stopping smoking and illicit drug use
• having safe sex
• spirituality, religion or some form of belief
• moderation in everything

Chemical carcinogenesis

Chemical carcinogenesis has increasingly been recognized as an important cause of cancer, and can either cause cancer directly or after being metabolized by organs such as the liver. Polycyclic aromatic hydrocarbons are derived from the burning of fossil fuels and tobacco, and can have a wide range of activity in the body, producing cancer in the site of exposure (e.g. the lungs). Similarly, mineral oils and tar products contain benzopyrene and other hydrocarbons that have been shown to produce skin cancer when applied topically. Aflatoxins are natural compounds produced by some Aspergillus species, commonly found as a contaminant in food supply, especially in peanuts and grains that have been stored in warm, moist conditions. Aflatoxin is a potent agent in liver cancer. Aromatic amines and azo dyes found in aniline dyes have been shown to promote liver and bladder cancer, after metabolism by the liver. Nitrosamines are potent carcinogens used in food preservation. Metals such as nickel, lead, cadmium, cobalt and beryllium have electrophilic properties and thus can react with the organic constituents of the body, metal ions reacting with the guanine and phosphate groups of DNA. The inhalation of asbestos has been shown to promote pulmonary asbestosis and asbestos-associated lung cancers such as mesothelioma. This is by no means a complete list, but represents the most commonly identified chemical toxins. It is important to note that we are exposed to hundreds of
different potential chemical toxins, many of which are found in low concentrations in food. (Rubin 2001, 107-112)

There are several factors that can influence chemical carcinogenesis. For example, a woman that becomes pregnant at a younger age, has more children and lactates longer has the lowest risk of breast, uterine and cervical cancer of all groups. Conversely, a woman that has an early menarche, a late menopause and no pregnancy has the highest risk. (Rubin 2001, 107-112)

**Physical carcinogenesis**

Physical carcinogenesis for the most part related to sources of radiant energy. Non-ionizing *ultra violet* energy from the sun has been associated with basal cell carcinoma, squamous carcinoma and melanoma, and is predominant in white-skinned populations. *Ionizing* radiation, from sources such as x-rays, the therapeutic administration of radioactive isotopes, industrial mining and processing of radioactive substances, and from atomic explosion is a potent physical carcinogen. This radiation causes disjunctions in normal chromosomal structure, with the subsequent random fusing of broken ends leading to mutations in the genetic code. (Rubin 2001, 107-112)

**Viral carcinogenesis**

As mentioned earlier, viruses have been shown to be an important factor in cancer, responsible for an estimated 15% of all cancers. Many of these viruses have genes that encode for proteins, which interfere with the activities of tumor suppressor genes. Thus these cells become more likely to undergo neoplasia. Further, once infected, viral DNA is inserted in the host’s genetic structure, either replacing or activating human proto-oncogenes in such a way that they code for malignant transformation. Examples of DNA viruses include human papilloma virus, herpes, Epstein-Barr and hepatitis B2 virus. (Rubin 2001, 107-112)
Emotional Stress

Emotional blocks or longstanding emotional congestion can cause physical congestion in the corresponding tissue. This congestion reduces circulation, oxygen and communication with the rest of the body. It will also attract viruses, environmental toxins and even ion congestion to the area, further decreasing communication with the rest of the body. This creates a perfect environment for mutations and thus cancerous tumors to arise.

Part Two: Etiology and Pathology of Cancer

Systemic effects of cancer

Cancer patients can be seen to exhibit systemic manifestations of cancer, called paraneoplastic syndromes. Generalized manifestations include fever, anorexia and weight loss. In the case of malignant tumors within the endocrinal tissues, there may be an alteration in hormonal secretion that can have systemic effects, such as Cushing’s syndrome (tumor of the adrenal cortex), and hyperprolactinemia (tumor of the pituitary). Neurological symptoms of cancer may include dementia, cerebellar degeneration, encephalitis and optic neuritis. Upon laboratory investigation there may be erythrocytosis or anemia, granulocytosis, eosinophilia and thrombocytosis. (Rubin 2001, 117-119)

Cancer epidemiology

In North America cancer currently accounts for 25% of all causes of mortality, and is the leading cause of death, now surpassing cardiovascular disease. Some types of cancers appear to be on the decrease and others on the increase, depending upon geographic and ethnic differences.
• The rate of **nasopharyngeal cancer**, associated with EBV, is rare in most parts of the world, but significantly higher in parts of East Asia.

• **Esophageal cancer** is associated with several factors, including excess alcohol consumption and smoking, occurring in higher rates in Asia, in certain areas of Africa, and among African-Americans in the United States.

• Rates of **stomach cancer** have undergone a significant decrease in the West since the turn of the last century, but continue to be very high in Japan. It is thought that highly salted foods may promote stomach cancer.

• The incidence of **colorectal cancer** is highest in the United States, thought to be associated with low fiber intake.

• **Liver cancer** is endemic to sub-Saharan Africa and Asia, thought to be associated with aflatoxin-contaminated food.

• **Skin cancer** is noted as being highest among white-skinned people, found highest in Northern Australia and the American South.

• **Lung cancer** has undergone a dramatic rise all over the world, and is directly attributed to smoking, as well as the recent increase in environmental air-borne pollutants.

• **Breast cancer** is the most common female cancer in North America and Europe, with rates in developing countries one-fifth to one-sixth as high.

• **Cervical cancer** has been associated with a low socioeconomic status, as well as early sexual activity and multiple sexual partners, and is associated with venereal disease.

• The risk of **prostatic** and **testicular cancer** in the West has increased dramatically over the last few decades, with very low incidences reported in Asia, particularly in Japanese men. (Rubin 2001, 119-121)

**In Canada:**

• An estimated 177,800 new cases of cancer (excluding 74,100 non-melanoma skin cancers) and 75,000 deaths from cancer occurred in Canada in 2011.

• Of the newly diagnosed cases, about one-half will be
l lung, colorectal, prostate and breast cancers.

- Over one-quarter (27%) of all cancer deaths are attributed to lung cancer.
- Colorectal cancer has a significant impact on mortality for men and women combined, with an estimated 8,900 deaths (12% of all cancer deaths).

The number of people getting cancer has increased, due to an aging of the population over the last decade. But the mortality rate for cancers however has been in decline for a number of years, with the notable exception of thyroid cancer, which has been rapidly increasing in both men and women. (NCIC 17-18)

Among men, 75% of new cancer cases and 82% of deaths due to cancer occur among those who are at least 60 years old, whereas among women, 63% of new cases and 78% of cancer deaths occur among those who are at least 60 years old. The probability of developing and dying from cancer is 38% for women and 41% for men. The overall prevalence of cancer in the Canadian population is approximately 2% among men and 2.5% among women. Less than 1% of the female population are survivors of breast cancer and just over 0.5% of the male population are survivors of prostate cancer. In children, the most common cancer is leukemia, accounting for over 26% of new cases and 32% of deaths. Non-Hodgkin's Lymphoma incidence rates have almost doubled since 1974, and generally speaking, are highest in North America, Australia and New Zealand.
The following is a table of the estimated new cases of cancer and cancer-related deaths in Canada in 2011, the body sites affected, according to gender:

<table>
<thead>
<tr>
<th>Body Site</th>
<th>Estimated # of New Cases</th>
<th>Cases per 100,000</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Men</td>
</tr>
<tr>
<td>All Cancers</td>
<td>177,800</td>
<td>93,000</td>
</tr>
<tr>
<td>Lung</td>
<td>25,300</td>
<td>13,200</td>
</tr>
<tr>
<td>Breast</td>
<td>23,600</td>
<td>190</td>
</tr>
<tr>
<td>Prostate</td>
<td>25,500</td>
<td>25,500</td>
</tr>
<tr>
<td>Colorectal</td>
<td>22,200</td>
<td>12,500</td>
</tr>
<tr>
<td>Non-Hodgkins Lymphoma</td>
<td>7,700</td>
<td>4,200</td>
</tr>
<tr>
<td>Bladder</td>
<td>5,700</td>
<td>1,200</td>
</tr>
</tbody>
</table>

Lung cancer

Lung cancer is the second most common cancer diagnosed in North America, and the leading cause of cancer-related deaths in both the United States and Canada. The mortality of lung cancer accounts for 27% of all cancer related deaths in Canada.

Tumors of the lungs can be benign or malignant primary tumors, or metastases from primary cancers of other organs and tissues. Primary lung tumors include bronchogenic carcinoma (the most common type of lung cancer), bronchial carcinoid tumor, and a number of rarer forms.

Bronchogenic carcinoma

Bronchogenic carcinoma is a highly malignant primary lung tumor that accounts for more than 90% of all cases of lung cancer and has a very poor prognosis. It is the second most common cancer in both men and women, and is the leading cause of cancer death for both sexes. The etiology of bronchogenic carcinoma appears to be directly related to smoking, accounting for 87% of all lung cancers. A small proportion of lung cancers appear to be related to
occupational hazards, often concomitant with smoking, including exposure to asbestos, radiation, arsenic, chromates, nickel, chloromethyl ethers, mustard (poison war) gas, and coke oven emissions. The exact role of air pollution has yet to be defined, but probably plays a role. Genetic alterations in lung cells that accumulate and lead to neoplasm include activation of the oncogenes c-myc in small cell carcinomas and K-ras in adenocarcinomas. Studies in families have suggested a genetic predisposition, with polymorphisms in the cytochrome P450 gene, CYP1A1. Other genetic factors include the expression of vascular endothelial growth factor (VEGF) and proliferating cell nuclear antigen (PCNA), a loss of heterozygosity (i.e. normal gene activities) on chromosome 18q and chromosome 3p, and p53 mutations. (Rubin 2001, 339-342; Berkow 1992, 731-733)

Four histologic types of bronchogenic carcinoma are typically described:

1. **Squamous cell carcinoma** refers to a proliferation of malignant epithelial cells. The cytoplasm of cells is eosinophilic suggesting keratin production and there is squamous "pearl" formation. Cytologically the nuclei are pleomorphic (vary in size and shape) and hyperchromatic (increased chromatin, darker stained).

2. **Undifferentiated small ("oat") cell carcinoma** is composed of small round to oval cells with little cytoplasm and dark nuclei. The cells are slightly bigger than lymphocytes and are called oat cells. There may also be spindle-shaped forms. Nuclei are hyperchromatic and exhibit nuclear molding.

3. **Undifferentiated large cell carcinoma** is composed of large polygonal cells with no evidence of glandular or squamous differentiation.

4. **Adenocarcinoma** refers to the malignant proliferation of epithelial cells that produce glandular structures with central lumina some of which contain intraluminal mucinous-like material. (Rubin 2001, 339-342; Berkow 1992, 731-733)

Clinical manifestations depend on the tumor's location and type of spread. Most bronchogenic carcinomas are endobronchial and thus patients typically present with
cough, with or without hemoptysis. The sputum arising from an ulcerated bronchial tumor is usually not excessive, but contains inflammatory exudate and is often blood-streaked. Later symptoms include fatigue, weakness, decreased activity, worsening cough, dyspnea, decreased appetite, weight loss, and pain. Carcinomas of all types most frequently metastasize to the regional lymph nodes, in particular the hilar and mediastinal nodes. The most common site of extranodal metastasis is the adrenal gland. (Rubin 2001, 339-342; Berkow 1992, 731-733)

**Bronchial carcinoid tumors**

Bronchial carcinoid tumors are a group of neuroendocrine, pulmonary neoplasms derived from the pluripotent basal layer of the respiratory epithelium. Bronchial carcinoid tumors may be benign or malignant, occur equally in both sexes, and do not appear to be related to smoking. Bleeding from the overlying mucous membrane often occurs, and recurrent pneumonia within the same lung zone and localized overlying pleural pain are common. Metastases are uncommon but may occur to regional lymph nodes. (Rubin 2001, 342; Berkow 1992, 731-733)

**Breast cancer**

Along with lung cancer, breast cancer is one of the most common cancers in North America, and in the most recent Canadian statistics (2011), accounts for 23,600 cases of cancer. In North America, women have about a 13% chance of developing breast cancer in their lifetime (Boik 1996, 2). Within Canada, it is estimated that 22% of breast cancer cases occur in women under age 50, 48% occur in women aged 50 to 69, and 31% in women aged 70 and over. In Canada, breast cancer is the third leading cause of cancer-related deaths.

Breast cancer is classified according to the location of the cancer. **In situ carcinoma** it is contained entirely within the breast duct, with no invasion of adjacent normal tissues. Such cancer now accounts for more than 15% of all breast cancers diagnosed in North America. **Ductal carcinoma in**
situ (DCIS) accounts for 43% of breast cancers diagnosed in women aged 40 to 49 and 92% of cases diagnosed in women aged 30 to 39. **Lobular carcinoma in situ (LCIS), or lobular neoplasia**, occurs predominantly in premenopausal women and is usually found incidentally because it does not form a palpable mass. Microscopically, LCIS appears distinctly different from DCIS. Between 25% and 35% of patients with LCIS develop invasive breast cancer after a latency of up to 40 years. These invasive cancers occur with equal frequency bilaterally. Invasive ductal and lobular tumors are the most common histologic types of invasive cancer (about 90%). Patients with less common histologic types (e.g. medullary or tubular lesions) have a somewhat better prognosis. (Rubin 2001, 544-547; Berkow 1992, 1817-1818)

The status of hormone receptors and other tumor markers in breast cancer cells is used in conventional treatment and prognosis. Generally speaking, cancers in which the cells express the **estrogen receptor (ER)** in their nuclei will have a better prognosis, because the cells are generally better differentiated and can respond to hormonal manipulation (e.g. tamoxifen). Some 35% of breast cancers in women of childbearing age are estrogen-dependent. Symptoms are often dramatically decreased by the removal of the ovaries, which is the major source of estrogen. The significance of the **progesterone receptor (PR)** in breast cancer is less well understood, and is generally found in association with ER positive cancers. Breast cancer cells that are PR positive but not ER positive however, may have a worse prognosis. Other markers that are used to identify breast cancer include HER-2 and cathepsin D. HER-2 is a gene that helps control cellular growth, proliferation and repair. Cancers with too many copies of the HER-2 gene or too many HER-2 receptors tend to proliferate too quickly, and are associated with invasion and metastasis. Cathepsin D is an acidic lysosomal protease, synthesized under the influence of steroid hormones such as estrogen. Although its specific role is unclear, increased levels of cathepsin D is associated with an increase in mortality. (Witliff 2005; Rubin 2001, 547-48; Berkow 1992, 1817)
Risk factors

The risk factors for developing breast cancer include a family history of breast cancer, in which first-degree familial prevalence (parent, sibling, child) doubles to triples a woman's risk. When two or more first-degree relatives have had breast cancer the risk may be 5 to 6 times higher. About 5% of women with breast cancer carry one of the two breast cancer genes, $BRCA1$ or $BRCA2$. Women who do not have a family history of breast cancer in at least two first-degree relatives are not thought to carry this gene. (Rubin 2001, 542)

Other factors that contribute to breast cancer include an early menarche, late menopause, a late first pregnancy, nulliparity, a history of fibrocystic breast disease (FBD), and hormone replacement therapy (Rossouw et al 2002). The long term use of oral contraceptives is weakly connected to breast cancer risk. Regular alcohol use and cigarette smoking also appear to increase breast cancer risk. Previous radiotherapy significantly increases the risk of breast cancer, as does previous trauma to breast tissue (Rigby et al 2002). Obese women are at a higher risk as well, as are those who eat a diet low in fiber. The dietary consumption of saturated fat is often linked with an increase in risk, based upon experimental evidence, but this has not been demonstrated in the epidemiology nor in clinical trials (Brown et al 2002; Saadatian et al 2002).² Some studies have linked the consumption of red meat with incidence of breast cancer, but other studies have shown a preventative effect for meat consumption (Biesalski et al 2002). Some of the research exploring the link between red meat and breast cancer has focused on the production of carcinogenic heterocyclic aromatic amines (HAAs) found in cooked (blackened) meat. Nonetheless, other studies have demonstrated that HAAs do not increase risk (Delfino et al 2000). All in all, the risk of consuming meat appears to be negligible, and is probably not related to meat consumption per se, but additives (e.g. nitrosamines) and quality (i.e. conventional vs. organic).

² If there is a risk associated with saturated fat, such studies should take care to source the supply of saturated fat and do a comparison. Compounds such as xenoestrogens may very well bioaccumulate in animals fed hormones and non-organic feed in greater amounts, thus increasing the risk of such fats promoting cancer.
Diagnosis

Most tumors of the breast are discovered as a lump by the patient, although some may present with non-cyclic breast pain and generalized breast enlargement. Often the tumor will feel distinctly different from the surrounding breast tissue, but may present with diffuse fibrotic changes that feel firm and thickened. Advanced cases are characterized by a mass that is fixed to the chest wall or the skin, the presence of nodules or ulcers in the skin, or lymphedema. Further diagnostic tests include ultrasound, x-ray, fine-needle aspiration (FNA) and biopsy (Berkow 1992, 1817).

The most current recommendations suggest that routine screening should only involve the age groups at highest risk of developing breast cancer, between the ages of 50 and 70 years. Mammography is typically recommended as the primary method of early breast cancer detection, although there is significant controversy on this issue. Apart from the emotional trauma of false positive detection (up to 75% of lesions detected), and breast injury from unnecessary biopsy, there is concern over the risk of low dose X-rays used in mammography. *In vitro* models have demonstrated that low doses of low-energy X-rays produce an increased risk of oncogenic transformation by a factor of 2 (Brenner et al 2002). For women under 49 years self-breast examination is recommended. Ultrasound is another screening technique that poses little or no risk, and is especially helpful to distinguish a breast cyst from a solid mass. (Berkow 1992, 1812-14)

Breast cancer staging

To discern the severity of breast cancer, each case is usually graded according one of several systems of classification. The most common system of classification is numeric:

- **Stage 0**: Cancer in situ (limited to surface cells), with no evidence of invasion.
- **Stage 1**: Cancer limited to the tissue of origin, evidence of tumor growth. The tumor measures up to two
centimeters in diameter, with no lymph node involvement.

- **Stage II**: Limited local spread of cancerous cells. The tumor measures at least two centimeters, but not more than five centimeters. Cancer may also have spread to the axillary lymph nodes on the same side as the breast cancer.

- **Stage III**: Extensive local and regional spread. Usually divided into two sub-classifications:
  - stage IIIA: invasive breast cancer in which the tumor measures more than five centimeters, or the tumor has spread to lymph nodes, and nodes are clumping or sticking to one another or the surrounding tissue.
  - stage IIIB: invasive breast cancer in which a tumor of any size has spread to the skin of the breast, chest wall, or internal mammary lymph nodes. Stage IIIB includes inflammatory breast cancer.

- **Stage IV**: Distant metastasis, the tumor has spread beyond the breast, underarm, and internal mammary lymph nodes, including the supraclavicular lymph nodes, lungs, liver, bone, or brain. (Berkow 1992, 1271; Lahans 1998; Yance 1999, 318)

### Prostate cancer

**Prostate cancer** is the most common cause of cancer in Canada 25,500. Up to 53% of prostate cancer cases and 86% of deaths related to prostate cancer occur in men over the age of 70. Adenocarcinoma of the prostate accounts for 95% of all forms of prostate cancer, and is the second leading cause of cancer-related death in men more than 50 years of age; the incidence increases with each decade of life. Prostate cancer is often discovered during autopsies carried out following other causes of death, with a much greater incidence than cases of clinical cancer, upwards of 80% by the age 80. (Theodorescu 2005; Rubin 2001, 503-505)

Prostate cancer generally is slowly progressive and may cause no symptoms. It develops when the rates of cell division and cell death are no longer equal, leading to uncontrolled tumor growth. After this initial event
mutations of p53 and Rb may lead to tumor progression and metastasis. (Theodorescu 2005)

The prostate is a mostly glandular organ that can be divided into four basic zones: the peripheral zone, central zone, transitional zone, and fibromuscular zone. The peripheral zone forms about 70-75% of the glandular portion, located in the posterior portion of the prostate, its ducts opening into the distal prostatic urethra. Immediately anterior to the peripheral zone is the central zone, forming about 20-25% of the glandular prostate, its ducts opening mainly into the middle prostatic urethra. Anterior to the central zone is the transitional zone, only comprising about 5% of the remaining glandular prostate, consisting of two small lobes, the ducts opening close to the sphincteric part of the urethra. The remaining portion of the prostate consists of an aglandular portion called the fibromuscular zone, forming the entire anterior surface of the prostate as a thick apron of tissue, fusing with the prostate and hiding the other zones beneath it. (Theodorescu 2005; Rubin 2001, 503-505)

Whereas most cases of BPH relate to the hypertrophy of the central, transitional and fibromuscular zones, and do not affect the peripheral zone, 70% of diagnosed cases of prostate cancer affect the peripheral zone, followed by the central (15-20%) and transitional zones (15-20%). Adenocarcinomas of the prostate are however usually multifocal, involving multiple zones. (Theodorescu 2005; Rubin 2001, 503-505)

As the condition progresses, the most common clinical symptoms include urinary frequency (38%), decreased output (23%), urgency (10%), and hematuria (1.4%), but up to 47% of patients are asymptomatic. Metastases to the
bones including the pelvis, ribs, and vertebral bodies are common, and may cause bone pain. Locally advanced prostate cancer may exhibit extension of induration (hardening) to the seminal vesicles and fixation of the gland laterally. (Theodorescu 2005; Rubin 2001, 503-505)

**Risk factors**

The specific cause or causes of prostate cancer are unknown, but hormonal influences are thought to play an important role in the etiology of adenocarcinoma, primarily the activity of the androgens, although this is far from conclusive. Incidence rates of prostate cancer are high in northern Europe and North America, intermediate in southern Europe and Central and South America, and low in eastern Europe and Asia. This data in conjunction with migration studies suggest that environmental factors, such as diet, may play a significant promoting role in the development of a prostate cancer, but specific factors such as saturated fat and meat intake are inconclusive. Some studies have linked milk consumption to prostate cancer as estrogen levels in prostate fluid are correlated with prostate cancer (Qin et al 2004). Other data shows that phytoestrogens (e.g. soy) may be protective, and omega-6 fatty acids are positive stimulants of prostate cancer cell growth, while omega-3 fatty acids are negative stimuli. Deficiencies of vitamins A, D and E, as well as selenium are also thought to play a role. Overall, there are five basic risk factors thought to be involved in prostate cancer:

1. **Age:** prevalence increases with age. In Canada 53% of cases and 86% of deaths occur in men over 70. (NCIC 2003, 51-52)

2. **Race:** Incidence is highest in African-American men, although the disease is uncommon in African men living in Africa. This risk is similar for other races as well, as in Japanese men, who display a low rate of incidence in Japan, but experience a nine-fold increase if living in the West. (Theodorescu 2005; Rubin 2001, 503-505; Yance 1999, 340)

3. **Family history and genetics:** Epidemiological studies have shown that for men with a first degree relative with prostate cancer, there is a two-fold increase in risk. When both a first and second degree relative is affected,
the risk increases by a factor of nine. (Theodorescu 2005; Rubin 2001, 503-505; Yance 1999, 340)

4. **Reproduction:** Studies show a higher risk of prostate cancer in men who have undergone vasectomies. Other factors include frequent sexual activity, multiple sexual partners and a history of STDs (Yance 1999, 340). While an increase in prostate cancer risk from sexual activity is probably related to infection, other studies have shown that ejaculating five or more times per week has a protective effect, a factor perhaps more relevant in older men (Giles et al 2003).

5. **Dietary factors:** To date 33 case-control and cohort studies have examined the relationship between prostate cancer and dietary fat, eight of them suggesting a statistically significant association (Fleshner et al 2004). Overall the risk of consuming specific fats hasn’t been clearly identified except for the excessive consumption of animal fats or omega-6 fatty acids (Meyer et al 1999). More convincing evidence has demonstrated that the risk of prostate cancer decreases with the consumption of antioxidant-rich foods such as vegetables and fruits, and fiber-rich foods (Trichopoulou at al 2000; Etminan et al 2004; Hodge et al 2004).

**Diagnosis of prostate cancer**

**Prostate specific antigen (PSA),** a glycoprotein produced by the prostate gland, is the most commonly used marker for monitoring prostate cancer progression and response to therapy. Even though many authorities now consider it not that reliable to indicated prostate cancer, it is the best that has been found. Functionally, PSA acts to hydrolyze peptide bonds, thereby liquefying semen. It is concentrated in prostatic tissue, and serum PSA levels are normally low. If there is a disruption of the normal prostatic architecture, such as by prostatic disease, serum PSA levels may increase. Thus elevated PSA has been considered a marker for prostate cancer but is also present in other diseases of

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3 Many of these studies are performed by having the patient fill out a questionnaire after diagnosis, and then are statistically analyzed. Since a high intake of antioxidants such as vitamin C is associated with a decreased risk, the association between animal fats and prostate cancer could be a reflection of a decreased intake of such antioxidants concomitant with a “high fat” diet, but not a specific issue with animal fat per se.
the prostate including infection or benign prostatic hypertrophy (BPH). The levels of PSA can vary, and can be increased with ejaculation, digital rectal examination and prostate biopsy. The upper limit of normal PSA levels is 4 ng/mL, and any value above this may indicate a need for further testing (i.e., biopsy). Serum PSA levels are moderately elevated in 30-50% of patients with BPH, however, and not all patients with prostate cancer have elevated serum PSA. A recent development in PSA testing measures the ratio between bound and free PSA, assisting in discriminating between patients with a mildly elevated PSA from cancer and those with diseases such as BPH. In this test, the lower the ratio of free-to-total PSA, the higher the likelihood of cancer. (Theodorescu 2005; Rubin 2001, 503-505; Yance 1999, 341-42)

Besides PSA testing, the other most commonly used method of assessment is digital rectal examination (DGE). Normally the prostate feels smooth, regular and rubbery, but in cancer the prostate very often feels enlarged, hard, irregular and nodular. (Theodorescu 2005; Rubin 2001, 503-505; Yance 1999, 341-42)

The severity of prostate cancer is most commonly classified according to TNM staging: ‘T’ representing ‘tumor’ size, ‘N’ representing lymph node involvement, and ‘M’ denoting ‘metastasis.’ The system is as follows:

**T Staging**
- **T1**: indicates the cancer cannot be seen or felt, but has been confirmed by biopsy.
  - **T1a**: indicates the cancer is present in 5 percent or less of the prostate tissue that has been examined.
  - **T1b**: indicates the cancer is present in more than 5 percent of the prostate tissue.
- **T2**: indicates the cancer is confined to the prostate.
  - **T2a**: indicates the cancer is on one side of the prostate.
  - **T2b**: indicates the cancer is on both sides.
- **T3**: indicates that the cancer has spread beyond the prostate to nearby tissues and organs.
  - **T3a**: indicates the cancer is not found in the seminal glands (which contribute toward the production of semen).
- **T3b**: indicates the cancer has spread to the seminal glands.
- **T4**: indicates the tumor has spread to other organs, such as the bladder or rectum.

**N staging**
- **N0**: indicates the cancer has not spread to the lymph nodes.
- **N1**: indicates the cancer has been found in the lymph nodes.

**M staging**
- **M0**: indicates the cancer has not spread to distant tissues.
- **M1**: indicates the cancer has been found in distant tissues.
  - **M1a**: indicates the cancer has been found in lymph nodes beyond the pelvic area.
  - **M1b**: indicates the cancer has been found in the bone.
  - **M1c**: indicates the cancer has been found in other sites. (Theodorescu 2005; Rubin 2001, 503-505; Yance 1999, 343-44)

**Colorectal cancer**

**Colorectal cancer** is the second most common cancer in the United States, and the fourth most common cancer in Canada, just behind breast cancer. It is a disease that is associated with a greater degree of mortality than prostate cancer, and within Canada, accounts for 12.3% of all cancer-related deaths, second only to lung cancer. Most forms of colorectal cancer are adenocarcinomas. (NCIC 2003, 19; Rubin 2001, 388-89)

**Risk factors**

Colorectal cancer is thought to begin with the formation of **benign polyps**, which may increase in size and become cancerous. Among the factors that are associated with colorectal cancer are age, a history of certain bowel diseases, diet and obesity:
1. **Age**: the incidence of colorectal cancer increases with age, in Canada, beginning in the fifth decade and increasing to its highest incidence during the seventh decade (NCIC 2003, 51-25)

2. **Bowel disease**: patients suffering from ulcerative colitis for 25 years or more have a 12% increase in the risk of developing colorectal cancer (Rubin 2001, 389)

3. **Familial adenomatous polyposis (FAP)**: FAP is a group of rare inherited disorders of the gastrointestinal tract, characterized by benign adenomatous polyps in the mucosa. FAP is associated with a mutated *APC* (adenomatous polyposis coli) gene, which codes for a protein that targets the degradation of beta-catenin, a protein that is a component of a transcriptional complex that activates growth-promoting oncogenes such as *cyclin D1* and *c- myc* (Rubin 2003, 389; Batchelder 1998)

4. **Diet**: Generally accepted dietary risk factors for developing colorectal cancer include alcohol consumption, a low fiber intake, and a folate deficiency (associated with mutations of the *Ki-ras* oncogene, which are found commonly in colorectal cancer). A high fat diet has also been implicated as a risk factor for colorectal cancer according to animal models, but epidemiological and human trials suggest that this link is weak (Rubin 2001, 389; Batchelder 1998; Zock 2001).

5. **Obesity, insulin resistance and diabetes mellitus**: Several studies have demonstrated a link between obesity, a high caloric intake and low physical activity as important risk factors for colorectal cancer (Mao et al 2003; Giacosa et al 1999). Studies have demonstrated a link between high circulating levels of insulin and IGF, and colonic neoplasia (Giovannucci 2001; Kim 1998).

Besides the risk factors mentioned above, researchers have identified a number of important genetic alterations that contribute to the eventual development of colorectal cancer:

- an imbalance in DNA methylation, with general hypomethylation leading to oncogene activation, and focal regions of hypermethylation that silence the tumor suppressor gene expression
• ras gene mutations, which play a role in the growth of polyps and have been detected in the stool of patients with colorectal cancer
• deletions within the DNA, involving DPC4 (involved in the TGFß growth-inhibitory signaling pathway) and DCC (a gene frequently deleted in colon cancer)
• tumor suppressor p53 mutations
• Bcl2 over-expression leading to inhibition of cell death signaling has been observed as a relatively early event in colorectal cancer development.
• mutations in one of several genes involved in DNA repair, including MSH2, MLH1, and PMS2 (Rubin 2003, 388; Batchelder 1998)

**Diagnosis of colorectal cancer**

Colon cancer often is found by routine screening and is often asymptomatic. About 50% of patients present with abdominal pain, 35% with altered bowel habits, 30% with occult bleeding, and 15% with intestinal obstruction. Tumors on the right side tend to be larger and more likely to bleed, whereas tumors on the left tend to be smaller and are more likely to obstruct bowel functions. (Rubin 2001, 389-90; Batchelder 1998; Berkow 1992, 853)

The clinical features of colorectal cancer include occult intestinal bleeding or bright red blood, depending on whether the tumor is distal or proximal to the rectum. On the right side of the colon, particularly in the cecum, the tumors can grow to a large size without promoting symptoms of obstruction. Among the first indications of this form of colorectal cancer is severe anemia. For tumors that develop on the left side of the colon, which is smaller in diameter than the right side, there are typically symptoms of obstruction, with a change in bowel habits, flatulence, and abdominal pain. (Rubin 2001, 389-90; Batchelder 1998; Berkow 1992, 853)

The prognosis of colorectal cancer is reflective of the extent to which the tumor extends through the wall of the colon. They are classified accordingly:

• **Stage A**: tumor confined to the mucosa
• **Stage B₁**: tumor invading into the muscularis propria
• **Stage B₂**: tumor invading into the serosa, without lymph node involvement
• **Stage C₁**: B₁ tumors with metastases to local lymph nodes
• **Stage C₂**: B₂ tumors with metastases to local lymph nodes
• **Stage D**: distant metastases (Rubin 2001, 390)

**COMMON TUMOUR MARKERS**

Some cancer release certain molecules into the blood or tissue; these markers can be measured or identified and give a ‘rough guess’ to the probability of a cancer, or even the success of the treatment.

**PSA**: Prostate-Specific Antigen (PSA) Prostate-specific antigen (PSA) (normal 0–4 ng/mL). PSA is a glycoprotein that is prostate-specific, **NOT** cancer-specific. A variety of conditions can raise PSA levels: prostatitis (prostate inflammation), benign prostatic hypertrophy (prostate enlargement) and prostate cancer. PSA levels tend to increase with age. Some prostate glands normally produce more PSA than others and the levels can vary with race: African Americans often have higher PSA levels; Asian men often have lower PSA levels. However, PSA is sensitive for the presence of prostate cancer as well as benign prostatic hypertrophy. Many practitioners (due to several studies) feel that PSA is not a valid measure of prostate cancer.

**CEA**: Carcinoembryonic antigen (CEA) is a protein found in many types of cells that is associated with tumors and the developing fetus. It is found in the plasma membrane of tumor cells. Elevated levels are detected in a variety of cancers including colon, pancreatic, gastric, lung, ovary and breast cancer. It is also detected in benign conditions including cirrhosis, inflammatory bowel disease, chronic lung disease and pancreatitis. The CEA was found to be elevated in up to 19% of smokers and in 3% of a healthy control population. CEA may have some prognostic value for colon cancer, and values have been correlated with staging of the cancer. The normal range for CEA in an adult non-smoker is less than 2.5 ng/mL and for a smoker less than 5.0 ng/mL.
CA 19-9: is a monoclonal antibody generated against a colon carcinoma cell line to detect a monosialoganglioside found in patients with gastrointestinal adenocarcinoma. It is found to be elevated in 21–42% of cases of gastric cancer, 20–40% of colon cancer, and 71–93% of pancreatic cancer. Normal range is less than 40 U/mL.

CA15-3: An elevated CA15-3 in conjunction with high alkaline phosphatase has been shown to predict the chances of early recurrence in breast cancer. Normal values can vary but should be less than 25 U/mL.

CA 125: is an antigen present in 80% of ovarian carcinomas, its level corresponding with the patient’s clinical condition. For ovarian tumors it is more accurate than CEA. It is also elevated in other cancers including endometrial, pancreatic, lung, breast and colon cancer, and in menstruation, pregnancy, endometriosis and other gynecological and non-gynecological conditions. Normal value varies between laboratories but is generally less than 35 U/mL.

BRCA1 and BRCA 2: human tumor suppressor genes which produce proteins, called breast cancer type 1 susceptibility protein and breast cancer type 2 susceptibility protein. They are found in the cells of the breast and other tissues, where they help repair damaged DNA and destroy the cell when DNA cannot be repaired. If BRCA1 or 2 are damaged, the damaged DNA can let the cell duplicate without control, and turn into cancer. The position of a mutation in the BRCA1 or BRCA2 gene can reflect the relative incidences of breast and ovarian cancer within a family. The BRCA2 mutation carries an increased risk for cancers of the prostate, pancreas, gallbladder/bile duct and stomach, as well as for malignant melanoma. The BRCA2 mutation also appears to confer higher risks for male breast cancer. BRCA1 mutation confers a higher incidence of ovarian cancer than does BRCA2 mutation. BRCA1 and BRCA2 proteins were also found to collaborate with one another on a common pathway of tumour suppression, through the S phase of the cell cycle. Both BRCA 1 and BRCA2 are sensitive to oxidative damage and particularly to radiation damage. Young women exposed to radiation particularly in the stage of breast development have a specifically increased risk of subsequent breast cancer as adults.
**AFP:** Alphafetoprotein (AFP) is a normal fetal serum glycoprotein synthesized by the liver, yolk sac and gastrointestinal tract that shares fetal origins with albumin. AFP is of importance in the diagnosis of hepatocellular carcinoma. It can also be elevated in normal pregnancy and benign liver disease such as hepatitis and cirrhosis. AFP can also be elevated in malignant pancreatic cancers, gastric cancers, colonic cancers and bronchogenic cancers not necessarily associated with liver metastases. Normal range is less than 10 µg/L.

**HCG:** human chorionic gonadotropin, normally elevated in pregnancy, can also be found in gestational trophoblastic disease and germ cell tumors as well breast, lung and GIT tumors.

**Tumor Staging**

Staging is a description of the extent or severity of an individual’s cancer based on the extent of the original (primary) tumor and the extent of spread in the body.

<table>
<thead>
<tr>
<th>Tumor Staging</th>
<th>Definition</th>
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<tbody>
<tr>
<td><strong>Stage 0</strong></td>
<td>Carcinoma in situ (early cancer that is only in the layer of cells in which it began)</td>
</tr>
<tr>
<td><strong>Stage I, stage II, and stage III</strong></td>
<td>Higher numbers indicate more extensive disease: greater tumor size, and/or spread of the cancer to nearby lymph nodes and/or organs adjacent to the primary tumor</td>
</tr>
<tr>
<td><strong>Stage IV</strong></td>
<td>The cancer has spread to another organ</td>
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**Cancer is like a terrorist cell**

One can easily become overwhelmed when faced with all the data, facts and theories on cancer. For our purposes we can create a simple analogy to look at basic aspects and probable treatment modalities.
Basically cancer is an issue of communication. More specifically, it is a lack of being part of the whole organism; thus convoluting ones energy inwards without proper communication with the larger organism; or in other words isolating and forming a tumor.

**Evolution of a Terrorist Group**

If a person has a strong point of view, they usually want to communicate it to someone. If others around them don’t listen, marginalize them or shut them out, the person may begin to keep their thoughts to themselves. They may drop the issue, bury it in their psyche or even ferment on it. They may begin to feel bad or resentful about it, thus creating a small irritation in the back of their mind.

If they are given a chance to share their views with others, through dialogue their feelings may change and become less of an issue. Often finding some comrades with sympathetic ears is all that is needed. They start a small group, communicating about these and maybe other issues, and it doesn’t influence anyone outside their group. These would be the benign tumors.

But sometimes an idea will grow and fester in a person. In some incidences these ideas might grow into a plan of action on how to make other people see, or believe their ideas as being important. This could be as simple as bullying another, or as dramatic as a Columbine-like incident where a group of teenagers went into the school and started shooting people. This type of behavior is like a cell of activity that starts to affect the health of other local cells (Malignant tumors). In these kinds of incidences, the “authorities” (T-cells, NK cells and macrophages) come in to deal with it, shutting the whole operation down. Regardless of whether the cause is/was good or bad, it was affecting the status quo of the surrounding environment, and thus an order was given to “deal with it”.

Sometimes these ideas get bigger and bigger, influencing a large group of people. (Oncogene over-expressive & tumor suppressor gene under-expressive) This could be as simple as a coup attempt to take over an organization or even a government. This process can be called evolution. Sometimes evolution starts with revolution. If all goes
smoothly revolution leads to evolution and a new way (hopefully a better way) of dealing with things. Other times the battle evoked by the new ‘resistance group’ causes bigger problems and the resistance group is considered a terrorist group by the larger organism.

If the terrorist group is small enough and isolated enough, it will be ‘neutralized’ and the problem resolved. (Our body is continuously dealing with ‘terrorist cells’ and for the most part can neutralize them.) If the terrorist cell goes unnoticed for long enough it can create a stronger center of gravity and a strong sense of personal authority making it harder to neutralize (TGF-β helps keep cell undetected). It can also diversify and create more independent or semi-independent ‘terrorist cells’ so if one ‘terrorist cell’ is neutralized, it will not destroy the whole ‘movement’ (Initiation and promotion stages).

Once the movement gets to a certain stage, fighting becomes almost impossible. Propaganda and the war machine further confuse communication, deepening the original problem. Creating an independent Palestine is more sensible than going in with a war machine and trying to level all the various sites of conflict. Fighting in a situation like Afghanistan, Iraq or Iran is a war that can never be won, as it is philosophical. You can’t win by simply killing or capturing a leader. Often ‘blindly’ going in and exerting force can cause the terrorist cells to splinter, go underground and create sympathy groups in the area (metastasis). This is what seems to have happened in much of the Middle East. The actual act of war harms more innocent people than terrorists or soldiers and often leads by-standers to side with the terrorist by providing hiding places, food and financial support (produces angiogenesis). The terrorist groups are ‘selfish’ in that they rely on their own survival skills to overtake the ‘outside’ collective.

Surgical removal often isn’t complete and can cause the spread of cancerous cells. Chemo therapy and especially radiation therapy can be successful by killing the cancer, but sacrifices the patient in the process by destroying or mutating enough healthy cells to compromise the person.
Part Three: Medical Treatment of Cancer

The treatment of cancer in modern medicine rests on only these primary modes: chemotherapy, radiation and surgery, hormonal manipulation, and immunotherapy. New drugs that target specific genes are still in the experimental stage. In some cases one therapy may be used as the primary therapy, and others will be added as adjuvants to assist in the overall efficacy of promoting tumor death. The choice of treatment depends upon a variety of factors including the type and location of the tumor, its sensitivity to certain agents, whether or not it has metastasized, the data that indicates the efficacy of the therapy, as well as the age and overall health of the patient.

The primary therapy in modern oncology is to kill cancer cells (wage war), and success is measured against the degree of tumor regression and the impact of the therapy on the patient. Somewhat paradoxically, many of the therapies used to kill cancer cells also kill or injure healthy cells, and can in turn promote cancer. Natural medicine can have an important role to play in such scenarios, to both protect the body against the health-damaging effects of conventional treatment, and to treat common side effects including nausea, cachexia, immune suppression and pain. Although many oncologists are antagonistic to using natural therapies in conjunction with conventional treatments, there are many studies that show benefit in using them together. For example, in Japan and other oriental countries extracts of fungi including PSK from Coriolus or Shiitake, Maitake, Ganoderma or Cordyceps are used to reduce the side effects of radiation and/or chemotherapy. The Amala Cancer Institute in India and other researchers have shown that the Indian herb Ashwagandha (*Withania somnifera*) has significant antitumor and radiosensitizing effects in experimental animals, inhibiting tumor growth, reducing leucopenia induced by cyclophosphamide, and increasing survival time (Devi 1996; Devi et al 1995; Kuttan 1996).

**Biopsies**
While biopsies are considered important for accurate medical diagnosis, there is evidence to suggest that biopsies are safer if taken after the tumor is removed, particularly in solid tumors (excisional biopsies), rather than breaking the tumor capsule with the needle and risking spread. Often the cancer is spread through the biopsy process. If a person wants to get a biopsy, we strongly suggest they take a medicinal mushroom blend with Coriolus in it for at least 1 – 4 weeks before biopsy if possible. Other important supplements would be high-dose vitamin C and Beta Carotene. Diet becomes very important during this time. Several of the foods and supplements suggested here would also be very good to do before biopsy.

**Surgery**

Surgery is perhaps the oldest method for treating cancer, particularly in solid tumors that haven’t metastasized. In some cases such measures are thought to be curative, such as the removal of a cancerous polyp in colorectal cancer. **Radical surgical** treatments involve the removal of adjacent tissues if the surgeon suspects the tumor has spread, such as removing a tumor and the associated lymph nodes. **Palliative surgery** involves procedures that remove a tumor to relieve pressure or pain, or the removal of a glandular tumor that secretes hormones to excess (e.g. adrenals, ovaries, testes), and is causing problems with metabolic activities. Other surgical techniques include **electrosurgery, cryosurgery** and **laser surgery**. (Yance 1999, 275; Berkow 1992, 1275-76)

Removing the bulk of the tumor can assist the immune system to recover. However, this is only symptomatic treatment and is not dealing with the cause of the problem. Protect the person from the side effects of the surgical procedure by giving vitamin C to allay the side effects of anesthesia (but check blood clotting to ensure safety), Arnica 30x (or Traumeel) for bruising and surgical shock, zinc and vitamin E to help with healing and to reduce scarring. Often leakage occurs during surgery, aiding in the spread of the cancer. This is particularly true of exploratory surgery or biopsies where the seal or cyst around a tumor system is destroyed. It is strongly suggested to follow some
of the ideas listed under biopsies, to assure lower spread rates of the cancer.

Of course removing the tumor doesn’t deal with the source of the cancer in the first place. May it be a chemical irritant or blocked emotion (or combination), these details have to be dealt with before the person can be said to be cancer free.

Radiation therapy

Radiation therapy or radiotherapy is a local treatment that uses a high energy photonic beam or radioactive implants to promote tumor death (burn). The most common form of radiotherapy is x-rays, beamed onto the body in varying degrees of intensity to destroy tumors of varying depths. Gamma-rays are another form of photons used in radiotherapy, produced by elements such as radium, uranium, and cobalt 60 as they decay. Another technique is called internal radiotherapy, which delivers radiation to cancer cells by placing radioactive implants (often beads) directly into a tumor or body cavity, or given by injection.

Radiotherapy is used in a variety of ways, both as a primary treatment and adjuvant. As a primary treatment, radiation is used to treat localized tumors such as cancers of the skin, tongue, larynx, brain, breast, or uterine cervix, as well as leukemia and lymphoma. Radiation is used as an adjunct to chemotherapy or surgery to eliminate cancer cells that were once present in a specific location, such as colorectal cancer, although its benefit has been questioned. Radiation is also used as a palliative remedy, to shrink painful tumors that are metastasizing (e.g. bone metastasis) or where tumors are pressing on sensitive tissues (e.g. spinal cord). A relatively recent development in radiotherapy is the use of radiolabeled antibodies injected into the bloodstream to deliver doses of radiation directly to the cancer site, called radioimmunotherapy. These antibodies recognize and bind to specific antigens on tumor cells, which are then destroyed by the radiation. (Yance 1999, 275-76; Berkow 1992, 1276-77)
Radiation destroys tumor cells by damaging their DNA, inducing the generation of free-radicals and genetic mutations, interfering with growth and proliferation. Radiation also damages normal cells, although the latter are thought to be able to repair themselves. Unfortunately the surviving normal cells may be unable to repair this damage, and can lead to secondary cancers, such as leukemia. (Yance 1999, 275-76; Berkow 1992, 1276-77)

Radiotherapy has a number of adverse-effects, besides an increase in cancer risk, depending on the body part treated. These include organ damage, immunodeficiency, nerve damage, skin reactions (dryness, itchiness, rashes, redness, irritation, blistering, cracking), hair loss, permanent hoarseness, dry mouth, sore throat, dysphagia, nausea or vomiting, diarrhea or constipation, abdominal pain, fatigue and weakness, impotence and infertility. (Yance 1999, 275-76; Berkow 1992, 1276-77)

Basically, with both radiation therapy (burn) and chemotherapy (poison), the gamble is that the person’s vital center of gravity (strength) is stronger than both the tumor system and the radiation/chemotherapy. It is often the job of the herbalist to assure this by the use of certain remedies and constant monitoring of the vitality levels.

**Chemotherapy**

**Chemotherapy** refers to the use of toxic drugs to kill tumor cells (poison), usually given intravenously, but also given by injection, orally or applied topically. Chemotherapeutic drugs inhibit or interfere with the growth and development of tumor cells, and function in a variety of ways, depending on the class of drug. Chemotherapy is most often used in fast-growing tumors, which are more susceptible to its effects, including leukemia and lymphoma, as well as solid tumors that have metastasized. The dose given in most cases is the highest tolerated dose, and because tumor cells are resistant or become resistant, chemotherapy is usually given in combination. The Cochrane database ([http://www.cochrane.org/](http://www.cochrane.org/)) is one of the easiest and best sources of information on various
Chemotherapies and radiation therapies. The most common chemotherapeutic drugs include:

- **Alkylating agents:** Alkylating agents are non-specific agents that act on the tumor cell at any point during the cell cycle. They act directly on the DNA, causing the cross-linking of DNA strands, abnormal base pairing, or breaks in DNA strands, thus preventing cell division. Although alkylating agents may be used for most types of cancer, they are generally used for slow-growing cancers. Examples of alkylating agents include chlorambucil, cyclophosphamide, thiotepa, and busulfan. Alkylating agents are often used in ovarian, breast and lung cancer, as well as leukemia and lymphoma. (Yance 1999, 276-77; Berkow 1992, 1278)

- **Antimetabolites:** Antimetabolites cause tumor death by supplanting inert substances in place of building blocks in DNA molecules, thereby altering the function of enzymes required for cell metabolism and protein synthesis. The net result is the formation of defective DNA, which breaks the cycle of proliferation. Antimetabolites are most effective during the S-phase of cell division (DNA and centrosome replication), because they act upon cells undergoing synthesis of new DNA for formation of new cells. Examples of antimetabolites include purine antagonists, pyrimidine antagonists, and folate antagonists. Antimetabolites are often used in leukemia, ovarian, breast, colorectal and pancreatic cancers. (Yance 1999, 276-78; Berkow 1992, 1278)

- **Plant alkaloids:** Plant alkaloids are derived from plants, acting specifically to block the ability of a cancer cell to undergo mitosis. Although they act throughout the cell cycle, some are more effective during the S-phase (replication of DNA and centrosomes) and M-phase (mitotic phase, i.e. prophase, metaphase, anaphase, telophase). Examples of plant alkaloids used in chemotherapy are vinblastine, vincristine, actinomycin D, doxorubicin, and mitomycin. Plant alkaloids are used in Hodgkin’s disease, choriocarcinoma, neuroblastoma, lymphocytic
leukemia, and in breast, lung and testicular cancers. (Yance 1999, 276-78; Berkow 1992, 1279)

- **Tumoricidal antibiotics:** Antitumor antibiotics are nonspecific cell cycle inhibitors derived from substances that are produced by soil fungi, which act by binding with DNA, essentially causing it to uncoil, thereby preventing RNA and protein synthesis. Examples include doxorubicin (adriamycin), mitoxantrone, and bleomycin. Tumoricidal antibiotics are used in Hodgkin’s disease, and cervical, breast, testicular, bladder and thyroid cancers. (Yance 1999, 276-78; Berkow 1992, 1279)

Chemotherapeutic drugs are highly toxic compounds that not only destroy cancer cells but also affect normal cells. Thus they are responsible for many adverse effects, including nausea and vomiting, loss of appetite, mouth ulcers, weakness and fatigue, hair loss, menstrual alterations, immunosuppression, anemia, heart problems, kidney and/or liver toxicity, and even cancer. Most side effects are temporary but some can be permanent. Of note is the drug doxorubicin (adriamycin), which can damage the heart. (Yance 1999, 276-78; Berkow 1992, 1278)

<table>
<thead>
<tr>
<th>SIDE EFFECTS</th>
<th>CAM THERAPY</th>
</tr>
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<tbody>
<tr>
<td>Mouth Ulcers</td>
<td>Calcium phosphate, honey, zinc, slippery elm</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Berberini, Glutamine</td>
</tr>
<tr>
<td>Constipation</td>
<td>LBT-3</td>
</tr>
<tr>
<td>Intestinal permeability</td>
<td>Berberini, Glutamate, probiotics</td>
</tr>
<tr>
<td>Radiation enteritis or enteropathy</td>
<td>Medicinal Mushrooms, Hyperbaric oxygen chambers, probiotics during radiation, glutamine during radiation</td>
</tr>
<tr>
<td>Neutrophilia</td>
<td>Vitamin E, medicinal mushrooms</td>
</tr>
<tr>
<td>Anemia</td>
<td>Iron</td>
</tr>
<tr>
<td>Fatigue</td>
<td>L-carnitine, coenzyme Q₁₀, Medicinal Mushrooms</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>Alpha-Lipoic acid, Medicinal Mushrooms</td>
</tr>
<tr>
<td>Memory loss</td>
<td>Egg phosphocholine and low-dose vitamin B₁₂, Medicinal Mushrooms</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Omega-3 fatty acids</td>
</tr>
<tr>
<td>Weight gain</td>
<td>Dietary suggestions to keep BMI in range</td>
</tr>
</tbody>
</table>
Hormone therapies

Hormone therapies are used in cancers that are thought to be hormone dependent including breast and prostate cancer. In such cases, one method of intervention is to simply remove the ovaries and testes, which produce hormones that promote glandular proliferation in these tissues. On a similar basis, synthetic hormones and hormone-blocking agents that have opposing effects upon a glandular tumor may also be given. (Yance 1999, 281-82; Berkow 1992, 1277)

In breast cancer, androgens such as dehydroepiandrosterone (DHEA) may be given, or drugs such as tamoxifen. Tamoxifen binds to specific estrogen receptors, thereby inhibiting cellular growth and proliferation in estrogen-dependent cells in the breast. Tamoxifen can also promote estrogen like effects however, and is thus considered for usage in other estrogen-deficient conditions, including osteoporosis, menopausal symptoms and hyper-cholesterolemia. Estrogen-dependent tumors with a mutated estrogen receptor gene may actually be stimulated with tamoxifen, thus defeating the purpose of its usage. (Yance 1999, 281-82; Berkow 1992, 1277, 1820)

In prostate cancer an example of hormone therapy is the potent estrogen DES (diethylstilbesterol), which is thought to inhibit prostate growth, given along with flutamide, a non-steroidal antiandrogen. (Yance 1999, 281-82; Berkow 1992, 1277, 1751)

Hormonal therapies can have dramatic effects, both to reduce tumor growth, as well as to significantly alter secondary sexual characteristics. Thus in women, hormone
therapy in cancer could promote male secondary sexual characteristics, such as deepening of the voice, increased libido, growth of body hair, menopausal symptoms in premenopausal women, and bleeding in postmenopausal women. In men, hormone therapy may cause a raising of the pitch of the voice, decreased sexual desire, impotence and gynecomastia. (Yance 1999, 281-82; Berkow 1992, 1277)

Immunotherapy

**Immunotherapy, biological therapy or cell-transfer therapy**, are all terms used to describe the use of medications to artificially mimic specific immune substances called **biological response modifiers (BRMs)**. BRMs are natural substances found in the body that form part of the mechanism of non-specific resistance and immunity. In immunotherapy, BRMs are synthesized in the laboratory and injected into the body, often combined with chemotherapy drugs. Synthetic BRMs include various cytokines such as interferon and interleukin-II, and monoclonal antibodies, the latter of which is still in the experimental stage. Tumor vaccines are an experimental drug prepared from tumor cells, used to stimulate an immune response against the tumor similar to conventional vaccination, but can be made up so that each vaccine is unique to an individual’s particular tumor. Another experimental drug in immunotherapy is bacillus Calmette-Guerin (BCG), injected into the body to provoke an immune response. (Yance 1999, 282-83; Berkow 1992, 1291)

Immunotherapy is generally not observed to be all that effective against cancer, and is usually used in combination with conventional chemotherapy. A number of temporary side effects are associated with immunotherapy including loss of appetite, nausea, vomiting, diarrhea, chills, fever, muscle aches, weakness, rashes, easy bruising, immune suppression and hemorrhage. (Yance 1999, 282-83; Berkow 1992, 1291)
Gene therapy

Given the relative toxicity of conventional cancer treatments, their propensity to cause adverse effects, and even cancer, researchers are scrambling to find new drugs that target specific mutant genes. Many of these therapies use novel techniques, such as genetically modifying a virus (such as the common cold virus ONYX-015) to infect a tumor cell and make changes to the mutated DNA, such as the insertion of tumor suppressor genes. It is thought that the insertion of a gene that encodes for a tumor suppressor protein such as p53 may be able to restore the ability of the cells to keep the tumor in check. The p53 protein is responsible for arresting cell cycle following DNA damage via upregulation of p21, coding for an inhibitor of cyclin-dependent kinases, upregulation of bax, down-regulation of bcl-2, and inducement of apoptosis. Research has shown that the delivery and expression of the wild-type p53 gene can cause regression in established human tumors, prevent the growth of human cancer cells in culture, or render malignant cells from human biopsies non-tumorigenic. In most recent studies gene therapies are used in combination with conventional treatments, and in some cases, have had a small but statistically significant benefit. (McCormick 2003)

Part Four: Holistic Treatment of Cancer

Although the overall prevalence of cancer probably increased with the advent of the industrial revolution, it can be seen from a survey of the traditional literature that cancer has long been with us. Thus holistic medicine has much to offer conventional medicine from a variety of perspectives, some of which inform modern practice, such as the use of plant alkaloids like vincristine, antioxidants and immunomodulants such as beta-carotene, Ganoderma, Chaga and Coriolus and the use of mind-body therapies such as meditation and prayer.

Perhaps the most important perspective that holistic medicine can convey is that cancer is as much a spiritual
disease as it is a physical one. There are many holistic traditions that express the concept of wholeness and vitality. The Ayurvedic tradition has a clear concept of this, reflecting other traditions. In Ayurvedic medicine the cohesiveness of the body is maintained by self-identity (ahamkara), a psychospiritual principle and emanation of the divine order (mahat) that provides each of us and every cell with a self-identity. Indeed, each person’s concept of self is no different from others: we all perceive a self-identity, and it is this self-same identity that is operant in every biological system, only the conditions are different.

It is remarkable, when examined from a strictly biological perspective, how the eukaryotic cell evolved, and how some organelles within each and every cell are semi-autonomous, such as the mitochondria, that despite the fact that they have their own DNA, function as a collective to form the normal human cell. What is it that told these primitive beings to evolve in a collective vision? The myriad nature of our biology and all its various components held within this collective vision is what is called ahamkara. From the highest perspective ahamkara is the self-identity of the Self, or atma, the ‘great soul’ of the universe that exists in all dimensions and planes of existence. Although Indian spiritual philosophy suggests that it is this ahamkara (ego) that serves as an obstruction to deeper spiritual realizations, in reality the problem is only confusion between what is Self and what is condition. Self exists as a timeless essence: spiritual ignorance is when we confuse this Self for our life condition, so that we believe that we are this conditioned existence that is born, experiences pleasure and pain, and then fades away with death. According to Vedanta, the most esoteric and highly evolved component in Indian spirituality, true wisdom (jnana) comes when we see that these factors are superimposed illusions (maya) that mask the face of the ‘great soul’ that is each and every thing.

The nature of ahamkara (self) provides for proper self-regulation, and is easily affected by life conditions. In particular, negative emotions and thoughts are thought to disturb the balance of ahamkara, and promote a state of disunion within. Any experience, emotion or thought that splits our awareness of ourselves in separate and distinct
fragments is a factor in the process of self-dissolution. From this perspective, it can be seen that cancer is a fundamental disruption of this ahankara self-knowledge, where negative emotions and thoughts send messages from our awareness to our body, telling it to dissolve and spoil this collective vision of self. Negative thoughts and emotions cause each cell in the body to lose trust in itself, and in its neighbor, and even though the notion of unity is essentially hardwired into these cells, a potent experience or sustained emotion or thinking pattern is sufficient to tear away at the fabric and notion of collective unity. Thus in the prevention and treatment of cancer, it is key to learn and practice positive thoughts and emotions, and to dissolve negative feelings and emotional obstructions. Just like a plant in springtime rises up through the hard frozen ground and snow and meets face to face the brilliance of the sun, the One, the source from which all things emanate, the power that resides within, unites with that which exists without, completing the cycle, knowing that we are That, that we are Self, that we are God. In Ayurvedic medicine cancer can be seen as an opportunity to reunite with the source within self that reflects the Supreme Being of existence. This can be done while maintaining a body, or by dying and doing it after one has left the body.

This notion of collective unity also extends beyond individual self, into the community at large. We are part of a grand vision; a collection of communities within communities, and thus external dysfunctions in community can also pull self-apart. One of the most dynamic components in our sense of collective unity is our relationship with our loved ones: parents, siblings, spouses, children, friends and co-workers. These people and our relationship with them are an external map of our own consciousness, the characters in the tragicomedy we call our lives. When a dysfunction exists within these key relationships, the messages we receive affect self, and self in turn affects these relationships. Thus in the treatment of cancer it is key that we heal old wounds and unite broken spirits, reaching out at all costs without expectation of reward, to heal the communal wounds and bring a return to balance, order and harmony, both within and without. In North American Amerindian traditions this often means the medicine person creating a deep play (often lasting days)
between members of the family and community to produce an almost gestalt-like therapy, to aid in reforming the perception and thus healing of self.

Another dynamic component in our sense of collective unity is our relationship with the world at large, our physical environment, what we call nature and the myriad creatures that live on this earth. We are but a cell in this giant organism, and can choose either to integrate ourselves within the collective vision of this organism called earth (Gaia, Ashtie, Pacha Mama), or ignore it, like a cancer cell, and isolate ourselves from this communion. Our relationship and attitude with nature and all its creatures is very important in mediating influences within self. The rise of certain cancers secondary to environmental pollutants is simply a reflection of this dysfunction, where as a collective, humans have attempted to isolate themselves from nature and pillage the earth’s resources like a tumor cell hungry for oxygen and glucose, thinking of nothing except its own growth and proliferation.

The next time you hear somebody referring to growth in the economy, remember what fuels this economy, and what happens when growth isn’t balanced in a collective vision. Thus, besides thinking and creating positive thoughts and emotions, healing wounds between family and friends, the treatment of cancer requires that we engage with nature, that we surround ourselves with the wisdom of this great natural community: communion with it will speak to us, will speak to ahankara and all the cells that comprise it, gently reminding us of this collective vision. Sometimes getting back into nature’s sync is one of the best therapies. Knowing the moon’s phase, tides if you are near the ocean, or other larger rhythms of the environment around you will help to regain a perspective of self in the larger organism of Earth energy.

Ultimately, life is a training ground for the transition we call death. Nobody escapes this truth, and yet death and dying are often ignored in our daily lives. We try not to think of it, or try to think of other things instead, putting it off until some indefinable time in the future. Whether caused by cancer, another disease, an accident, or simply aging, death represents the ultimate dissolution of the
collective spirit of the body. Sometimes cancer is a fatal disease: the condition is advanced and the prognosis is poor, and there is nothing left to do except die. Although this can generate great fear and sadness in both the dying patient and their loved ones, it is important to die well: complete, happy, empowered, and focused on the next stage of the journey. According to many spiritual traditions, death is a journey that decides the direction of your next existence: in fear, anger, and grief death directs consciousness to lower levels of being, where these feelings are perpetuated until this state too dissolves. Living in fear, living in anger and grief, it is easy to see how it is difficult to know anything else, and thus upon this death if you choose these thoughts and emotions you may never escape from them. Thus while you still have the capacity to make a decision, direct your consciousness to love, and allow the spirit of self (ahamkara) to merge with itself; let the conditions of your life fall away, because they were never important anyway.

Traditional Asian perspectives on cancer

In both Chinese and Ayurvedic medicine, a tumor is visualized and observed to be a mass of stagnant energy that occludes and blocks the flow of energy to other tissues, eventually causing the body to become weak.

Chinese medicine

In Chinese medicine a tumor relates to both internal and external pathological factors. External pathological factors consist of the accumulation of phlegm and toxins. Possible concomitant or independent internal factors are blood stasis, and deficiencies of yang, yin, and qi, either singly or in combination. According to traditional Chinese medical theory, the initial stage of tumor development may begin with the local accumulation and stasis of Phlegm. Phlegm stasis is believed to underlie soft tumors and poorly defined masses. Phlegm may also disrupt the flow of qi and lead to blood stasis, or if the qi is deficient, Blood stasis may result. The prolonged stagnation of qi and Blood is another mechanism for tumor development, independent of Phlegm accumulation, in particular in tumors that survive hypoxic
conditions and are thus less susceptible to conventional
treatment.\(^4\) For tumors that evolve without a specific cause,
but are accompanied by local or systemic inflammation and
pain, the cause is usually ascribed to the presence of
Toxins. From a medical perspective this includes a diverse
range of ailments, from viral and bacterial infections, to
snake and insect bites, as well as certain tumors: any region
that is swollen, painful and inflamed. Finally, irrespective
of cause, the net result of tumor formation is a drain upon
vital energy, leading to deficiencies in qi, Blood, yin and
yang. Overall, treatments typically consist of those that:

1. Resolve Phlegm and soften hard lumps, e.g. Chen pi
\((Citrus aurantium)\), Ban xia \((Pinella ternata)\), Gua Lou
\((Trichosanthes kirilowii)\)
2. Vitalize the Blood and Qi, e.g. Yu jin \((Curcuma longa)\),
San qi \((Panax notoginseng)\), Dan shen \((Salvia
miltiorrhiza)\)
3. Dispel Toxins, e.g. Jin yin hua \((Lonicera japonica)\),
Ban zhi lian \((Scutellaria barbata)\), Shi shang bai
\((Selaginella doederleinii)\)
4. Restore Blood, qi, yin and yang: Huang qi \((Astragalus
membranaceus)\), Ling zhi \((Ganoderma lucidum)\), Ren
shen \((Panax ginseng)\)

A number of traditional formulas are generally applied in
the treatment of cancer. Among these are the Kampo
formula WTTC, an acronym derived from the first letter of
the botanical name of the four ingredients: \(Wisteria
floribunda, Terminalia chebulae, Trapa natans, and Coicis
semen\). According to Hsu this formula is effective in
reducing inflammation, edema and pain, inhibiting the
growth of cancer cells and preventing metastasis after
surgery \(1982, 25\). For poor appetite and GI cancers it is
combined with Liu Chun Tzu Tang \((Six Major Herb
Combination)\).

\(^4\)Thus blood-vitalizing therapies are indicated in such cancers, as a synergistic component to enhance the
effectiveness of conventional treatment \(Dharmananda 1998)\)
Ayurvedic medicine

In Ayurvedic medicine a tumor is the most evolved manifestation of **ama**, a dysfunctional component of the body derived from both exogenous and endogenous sources, resulting from defects in digestion and metabolism and displaying cold, heavy, sticky, greasy, hard and firm qualities. Ama opposes the basic life principle, stealing energy away from the rest of the body like a parasite, leading to a diminishment in agni and ojas. Ama can associate with any one, two, or all three of the doshas, leading to differences in the morphology and type of tumor. Tumor formation however is not just dependent upon ama, but on the vitiation of all three doshas. According to the **Madhava nidanam**, in conjunction with ama, vata and the other three doshas increase and bring about abnormalities in the venous system, affecting the blood, muscle, and fatty tissues, causing the formation of tumors. Based on this underlying etiology, Ayurveda defines four basic diseases that correspond with the medical conception of cancer:

1. **Granthis**: a knot-like mass or tumor, often causing pain. Differentiated into vataja, pittaja, kaphaja, medaja and shiraja.
2. **Arbuda**: a slow growing cyst-like mass, immovable, deep-seated, non-suppurating and firm, with a predominance of kapha qualities. Differentiated in vataja, pittaja, kaphaja, raktaja, mamsaja, and medaja.
3. **Gulma**: an intra-abdominal lump that can be fixed or movable, caused by improper dietary habits and lifestyle factors. Differentiated into vataja, pittaja, kaphaja, tridoshaja and raktaja (arising from the vitiation of blood).
4. **Asadhavarna**: a kind of ulcer or sore that will not heal.

Generally speaking, in Ayurvedic medicine a tumor is treated with treatments that have a hot and penetrating nature, including purva karmas such as svedana (sudation) and abhyanga (oil massage), followed by shodhana (pancha karma) therapy and herbs that display a similar, penetrating property including Bhallataka (Semecarpus anacardium), Chitraka (Plumbago zeylanica), Guggulu (Commiphora mukul) and Haritaki (Terminalia chebula). Besides these measures topical therapies were often resorted to, including
the application of escharotic compounds, cauterization, leeches, maggots, venesection, and surgical excision. Although any of these remedies might be used, a full assessment is undertaken to determine the status of the doshas in individual patients, and based on this assessment, an antitumor therapy is applied in context with a general treatment to the affected doshas. Some specific formulations used in the treatment of cancer include Kanchanara Guggulu (3 g tid), Chandraprabha vati (500 mg tid), Kaishora Guggulu (3 g tid), and Abhaya arista (25 mL tid). Due to the deficiencies in ojas often seen in cancer patients, and to prevent or treat cachexia, rasayana therapies are often used concomitantly, including Shilajit, Triphala, Gokshura (Tribulus terrestris), Shatavari (Asparagus racemosa), Ashwagandha (Withania somnifera), as well as remedies to treat agni such as Trikatu (1-3 g tid) and Avipattikara churna (3-6 g tid).

Western herbal perspectives on cancer

Cancer treatment in the Western herbal tradition perhaps reached its apex in the late 19th and early 20th centuries with practitioners such as Eli G. Jones, an acclaimed specialist in cancer. Jones had a unique perspective on cancer, unlike the “regular” physicians of the day, and believed that cancer was a local manifestation of a systemic disease. Jones was sharply critical of treatments such as surgery, having observed many cases where the cancer would simply reappear, or manifest in another location.

From his extensive clinical experience Jones put together a number of factors that he felt were operant in cancer, which he describes in his landmark text, Cancer: Its Causes, Symptoms and Treatment:

1. **Worriment of Mind.** “Worrying weakens the nervous system, lowers the "nerve power" and thus opens the way for the invasion of cancer.”
2. **Vaccination.** “In all states and countries where there is enforced vaccination there you will find cancer on the increase.”
3. **Meat-eating.** “Meat-eating is a prolific cause of cancer.”

4. **Tea and Coffee.** “Tea and coffee weaken the coats of the stomach and the nervous system and produce various disorders in the human system.”

5. **Alcoholic Stimulants.** “The use of intoxicating liquors is a fruitful cause of cancer.”

Of course many of Jones’ assertions are simply that, and were not based on any kind of epidemiological or scientific analysis – they were simply his own thoughts on what the major factors were that caused cancer. Jones goes on to describe his ideas further:

“What our people need is to be taught how to live. There must be temperance in all things. Good pure water, good pure air helps to make good healthy red blood. Unadulterated food, mostly vegetables, easily digested, leaving out tea and coffee, keep the nervous system strong and vigorous. Stop worrying. In this way we can protect ourselves against the dreaded monster -- CANCER. A return to the "simple life" of our forefathers is what we need. Modern civilization, with all its luxury, high living and drinking, and filling the stomach with all kinds of food and drink (the most of it never intended for the human stomach), is only encouraging the inroads of cancer.”

Many of Eli Jones’ comments, addressed to his brothers and sisters close to a hundred years ago, seem even more relevant today.

Jones recommended a variety of treatments for cancer, including herbal and homeopathic therapies. One of the more important therapies he recommended, in much the

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5 Despite his admonishment of meat eating, in *Cancer: It's Causes, Symptoms and Treatment*, Jones states “In my own practice I have met with many cases of genuine cancer where the victim of that disease had lived on a strictly vegetable diet from fifteen to twenty years. In one case a lady had a cancer of the breast; she impressed it on my mind that she had followed a certain strict diet, and as a result of this she had the disease under control. When I saw the cancer it was open and discharging and in the very last stage. Another case I saw in consultation with a very fine physician in New York State. An old man who had lived on a strictly vegetarian diet for twenty years and it was about the worst case of cancer on the lip that I ever saw.”
same manner as Samuel Thomson, but influenced by the European Spa tradition, was an Epsom salts bath, to neutralize toxins and soothe the nerves.

You can find some of Eli Jones’ classical treatment in the appendix of this lesson.

**Natural compounds in cancer therapy**

Based on the overview of the mechanisms involved in carcinogenesis stated in Part One, a number of natural products have shown promise. Although most of the research is still in the preliminary stage, many of these compounds have a long history of usage in traditional medicine.

**The Antioxidant Debate**

There has been an ongoing heated debate for some years on the potential adverse effects of antioxidants and herbs supplemented while on chemotherapy and/or radiotherapy. As such there has been significant research on this issue.

Lamson\(^6\) states that supplementary antioxidants given concurrently with chemotherapy agents, which employ a free radical mechanism, might reduce their effectiveness. This statement should not serve as scientific closure on an adjunctive treatment of possible great promise in cancer therapy, as much published research indicates the cautious and judicious use of a number of antioxidants can be helpful in the treatment of cancer, both as sole agents and as adjuncts to standard radiation and chemotherapy protocols.

It was suggested that antioxidants might interfere with the oxidative mechanisms of alkylating agents as these drugs create substantial DNA damage, resulting in cell necrosis. The evidence, however, refutes this, as it indicates that a sizeable amount of chemotherapy damage is by other mechanisms, which trigger apoptosis or cell death. Given this evidence the argument that antioxidants are likely to

\(^6\) Lamson DW, Brignall MS; Antioxidants in cancer therapy; their actions and interactions with oncologic therapies; *Altern Med Rev.*, 1999 Oct;4(5):304-29.
interfere with most chemotherapy is too simplistic and probably untrue. Interactions between antioxidants and chemotherapeutics cannot be predicted solely on the basis of presumed mechanisms of action. Also many chemotherapeutic agents do not act via an oxidative mechanism (for example, 5-fluorouracil or tamoxifen).

A recent study was conducted on small-cell lung cancer in humans using combination chemotherapy of cyclophosphamide, adriamycin (doxorubicin) and vincristine with radiation and a combination of antioxidants, vitamins, trace elements and fatty acids. The conclusion was ‘antioxidant treatment, in combination with chemotherapy and irradiation, prolonged the survival time of patients’ compared with expected outcome without the composite oral therapy.

There is also a large body of evidence showing positive effects of antioxidants in the period immediately following chemotherapy administration, resulting in a higher percentage of successful outcomes.

The research has highlighted what can be otherwise a difficult topic in the circumstances of the life-threatening nature of the disease. We all want our patients to get well in the best way possible. Overall the research has shown that antioxidants enhance the anticancer effect of chemotherapy (with a few noted exceptions). Antioxidants also reduce the side effects of chemotherapy, improve the quality of life of the patient and reduce the chance of recurrence. Selected antioxidants at specific doses also have cancer preventative effects if taken regularly.

**Apoptosis**

Apoptosis refers to a cell’s timely and natural death; cancer cells appear to lack this capacity, are long-lived and proliferate rapidly. A number of natural compounds have been shown to induce apoptosis in cancer cells, some by increasing TGFβ expression or improving TGFβ signaling, including:

- vitamin A, especially beta carotene
- vitamin C
• vitamin E
• vitamin D3
• boswellic acids
• curcumin
• eicosapentaenoic acid (EPA)
• flavonoids (e.g. apigenin, luteolin, quenistein, quercetin)
• Garlic
• hypericin
• monoterpenes (e.g. limonene, perillyl alcohol)
• selenium (Boik 2001, 33)

Cell differentiation

One important feature of cancer cells is their marked lack of differentiation. A number of natural products have been shown to induce differentiation of cancer cells, in vitro, including:
• arctigenin
• vitamin A
• boswellic acid
• bromelain and other proteolytic enzymes
• daidzein
• genistein
• hypericin
• flavonoids
• selenium
• vitamin C
• vitamin E
• emodin
• EPA and DHA
• monoterpenes
• resveratrol
• vitamin D3 (Boik 2001, 391-396)

Signal transduction

Cancer cells often feature abnormal signal transduction, sequestering themselves from the communications of other cells, or altering how these communications are processed in the cell, or secreting their own growth factors for self-stimulus. Most cytokines that bind to the plasma membrane to affect cell activities rely upon phosphorylating kinases, and in cancer cells, some of these enzymes are produced in excessive amounts or are hypersensitive to stimulation,
including protein tyrosine kinases and protein kinase C. Cyclin-dependent kinases in particular promote mitosis and cellular proliferation, and are inhibited by proteins that are encoded by \( p21 \) and \( p27 \). Natural compounds that inhibit protein tyrosine kinases include curcumin, emodin, flavonoids, hypericin, and parthenolide. Compounds that inhibit protein kinase C include curcumin, emodin, flavonoids, hypericin, eicosapentaenoic and docosahexaenoic acids, selenium and vitamin E. Natural compounds that induce \( p21 \) or \( p27 \) activities include vitamin A, flavonoids, silymarin, vitamin D\(_3\), and vitamin E (Boik 2001, 40-43). Another factor in signal transduction is reactive oxygen species (ROS), produced by a variety of factors, including the activation of transcription factors such as nuclear transcription kappa-beta (NF-\( \kappa \beta \)), which also induces cellular proliferation in cancer cells. Natural compounds that inhibit NF-\( \kappa \beta \) include vitamin C, vitamin E, alpha lipoic acid, \( N \)-acetyl cysteine, flavonoids, and curcumin (Boik 2001, 59-60).

**Cell-to-cell communication**

Cancer cells often display abnormal cell-to-cell communication, mediated by cell adhesion molecules and gap junctions. Natural compounds that facilitate cell-to-cell communication include apigenin, vitamin A, vitamin D\(_3\), genistein, and selenium (Boik 2001, 72).

**Immune detection**

Cancer often evolves in patients already suffering from some degree of immunosuppression, with a decline in the number of NK cells and macrophages that seek out and initiate the destruction of mutant cells. Botanicals that enhance immune function include *Astragalus*, *Eleuthrococcus*, *Ganoderma*, *Chaga*, *Coriolus*, *Panax*, and *Lentinus*. Other compounds that enhance immune functions include flavonoids, and eicosapentaenoic and docohexaenoic acids (Boik 2001, 132-139).
Angiogenesis

Angiogenesis refers to the growth of blood vessels, in this context in cancerous tissue, promoting tumor growth and development. A variety of factors are involved in angiogenesis including cytokines such as VEGF and prostaglandins (PGE2) that promote inflammation and vascular permeability. Natural compounds that inhibit increased vascular permeability include anthocyanidins and proanthocyanidins, as well as herbs such as Gotu Kola, Butcher’s Broom and Horse Chestnut. Natural compounds that correct prostaglandin synthesis include boswellic acids, bee propolis, curcumin, eicosapentaenoic and docosahexaenoic acids, flavonoids, Garlic, and vitamin E. Other compounds that inhibit angiogenesis include vitamins A and D3 (Boik 2001, 91-97).

Invasion and metastasis

Invasion is the process by which a tumor secretes enzymes to digest the structure of the extra cellular matrix (ECM). Natural compounds that inhibit the activities of enzymes such as hyaluronidase include apigenin, boswellic acids, escin, luteolin, proanthocyanidins, vitamin C and the herb Gotu Kola. Natural compounds that enhance the integrity of the ECM include anthocyanidins and proanthocyanidins, eicosapentaenoic acid, curcumin, emodin, genistein, luteolin, vitamin A, vitamin C, medicinal mushrooms and Gotu Kola (Boik 2001, 110).

Resveratrol has been shown to modulate p53 to induce apoptosis (programmed cell death) as well as knocking out excessive p53, having a beneficial effect in preventing cancer. It has also been found to induce apoptosis via modulating other cellular molecules such as cyclin D1, p21, BCL2, BAX, Bcl XL, caspase 9 and p27.
**Green tea** has also been found to affect p53 and p21. One study has found that green tea enhanced *Zizyphus jujuba’s* selective cytotoxic activity by causing cell death via up-regulation of p53 and p21 while decreasing cyclin E levels in HepG2 cells. Green tea has also been found to inhibit tumor growth and angiogenesis as well as induce apoptosis.

**Genistein** has been found to influence p21 transcription by markedly inhibiting proliferative activity and inducing the expression of p21 plus ER beta (Estrogen Receptor beta).

**Vitamin D** also influences p53 and p21. Both p21 and p53 have vitamin D binding sites (VDR). If a patient is deficient in vitamin D, both p53 and p21 may be affected, possibly influencing cancer development or progression.

**Flavonoids**—such as quercetin—have also been found to influence p53 in relation to cancer. One study found that quercetin helped to stabilise p53 at both the mRNA and the protein level to reactive p53-dependent cell cycle arrest and apoptosis. This was achieved by quercetin inducing p53 phosphorylation and total p53 protein, but not up-regulating p53 mRNA at transcription. It also stimulates p21 expression and suppresses cyclin D1 to activate cell cycle arrest. Quercetin was also found to inhibit p53 mRNA degradation post-transcriptionally.

**Centrosomes** These microtubules aid cell mitosis by creating the polar rejection force that pushes the chromosomes away from the spindle poles by wobbling at high frequencies. A rise in **intracellular calcium** aids the regulation of the polar force at the onset of chromosomal splitting. If this is defective, chromosomal instability (characteristic of most cancers) can occur. It has also been found that cells that have dysfunctional p53s have more centrosomes.

**Vitamin A** plays a key role in centrosomes and chromosome replication through the retinoblastoma protein, which is a regulating protein of cyclins D and E, cdk4 and 6 (cyclin dependent kinase), cdk inhibitors p16, p15 and p53. A **vitamin B12** deficiency has been associated with enlarged, disrupted centrioles in monocytes and neutrophils. Proteasomes play an important role in a variety of cellular
processes such as cell-cycle progression, signal transduction and the immune response, and are important in maintaining rapid turnover of short-lived proteins. They also prevent accumulation of misfolded or damaged proteins.

**Zinc** increases proteasome substrates such as p5 and p21, as well as decreasing the enzyme that degrades these substrates in centrosomes.

**Folate** also plays a critical role in the prevention of chromosome breakage and hypomethylation of DNA. A folate deficiency can cause centromere defects that can induce abnormal distribution of replicated chromosomes during mitosis. It has also been found to be a risk factor for chromosomes 17 and 21 aneuploidy, which has been observed in breast cancer and leukaemia.

**Glycolysis and p53** A common theory sometimes postulated in the CAM treatment of cancer is that tumors feed on sugar. In part this is true, as cancer cells work differently to other cells biochemically. Research has found that most cancer cells have higher rates of glycolysis than normal cells. Research has found that, after a loss of functional p53, there were mitochondrial changes, up-regulation of rate-limiting enzymes and proteins in glycolysis and intracellular pH regulation, hypoxia-induced switching to anaerobic metabolism leading to higher lactic acid levels, and metabolic reprogramming.

Extrapolation of these results has led to various diets being developed to ‘starve’ the cancer. This theory has not yet been proven to work; however, it is still recommended that people with cancer decrease their sugar intake as it has been found that insulin-like growth factor 1 is involved in a number of cancers. One animal study has shown delayed growth of gastric cancer cells with administration of a ketogenic diet high in omega-3 fats.

The high usage of glycolysis by tumors is also why it is often recommended in traditional CAM theory to alkalize the body of a patient with cancer. It is hoped that increasing buffers and trying to decrease the acidic environment can decrease the spread of cancer. This theory has yet to be validated, though alkalinizing diets (higher fruit and vegetable
intake, and lower meat intake) generally have positive effects in cancer.

Another theory (the Warburg effect) suggests ‘oxygenating’ the body, as cancer cells use more anaerobic glycolysis than normal cells do and normal cells use the aerobic system of the mitochondria. Increasing oxygen availability (for example, through breathing exercises and circulatory stimulants) to cells generally may aid the normal cells rather than cancer cells, and also aid the removal of toxins and carcinogens.

Herbal Considerations

Many botanicals have been used by modern medicine in the treatment of cancer. Some of these have powerful effects in the human body and are therefore used as forms of chemotherapy, for example the vinca alkaloids (vincristine and vinblastine) from the periwinkle (Catharanthus rosea) and taxanes from the bark of the Pacific yew tree (Taxus brevifolia). Plant alkaloids are mitotic inhibitors. They can stop mitosis or inhibit enzymes for making proteins needed for reproduction of the cell. They are cell cycle specific and work during the M phase of the cell cycle.

But whether to use herbal (plant) medicines in combination with chemotherapy provokes a similar debate to that of the combination of antioxidants and chemotherapy or radiotherapy.

Korean ginseng (Panax ginseng) has been extensively studied in Asia with approximately 1500 studies done, 500 on the pharmacological properties. Overall the research shows that Korean ginseng is an excellent adaptogen for the elderly, for debility and to enhance the body’s capacity to cope with stress.

Large case control studies in Korea found an inverse association of cancer and ginseng consumption, with the relative risk of cancer being 50% less in ginseng users. The risk profile showed a clear dose response relationship, over time and with a particular type of ginseng (red ginseng powder). There was also a preventative effect after 1 year.
Concurrent use of Panax ginseng has been shown to improve outcomes with both chemotherapy and radiotherapy. Ginsengsides have been shown to induce differentiation of cancer cells (only undifferentiated cells can spread), by reducing inflammation through inhibiting COX2 expression, by up-regulating transcription of superoxide dismutase and catalase (antioxidants), inducing the process of apoptosis, reducing the overexpression of growth factors, decreasing the expression of oncogenes, and inhibiting the process of proliferation by decreasing angiogenesis and cell adhesion (as effective as 5-fluorouracil). Ginseng is also known as an immunmodulator as it lowers cortisol levels under stress and activates T cells.

Eleutherococcus senticosus (Siberian ginseng) and other ginsengs have similar properties.

Herbal Medicines and Multidrug Resistance

There is substantial research being conducted on the inhibition of P-glycoprotein (P-gp) transporters and the development of multidrug resistance – the bane of chemotherapy. P-gp expression triggers cells to start producing increasing numbers of metabolic pumps to expel toxins from the cells, particularly chemotherapy drugs. Therefore inducers of P-gp are unfavorable to the outcomes and inhibitors of P-gp are favorable.

Multidrug resistance to chemotherapy caused by P-gp expression is common in cancer cell lines. The individual expression of P-gp can vary eightfold and is encoded by various genes, for example the MDR1 gene in breast cancer. Women who express the MDR1 gene (and therefore higher levels of P-gp) are three times more likely to fail to respond to chemotherapy by developing multidrug resistance. Agents that induce P-gp (P-glycoprotein) transporters generate a poor response to chemotherapy; therefore agents that inhibit this activity (inhibit P-gp expression) are potentially useful in improving outcomes.

There has been significant research on P-gp transporters and the effect of herbal medicines in inhibiting these. The herbs that inhibit P-gp expression will therefore have a potential role in reducing the risk of chemotherapy resistance,
potentially leading to more effective therapies with less toxicity.

**Herbs that inhibit P-gp include:** Curcuma longa (turmeric), quercetin, Camellia sinensis (green tea), resveratrol (from grape skins), Silybum marianum (Milk thistle), and the berberine containing herbs – Hydrastis canadensis (goldenseal), Coptis chinensis (goldthread). P-gp transport inhibitors are also found in grapefruit juice, apple juice, Rosmarinus officinalis (rosemary extract) and genistein (from soy).

By the same token, herbs that induce P-gp such as Hypericum perforatum (St John’s wort) should be avoided. Hypericum has been found to cause the increased clearance of irinotecan (and possibly taxol and tamoxifen). Best practice therefore indicates that it is contraindicated for administration to patients who are undergoing chemotherapy.

Also avoid grapefruit juice while on chemotherapy with taxol or tamoxifen as this can alter the dosage (through liver metabolism pathways).

**p53 Suppressor Gene** Agents that increase the expression of the p53 tumor suppressor gene will improve outcomes in patients. p53 is a major trigger for apoptosis and is inhibited in oncogenesis. The p53 gene regulates genes important to the cells response to chemotherapy, largely through the multidrug resistance gene (MDR1) expression in tumors. MDRI has also been shown to inactivate the p53 tumors suppressor gene. Loss of functioning of p53 can reduce the body’s ability to resist the growth of tumors (e.g. tamoxifen) and the loss of functional p53 may induce P-gp expression and increase the development of multidrug resistance.

Research shows promise with herbal medicines that can inhibit these P-gp transporters and that can increase the activity of the p53 gene.

**Herbs that increase p53** and therefore apoptosis are, Camellia sinensis (green tea), Centella asiatica (gotu kola), Glycyrrhiza glabra (liquorice root), Terminalia arjuna, Poria
cocos (mushroom polysaccharides), Artemesia annua (Chinese wormwood), iscador (mistletoe preparation) and ginsengs.

**COX (Prostaglandin) Pathways** Research shows that substances that inhibit the COX pathways not only reduce inflammation but also may play a role in reducing P-gp expression. COX-2 inhibitors reduce inflammation and aromatase (for estrogen receptor tumors). Grape seed, Zingiber officinale (ginger), Allium sativum (garlic), Curcuma longa (turmeric) and the salicylate-containing herbs such as Filipendula ulmaria (meadowsweet) and Salix alba (white willow) bark are effective at inhibiting these pathways. Specific COX-2 inhibitors are Curcuma longa (turmeric), ginseng, Camellia sinensis (green tea), as well as fish oils, quercetin and genistein (soy).

**Camellia sinensis (Green Tea)** Catechins in green tea such as epigallocatechin gallate (EGCG) are powerful antioxidants and anti-inflammatories and, as we saw above, activate the p53 tumor suppressor gene. Green tea can be drunk along with chemotherapy and has synergistic activity. Green tea acts at different levels and via different mechanisms than chemotherapy. Green tea inhibits the COX-2 inflammatory pathways, promotes cell-cycle arrest and increases apoptosis (activates p53). It disables the multiresistant pumps and is a major antioxidant.

**Curcuma longa (Turmeric)** Apart from its activity as a P-gp inhibitor, it has been shown that curcumin works by way of another mechanism unrelated to P-gp. Some tumors produce high levels of glutathione S-transferase (GST), an antioxidant enzyme that helps protect the cells from oxidative injury by chemotherapy agents. Curcumin appears to inhibit this enzyme in vitro in cancer cells, potentiating the effects of the chemotherapy drug doxorubicin, even when P-gp levels are high. Compared with several other GST inhibitors (in vitro) in neoplastic cells, curcumin was, by far the strongest inhibitor. Resistance to chemotherapy drugs is commonly associated with elevations in cellular levels of the inflammatory mediator nuclear factor kappa B (NFkB), a process that is inhibited by curcumin, reducing drug resistance in cancer cells.
Curcuma plays many roles in improving the health of the person undergoing treatment for cancer. It reduces P-gp transporters, reduces MDR, reduces toxicity in the liver, as well as being antioxidant and anti-inflammatory through several pathways including COX-2.

**Integrated Treatment: Chemotherapy, Nutrients and Herbal Medicines**

**Glutamine:** When glutamine is supplemented in conjunction with chemotherapy, tumors decrease in size by 45%, compared with 25% when glutamine is not supplemented. The infection rate also drops dramatically to 3%, compared with 100% without glutamine. Survival time is improved if supplementing with glutamine.

**Dose:** 18,000 mg per day for the 3 days prior to each dose of chemotherapy or radiotherapy.

**Melatonin:** Melatonin protects against many of the toxic effects of chemotherapy. Melatonin also helps with sleep, is an antidepressant, regulates estrogen receptors and is a powerful antioxidant. Melatonin increases p52 expression and modifies cytokines.

**Dose:** The appropriate dose of melatonin can vary between individuals by a factor of 2000. Doses recommended are 3–40 mg taken after dark, and increase or decrease as required. Use the measure of an excellent night’s sleep as an indicator.

**Vitamin C:** Vitamin C reverses vincristine resistance and improves the effectiveness of adriamycin, cisplatin, doxorubicin and paclitaxel (taxol).

**Quercetin:** Quercetin was shown to inhibit adriamycin resistance in estrogen receptor negative breast cancer and to potentiate the action of many chemotherapeutic drugs while reducing side-effects.

**Dose:** 2000 mg per day, taken with bromelain to improve digestion.

**Glutathione (and Selenium):** Glutathione (and selenium) reduce the neurotoxicity, nephrotoxicity and myelosuppression of cisplatin, and reduce damage to the brain, the kidneys and bone marrow.
Aged Garlic (Allium sativum) protects the liver with methotrexate (along with folic acid).

Centella asiatica (Gotu kola): reduces chemotherapy resistance with vincristine and vinblastine.

Crataegus oxycantha (Hawthorn) and Coenzyme Q10: Hreduces heart damage from the side effects of herceptin and doxorubicin. Dose of coenzyme Q10: 300–400 mg per day with food.

Indoles (Brassica Family): inhibit P-glycoprotein activity by inhibiting the binding specifically of doxorubicin and vinblastine, making these more effective.

Rhodiola rosea (Rhodiola): protects the liver with adriamycin.

Rosmarinus officinalis (Rosemary): Rosmarinus officinalis reduces resistance to a broad range of compounds by increasing the intracellular accumulation of drugs such as doxorubicin, vinblastine (P-gp inhibitor). It also has antioxidant properties and is liver protective.

Withania somnifera (Ashwagandha): Withania somnifera reduces the leucopenia induced by cyclophosphamide and has an immunopotentiating and myeloprotective effect.

Astragalus membranaceus and Platinum-Based Chemotherapy: A meta-analysis of 34 randomized studies was conducted involving 2815 patients. When taking the Astragalus formula, 12 studies showed reduced risk of death at 12 months, 30 studies reported improved tumor response, two studies showed reduced risk of death at 24 months.

In four studies with 257 patients, a preparation of Panax ginseng, Astragalus membranaceus, Eleutherococcus senticosus and Mylabris cichorii (Blister beetle) stabilized or improved patient performance. The conclusion from this research was that: ‘Astragalus-based Chinese herbal medicine may increase effectiveness of platinum-based chemotherapy when combined with chemotherapy’. The researchers found evidence that Astragalus-based Chinese
herbal medicine may increase the effectiveness (by improving survival, tumour response and performance status) and reduce the toxicity of standard platinum-based chemotherapy for advanced non-small-cell lung cancer. Astragalus potentiates therapeutic activity in chemotherapy (mitomycin, cisplatin, cyclophosphamide, fluorouracil) and radiotherapy.

**Dietary intervention and cancer**

Diet is a contentious issue in the treatment of cancer, with the traditional associations between foods such as saturated fats and red meat undergoing a reassessment. While modern oncology does not address the diet in any significant measure, many complementary and alternative therapists tend to recommend a vegetarian, vegan style diet, with an avoidance of all animal products. From a traditional perspective, such a diet makes a great deal of sense. Cancer is viewed as a disease of excess and stagnation, and by implementing a diet that is nutritionally sub-optimal by decreasing calories and using foods that impair nutrient absorption (e.g. cereals and legumes), but at the same time are loaded with antioxidant and anti-inflammatory compounds (e.g. vegetables, fruits), the overall approach is to limit the growth and development of all cells. Given that cancer cells have a higher rate of metabolism than normal cells, such a diet would tend to favor an overall antitumor activity. Thus for these reasons a vegan style diet appears to make a great deal of sense, even if it may not be an optimal diet for otherwise healthy individuals.

There are some confusing data regarding the efficacy of vegetarian diets however, with some studies suggesting that vegetarians do not have a reduced risk of cancers of the breast, bowel or prostate cancer, three of the most common cancers that aren’t directly attributable to a specific cause such as smoking (i.e. lung cancer). Often reducing meat and having it in the form of soups and stews seems to be a great compromise. The use of ‘bone soup broth’ has been very useful in cancer cases, especially when appetite is reduced.

An important component in treating advanced cancer is preventing cachexia (or wasting disease, with loss of
muscle and body mass), and it would seem that a strictly vegetarian diet would not be the best for this. Besides being a dense source of calories, animal products also contain many helpful antitumor compounds including vitamin A, vitamin B12, folate, selenium and zinc, as well as health promoting fats such as conjugated linolenic acid (CLA), eicosapentaenoic acid (EPA), and docosahexanoic acid (DHA). Further, it has been recognized that a diet rich in omega 6 fatty acids enhances tumor growth and proliferation, which is a component of a strictly vegetarian diet from the consumption of grain, seed and nut oils. Lastly, an important factor in carcinogenesis appears to be insulin resistance and compensatory hyperinsulinemia. Thus a diet that is naturally low in rapidly digested carbohydrates appears to be of vital importance, and is something more difficult, but not impossible, to obtain in a vegetarian diet. One of the most important components in the diet is to lower the glycemic index by the use of fats, including monounsaturated fats such as olive oil and saturated fats such as clarified butter.

If animal products are included in a dietary regimen for cancer, it is important to be aware that environmental toxins such as xenoestrogens, organochlorines, and polychlorinated biphenyls are thought to accumulate and become concentrated in animal tissues in significant amounts. Thus if animal products such as milk, meat, fish, poultry or eggs are included in dietary therapy, sourcing the quality of meat would seem to be of vital importance.

The most important component in the dietary treatment of cancer is to ensure a maximal amount of antioxidant-rich, fresh vegetables and fruits, regardless if the diet includes animal products or not. Such foods should make up at least 50% of the bulk of the diet, and although they provide little in the way of calories, supply the body with abundant vitamins, mineral and phytochemicals that prevent carcinogenesis and assist in detoxification. This is where the ‘rainbow diet’ with many colors of fruits and vegetables really helps.

From a traditional Ayurvedic and Chinese perspective however, vegetables should be lightly cooked, perhaps steamed, as a common feature in cancer patients is
impaired absorption (i.e. mandagni, Spleen qi deficiency). Many practitioners agree however that fresh vegetable juices are well-absorbed, and can be an important adjuvant to dietary therapy. Carrot and or wheat grass juices have found great success in the treatment of cancer. The carrot juice should be relatively fresh and can include other vegetables (especially ginger root). It is suggested to have 3 – 6 cups daily. Wheat grass is usually 1 – 4 ounces daily.

**Holistic Interventions in Conventional Cancer Treatment**

**Cancer as a Model for Communication**

As stated from the beginning, a cancerous cell or tumor system is a system that does not communicate with the whole community that it evolved from. It is as if the cancer system has no desire to be part of the whole anymore. On the other hand, many of the medicinal substances we use to strengthen the body while working on cancer are just that, extreme examples of community communicators. In this we mean that many of these organism’s natural habitats are very large, intense communities. To be active and to survive in these communities, communication, or more important, living in harmony with the rest of the organisms in the community, is the ‘prime directive’. Some botanicals that fit into this model are of course the fungi. It has been shown that some of these native colonies are hundreds of miles in diameter and have lived in a specific area for thousands of years. Fungi such as *Ganoderma*, *Coriolus*, *Cordyceps*, *Chaga*, *Maitake* and *Shiitake*, form very large intense communities. The algae, like *Chlorella* also live in very dense colonies. As well, organisms like Krill ‘bloom’ in colonies of billions, being several miles across and quite deep in the Antarctic and other places on the planet.

Detailed chemical mechanisms of how these organisms work can be found in your other courses. From a holistic point of view they work because they vibrate very strong connections to community and communication. Filling your body with their vibrations sets up a harmonic or
vibration communicating with the bigger whole - may that be earth energy (Gaia, Ashtie, Pacha Mama) or even more esoteric vibration of higher dimensions. It is almost like these organisms are sitting there, chanting a universal OM and after a while our physical cells start to chant in harmony with them and before you know it, there is no room for cancer. It is like giving peace a chance.

Using this philosophy you can see how holistic therapy is the exact opposite of conventional allopathic therapy. Allopathic medicine wages war on the cancer, while holistic medicine ‘gives peace a chance’. Usually the cancer affects less than 1/100 of 1 percent of the body. Or in other words the body is 99.99% cancer free. Holistic medicine for the most part is concentrating on the 99.99%, while allopathic therapy is concentrating on the much smaller percentage. This also means that in the strictest sense there is no conflict or ‘drug interaction’, we are just assuring that after the cancer is ‘dealt with’ the rest of the community (body cells) is communicating with the whole and is as strong as possible.

Another very important point is that many oncologists advise their patients not to take any supplements such as vitamins and herbs. This is based on faulty assumptions and data. Be that as it may, by giving a person mushrooms they are technically not taking herbs. For many years now, fungi have been considered a separate kingdom and are not part of the plant kingdom, so in reality they are not really herbs. Plus I have never heard of a doctor saying that you can’t eat lobster or shrimp? As a Clinical Herbalist, understanding this “community and communication” approach is very important and a key component in the holistic treatment of cancer. Combining this with specific treatments for specific cancers creates a very thorough approach to cancer therapy.

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**Suggested program for General Cancer**

*Myco-Feron* 2-3 caps bid
- tid or 5 *Mushroom Super food powder* ½ - 1 tsp, bid
- Coriolus 3 cap bid
- Chlorella 5-10 tab bid
- Sharks cartilage 1 tsp bid
- Krill 2 cap bid
- Carrot juice 2 – 6 cups qd
- Wheat grass juice 1 – 4 oz qd

**Myco-Feron Supreme Blend**

- Reishi extract (15:1) 50 mg.
- Reishi Mycelium 50 mg.
- Cordyceps extract Cs-4 35 mg.
- Cordyceps mycelium 35 mg.
- Agaricus Mushroom extract 4:1 30 mg.
- Agaricus Mushroom powder 25 mg.
- Maitake extract (4:1) 30 mg.
- Maitake mycelium 40 mg.
- Shiitake (LEM) 30 mg.
- Chaga Mushroom (8:1) 25 mg.
- Poria Cocus 40 mg.
- Zu Ling (Polyporus) 35 mg.
Shark cartilage

As early as the 1970s, Dr. Judah Folkman of Harvard Medical School suggested inhibiting new blood vessel formation as a way to fight cancer.

In 1983, two researchers at the Massachusetts Institute of Technology published a study showing that shark cartilage contains a substance which significantly inhibits the development of blood vessels that nourish solid tumors, thereby limiting tumor growth.

Working independently, medical researchers at Harvard University Medical School found that if one could inhibit angiogenesis (the development of a new blood network), one could prevent the development of tumor-based cancer and metastasis.

IP6 (inositol hexaphosphate, or phytic acid)

- Currently, there are a growing number of studies that support IP6's cancer-fighting properties. Additionally, IP6 has been shown to have potent antioxidant properties and can help treat kidney stones as well as high cholesterol and lipid levels. IP6 goes by the chemical name inositol hexaphosphate.
- IP6 is basically a sugar molecule with phosphate groups attached.
- IP6 has been shown to inhibit various cancers in humans and animals.
- IP6 has been shown to be an effective treatment for kidney stones, high cholesterol and high blood lipids.
- Much of the scientific support for IP6 is derived from the research of noted scientist Abulkalam Shamsuddin, M.D., of the University of Maryland School of Medicine.
- Foods that contain significant amounts of IP6 include: soybeans, rice, sesame, beans, legumes, corn and cereals.
- IP6 has no known toxic effects.

IP6, a ubiquitous substance found in virtually all mammals, is composed of the sugar inositol with six phosphate groups attached to it. It is also an important component of cereals.
and legumes and may be the anti-carcinogenic ingredient in fiber.

IP6 has been shown in more than 25 studies to prevent the growth of tumors and shrink existing tumors. IP6 has been introduced by mouth, by injection directly into tumors, intramuscular injection, intraperitoneal injection, etc. Regardless of how IP6 was given, it consistently had the same effects, whether it was tested on a colon-cancer model, a breast-cancer model, smooth-muscle cells, skeletal muscle tumors, liver cancers, etc.

In addition to animal studies, there are several human studies that have shown that IP6 inhibits growth of human prostate cancer cells and adenocarcinoma. Scientists have observed that cancer cells can revert back to normal cells in the presence of IP6. It should be pointed out that most of the research has been done with animals. In order for IP6 to gain greater support by the medical community, more human trials are needed.

Abulkalam Shamsuddin, M.D., of the University of Maryland School of Medicine, is one of the leading authorities on IP6. He has an extensive scientific publication record on the topic of IP6. He reports his findings in his book IP6--Nature's Revolutionary Cancer Fighter (Kensington, 1998). According to Shamsuddin, the best evidence for the role of IP6 in fighting cancer is found in the comparison of cancer rates in Danish and Finnish populations. The fiber consumption of the Danes is nearly twice that of the Finns, yet the incidence of cancer in Danes is twice that of the Finns. This discovery suggests that the quality of fiber may be more important than the quantity of fiber consumed. Shamsuddin says, "The Finns actually eat a lot of porridge, which is where you have a lot of IP6. You see the Danes eat a lot of fiber, and that fiber does not have IP6."

Although Shamsuddin says for prevention, a normal, healthy individual should take 1 to 2 gm daily. Individuals with greater risk for cancer (due either to heredity predisposition or lifestyle factors), should take 4 gm daily as a preventative measure. If you want to make IP6 part of
your treatment for cancer, take up to 8 gm daily (dose depends on stage of cancer).

**Holistic therapies during surgery**

Surgery can be a traumatic event, both mentally and physically. After surgery it is vitally important to promote proper tissue healing, reduce inflammation and oxidative stress, and prevent scar formation. It is often observed that the immune system is somewhat suppressed after surgery, and needs to be supported through this process. Some helpful therapies include:

- Vitamin A, 25,000-50,000 IU daily
- Vitamin C ascorbate, 1-3 g bid-tid, to bowel tolerance
- Vitamin E, 800-1200 IU daily
- Zinc, 50 mg daily
- Bromelain, 500 mg, 3-4 times daily
- Siberian ginseng or Rhodiola for shock and healing potential
- Oligomeric proanthocyanidins (OPCs), 100 mg 2-3 times daily
- Traumeel (or arnica homeopathic) 1 tab tid
- Anti-inflammatory and vulnerary herbs: *Scutellaria baicalensis*, *Curcuma*, *Centella*, *Calendula*, *Plantago*, *Hypericum*, *Stellaria*, *Crataegus*, *Vaccinium*
- Immunomodulants: *Astragalus*, *Ganoderma*, *Withania*, *Centella*, *Echinacea*, *Schizandra*
- EPA/DHA, 1000-2500 mg each daily

These treatments can be applied one week before and for several weeks after surgery, to promote healing.

**Holistic therapies during radiotherapy**

Radiotherapy can have a number of negative effects, and each symptom should be addressed specifically. Many of the therapies used prior to and after surgery should also be used prior to and after radiation therapy. A common symptom experienced during radiotherapy is ulcerated, cracked and irritated mucus membranes. In such cases an infusion of botanicals including *Glycyrrhiza*, *Ulmus*,

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**Suggested program for surgery**

- Ener–Jazz 3 cap bid
- Traumeel 1 tab under tongue tid
- BEVC 3 tab bid
- Krill 1 cap bid, or fish oil 3 cap bid
- Reishi extract 3 cap bid
Symphytum, Althaea, Plantago, and Verbascum can be used, 1-2 litres daily. In addition, soothing medicated oils can be prepared with herbs such as Sida cordifolia and Asparagus and oils such as almond, ghee or coconut, given every few days as a full body massage. Specific formulas include Narayana and Mahanarayana taila.

**Holistic therapies during chemotherapy**

Chemotherapy is noted for causing a number of debilitating side-effects, including a loss of appetite, cachexia, nausea and vomiting, constipation or diarrhea, dry mouth, mouth sores, pain, neuropathy, hemorrhaging and anemia. The following is a sample of different treatments that can be applied for each of these conditions:

- **Loss of appetite**: bitter herbs including Gentiana and Centaurium erythraea; Nux vomica, digestive enzymes
- **Nausea and vomiting**: citrus fruits, Zingiber; Foeniculum; Cannabis
- **Cachexia**: EPA/DHA, whey protein, Deer Antler extract; anabolic herbs including Withania and Panax
- **Diarrhea**: synbiotics, bentonite clay, oral rehydration; antidiarrheal botanicals, including Rubus root, Cinnamomum bark, Hydrastis, Terminalia chebula (decoction or tincture), Myrica, Agrimonia, Geranium, Atropa
- **Constipation**: magnesium; herbal cholagogues including Taraxacum and Rumex, Triphala with Operculina; LBT-3, enemas, alternating oil and decoction enemas; coffee enema
- **Pain**: vitamin K1, alpha lipoic acid; analgesic herbs including Piper methysticum, Echinacea, Corydalis, Filipendula, Salix, Piscidia, Atropa
- **Neuropathy**: vitamin B complex, alpha lipoic acid, Shilajitu, Hypericum, Withania
- **Dry mouth**: Pilocarpus, Zanthoxylum, Spilanthes, Echinacea
- **Mouth sores**: Ulmus, Aloe, Glycyrrhiza
- **Hemorrhaging**: Panax notoginseng, Capsella, Achillea, Geranium, Cinnamomum

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**Ener-Jazz**

Siberian Ginseng root 100mg  
Astragalus root 25mg  
American Ginseng root 25mg  
Reishi Mushroom 25mg  
Licorice root 200mg  
Codonopsis root 25mg  
Fo-Ti root 50mg  
Dosage: 2 – 3caps, bid - tid
• **Anemia**: vitamin B complex; foods such as red meats, liver, leafy greens, beets, yams, prunes, figs and raisins; herbs such as Angelica sinensis, Urtica, Rumex, Lycium

• **Lymphedema**: Bromelain, OPCs, Aesculus, Centella, Ceanothus, Echinacea, Phytolacca, Galium, Fucus

**KEY POINTS**

- Treat the person first, not the disease.
- Cancer is a complex condition with a myriad of different causes and interactions. It is important to address this multifactorial condition from many different aspects by addressing each stage, rather than everything at once.
- Keep supplementation and diet simple, and don’t overwhelm the patient with too many things to take, especially if they are also undergoing traditional treatment.
- Many nutritional and lifestyle factors can be involved with cancer and it is important to address the specific factors associated with the person as well as the type of cancer.
- Always address nutritional deficiencies and ensure a balanced body weight.
- Encourage a supportive environment and refer if required for psychological support.
- Flower essences can be very helpful for the emotional stresses associated with cancer. Often it is necessary to give other members of the family FE also. They are a major influence on the patient being treated and they are also facing dramatic trauma, sometimes even more than the person with the cancer.

**References**


Appendix

From Cancer: It's Causes, Symptoms and Treatment by Eli Jones

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The following is a list, taken from Jones’ text, describing a variety of therapies he found of use in the general treatment of cancer:

“**Phytolacca.** In phytolacca we have a remedy of undoubted remedial value in cancer of the breast. It is indicated when the breast is hard and painful and of a purple hue. The breast is "Hard as cheese." This drug may be given in doses of five drops of the tincture once in three hours. In 1869 I formulated a Compound Syrup of Phytolacca that would fulfill many indications, and with it cured my first case of cancer of the breast. It is especially indicated in cancer in old people or patients past the middle age. It is different from most alterative syrups for it does not take away the patient's appetite (owing to the excess of sugar in the syrup.) It keeps the stomach and bowels in a healthy condition, keeps up the appetite and strength. Many a time it has seemed to me to prolong the life of these old patients for the reasons given above. The formula is as follows:


The fluid extract of phytolacca must be made from the green root. It is called COMP. SYRUP PHYTOLACCA and will be referred to in other parts of this book.

**Strychnine sulphate.** In cancer, as I have said before, there is a weak discouraged feeling to the pulse; this is especially well marked in advanced cancer and where the patient has had X-Ray treatment, or one or more surgical operations. These are all a shock to the system and enfeeble the nerve power. This condition of the pulse calls for sulphate strychnine and it should be given in doses of one-thirtieth grain (homeopathic 30x) before meals and at bedtime.

**Double sulphide.** It is one of the essential elements of success in the treatment of cancer to have a good germicide, a remedy that will destroy the germs of cancer
and at the same time act as an antiseptic to the stomach and bowels. Dr. William H. Burgess, of East Chattanooga, Tenn., has prepared a combination tablet called "Double Sulphide" that meets all the above conditions. I have used them in hundreds of cases of cancer for six years and regard them as a valuable addition to our remedies for cancer. The formula is as follows: --


The indication for this tablet is a coated tongue with red papilla prominent and symptoms of indigestion. In advanced stages of this disease the tablet is especially indicated. Dose, one tablet once an hour for eight hours, then one tablet once in three hours or one after each meal and at bedtime.

**Hydrastis canadensis.** Hydrastis canadensis is indicated in cancer of the breast when a lancinating pain in the growth is the principal symptom, with a broad indented tongue, flatulence, constipation, and distress in the stomach after meals. The best results are obtained with the 3 X dilution. Dose of this preparation five drops once in three hours.

**Kali Mur 3dX.** When there is a white fur on the tongue and bunches in the breast that feel quite soft and are very tender so that the tenderness is the prominent symptom, and more especially in recent cases of cancer kali mur 3d X is the remedy needed given in doses of three tablets once in three hours.

**Calcarea Flouride 6th X.** In cancer of the breast when the tumor is of longer standing and has a hard, knotty feeling to it Calcarea flouride 6th X is indicated. Dose, three tablets once in three hours.

**Cancer Drops.** The cancer drops should be alternated with the above in doses of ten drops once in three hours. These drops are made by the following formula:

Tr. Phytolacca, Tr. Thuja, Tr. Baptisia, a. a. 3i. Mix. Sig. Ten drops once in three hours.
**Baryta Iodide 3d X.** In another form of cancer of the breast of much longer growth, or with the adenoid form of cancer, the iodide baryta 3d X should be given in doses of three tablets once in three hours. In cases calling for this remedy the cancer is not yet ulcerated, the growth feels hard, of a stony hardness. In some cases it feels like a hard rubber ball loose in the gland. It may or may not be painful or movable.

**Condurango.** This much vaunted "specific" for cancer by the regular school in the seventies has been long since laid upon the shelf by them because it would not cure all forms of cancer. It has become obsolete like one hundred other so-called "cures" for cancer that the regular school has claimed as a cure for cancer. Nevertheless condurango has its place among our remedial agents for cancer. It is indicated in cancer of the breast when there are ulcers in the corner of the mouth; also in cancer of the breast when there are cramping pains in the stomach. Use the following prescription:

Tr. Condurango, 3ii. Aqua, 3vi. Mix. Sig. Teaspoonful three times a day.

**Bellis perennis (English daisy).** In recent cases of tumors in the breast (of course I refer to cancerous tumors) when the growth is caused by some injury to the breast as a blow, a bite or a fall, Tr. bellis perennis is the remedy indicated. It should be prescribed in ten drop doses three times a day.

**Arsenic.** I have never used arsenic locally in the treatment of cancer, but in some cases I have used the Iodide of arsenic, also Fowler's solution, for the burning pain in cancer of the stomach, giving three drops once in three hours.

**Conium maculatum.** This remedy has been tested by me in several cases. It is indicated in cancer of the breast with indurations of stony hardness and stabbing pains, but I have never seen any reason for believing that it will check the growth of the cancer although it does often ease the pain. It should be given in the 3d X dilution. Dose, ten drops once in three hours.
**Echinacea.** This remedy has no effect upon the growth of the cancer but it may be used in the last stage of cancer to ease the pain.

Tr. Echinacea, 3ss. Aqua, 3vss. Mix. Sig. Teaspoonful once an hour.

I used over 100 pint bottles of Tr. echinacea in several cases of cancer to test it thoroughly so as to find out if it did have any curative properties. The above statement is the result of my experience.

**Corydalis formosa.** This agent is regarded by the old writers on eclectic and homeopathic materia medica as one of our best remedies for syphilis, and so I have found it in my practice. I am of the opinion that a remedy that really does have a curative effect upon old syphilitic cases will also have a curative effect upon cancer. This is certainly the case with phytolacca and so it is also with corydalis. The more pronounced the cancer cachexia the stronger the indication for this remedy. Dose, ten drops of the tincture three times a day.

**Thuja 30th X.** The practice of vaccination by the regular school, the poisoning of the blood of healthy innocent children with their filthy serum is a great cause of the rapid increase of cancer in every country where there is enforced vaccination. It is a blot upon our civilization, a disgrace to the medical profession, using a virus that does not protect the victim from smallpox but has crippled and killed many innocent children. In some cases of cancer you will find now and then a patient that does not respond to the action of your remedies as fast as they ought. A careful investigation of their previous history will develop the fact of one or more vaccinations that caused an eruption to come out on the body, and bunches in the neck and breast. This tells the story of vaccinosis, the poisoning of the blood by the filthy vaccine virus. Fortunately we have a remedy indicated in such conditions. It is Thuja 30th X. Give it in doses of three grains once a day.

**Asterias rubens.** When the patient with cancer of the breast is of the lymphatic temperament, flabby with red face and the cancer has a lancinating pain we find asterias
rubens indicated. Dose, ten drops of the 3d X dilution once in three hours.

**Lachesis.** In cancer of the breast when the growth has a purplish appearance, is open and fungoid and bleeds easily, dark decomposed blood, the pain and suffering in the breast are relieved by the bleeding, lachesis is the indicated remedy. Dose, ten drops of the 6th X dilution in half a glass of water and give a teaspoonful of this mixture every hour.

**Kresotum 3d X.** In cancer of the uterus when there is awful burning in the pelvis like red hot coals, with a discharge of foul smelling clots, kresotum 6th X is the needed remedy. Dose, give three tablets after each meal and at bedtime.

**Acetic acid, 1st X.** In cancer of the stomach the 1st X dilution of acetic acid is the only remedy we know that will dissolve the cancer cells. It may be given in doses of five drops once in four hours and should be kept constantly applied by means of a compress to the external surface of the stomach.

**Galium aparine.** In cancer of the tongue galium aparine is the remedy when it has the appearance of scirrhous formation with a nodulated feeling like a boy's marble embedded in the tongue. There is more or less induration of the tongue. Dose, twenty drops of the tincture four times a day. Use a mouth wash of the tincture, diluted one-half with water, once in two hours. Hold it in the mouth against the tongue for several minutes.

**Sempervivum tectorum.** When the tongue has ulcers, bleeds easily, especially at night, patient complains of much soreness of the tongue with stabbing pains, this remedy should be given. Dose, five drops of the 2d X dilution once in three hours. Apply locally the tincture of sempervivum diluted with two parts of water or as strong as the patient can bear it. This wash should be applied several times a day to the diseased surface.

**Sanguinaria nitrate.** In cancer of the tongue where we find an ulceration on the side of the tongue we apply nitrate of sanguinaria to the diseased part. Add one grain nitrate
sanguinaria to one drachm of glycerine. Pour out two or three drops of this mixture on a glass plate. Then take a glass rod and dip it in the drops on the plate and apply it to the ulcer after having first cleaned it with cotton. Do this three times a day.

**Chimaphila umbellata.** In women with quite large breasts with considerable of the gland effected by the cancer yet not ulcerated chimaphila is the needed remedy. Dose, tincture chimaphila twenty drops three times a day.

**Lapis albus.** In fibroid tumors with intense burning pains through the part with profuse hemorrhages lapis album 6th X is the remedy. Dose, three tablets every three hours.

**Cholesterinum 3d X.** For cancer of the liver, enlargement of the liver, patient complains of burning pain in side, has sallow skin, when he walks, holds his hand at his side, it hurts him so to walk, cholesterinum 3d X is the remedy. Dose, six grains once in four hours.

**Iodine.** In cancer with rapid emaciation, canine hunger, feels hungry all the time, feels relieved by eating, feels worse in a warm room, you should prescribe tincture iodine 6th X dilution. Dose, ten drops in a little water once in two hours. It is the remedy for cancer of the pancreas.

**Silicea 6th X.** In superficial cancer or in cancer in the glands where the discharge is thick, yellow, and offensive silicea 6th X is the remedy. Give three tablets three times a day.

**Kali sulph 3d X.** In small epithelioma on the face with scabs and a red angry appearance I have given kali sulph. 3d X, three tablets once in three hours, and applied as a salve, of kali sulph. one drachm to the ounce of vaseline, three times a day with good results.

**Cerate Phytolacca folium.** When you get a case of ulcerated cancer near the eye and there is considerable discharge from the surface and it seems inclined to spread you may apply cerate phytolacca folium, a cerate prepared by Boericke & Tafel, of Philadelphia. Spread it on soft white cloth and apply three times a day. The cerate contains
about 20 per cent of the juice of the leaves mixed with vaseline. It causes no pain, only a drawing sensation. It seems to dissolve off all the diseased surface and heals it up at the same time. The reader should remember that the whole plant (phytolacca) contains about forty per cent of caustic potash. That is the reason it acts so nicely in such cases. I have made some very fine cures with this remedy and I use about twenty-five pounds of it a year.

**Thuja.** Thuja is indicated in cauliflower cancer of the uterus, also in cancerous tumors of the rectum, and fungous growths.

**Apis mel.** Apis is indicated in cancer when the pain is of a burning, stinging character, in cancer of the breast with induration.

**Belladonna.** Belladonna is indicated in cancerous tumors of the breast when the pain is worse from lying down.

**Arsenic iodide.** Iodide of arsenic is indicated when the glands in the axillae are swollen, hard, size of a hen's egg, exuding a fluid that forms a brown crust. The tumor in the breast is indurated, painful and sensitive, sore to the touch.

**Nitric acid.** Nitric acid is indicated in painful swellings of the submaxillary glands of a scirrhus nature.”