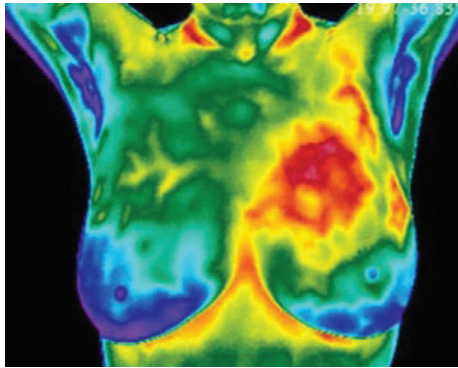


Inflammation is the Fuse that Ignites Cancer - Angiogenesis is the Pipeline that Fuels It

- An Important Aspect of Cancer Treatment

In Latin, the word “inflammation” means “I ignite, set alight” and like gasoline, that’s exactly what it does to cancer. A microenvironment of chronic inflammation can increase the risk of cancer, bolster chemotherapy resistance and turn on oncogenes, genes that can turn cells into tumors. Most importantly, inflammation promotes the spreading and mutating of cancer cells while continuing to push the mutations within the cancer cells’ development. Inflammation also enhances tumors ability to recruit blood supply (angiogenesis).



Unfortunately, inflammation and cancer signaling pathways are ignored for most cancers in the oncology world. Basically, inflammation is one of the leading factors that contributes to uncontrolled growth of cancers cells and spreading (metastasis). In this article we will explain helpful approaches to give cancer patients an edge in treatment and overall cancer planning. Uncovering and treating the cause of inflammation, rather than just treating the symptoms, is an important key when fighting cancer or chronic disease. To get to the root of the inflammation, we have to learn what causes inflammation and how to deal with it.

Inflammation is the body’s response to tissue damage, caused by physical injury, ischemic injury (caused by an insufficient supply of blood to an organ), infection, exposure to toxins, or other types of trauma. The body’s inflammatory response causes cellular changes and immune responses that result in repair of the damaged tissue and cellular proliferation at the site of the injured tissue. Inflammation can become chronic if the cause of the inflammation persists, or because of deregulation in the control mechanisms responsible for shut-

ting down the inflammation process. When these inflammatory responses become chronic, cell mutation and proliferation can result and often create an environment that is conducive to the development of cancer. This is often referred to as “the perfect storm.”

The Link Between Cancer and Inflammation

Despite popular belief, less than five percent of cancer is solely genetic (in the sense of being directly inherited by family members). Most cancers have a cause and those causes bring about chronic inflammation as part of the process. New research suggests an emerging link between infection, epigenetics and cancer. Changes catalyzed by

In biology, epigenetics is the study of cellular and physiological traits that are not caused by changes in the DNA sequence; Epigenetics describes the study of stable, long-term alterations in the transcriptional potential of a cell.

pathogenic inflammation can transform cells into cancerous tumors. According

to ScienceDirect.com, “Several types of inflammation—differing by cause, mechanism, outcome, and intensity—can promote cancer development and progression.” [1] A study by the Cancer Research Institute also agrees, saying, “Chronic inflammation plays a multifaceted role in carcinogenesis.” [2] Many cancers are linked to viruses or bacteria that promote reversible, epigenetic changes in the body’s cells. At minimum, 20 percent or more of cancers are linked to infectious disease, according to the Journal of American Medical Associates.

Some Well-Known Examples:

- Human Papillomavirus leads to cervical cancer.
- Hepatitis C leads to liver cancer.
- Epstein Barr leads to lymphoma.
- Herpes Virus Six leads to brain cancer.
- Helicobacter Pylori leads to stomach cancer.

We are thought to only have fully recognized about 13% of infections worldwide, making infection a bigger contributor than typically reported. These infections bring about changes and chronic inflammation as well. One thing anyone with chronic inflammation will tell you is that it causes heat. Abnormal body heat can also lead to thermogenesis and enhance metabolic spread of cancer during metastasis. The locations with the most metabolic hotspots may indicate the most common areas of cancer spread. This is seen in animal testing where various cancer images have been superimposed. Inflammation is known to cause other such changes in the microenvironment of cells. **Cells often undergo adaptive changes to survive stressful or toxic environments.** These adaptive changes can include: an increased expression of antioxidant enzymes; increased anaerobic respiration; and development of angiogenic factors. This adaptation is usually transient, however, and allows normal cells to survive only until the toxic condition is alleviated. That means it’s not enough to have a strategy to kill cancer cells – chronic inflammation needs to be blocked and stopped at its roots to prevent the cancer from mutating and spreading.

Inflammation Triggers DNA Damage, Epigenetics and Stage 4 Cancer

Inflammation triggers an immune response and alerts the body’s vascu-

lature to release inflammatory cells into a damaged tissue environment. The cellular activity involved in the inflammatory response can increase the production of reactive oxygen species (ROS), such as free radicals, and reactive nitrogen species (RNS). Cells are normally able to defend themselves against these two types of molecules. However, when production of these two types of highly reactive molecules is increased due to chronic inflammation, cells can no longer protect themselves, resulting in extensive damage to the essential enzymes involved in DNA repair, actual cell DNA muta-



tion, and mitochondrial damage. These various insults are linked to causes of cancer and often bring about epigenetic changes. Research suggests an emerging link between infection, epigenetics and cancer. Changes catalyzed by pathogenic inflammation can transform cells into cancerous tumors. Many cancers are linked to viruses/bacteria that promote reversible, epigenetic changes in the body's cells that lead to tumors. At minimum, 20 percent or more of cancers are linked to infectious disease according to the Journal of American Medical Associates. Moreover, the global medical community is probably only aware of an estimated 13 percent of infections that exist throughout the world. For this reason, it is likely that we shall find that infections play a far larger role in the cause of cancer than current estimates show.

The Inflammation Process and Stage 4 Cancer's Microenvironment

Inflammation is known to cause other such changes in the microenvironment of cells. Cells often undergo adaptive changes to survive stressful or toxic environments. These adaptive changes

can include: an increased expression of antioxidant enzymes, increased anaerobic respiration and development of angiogenic factors. This adaptation is usually transient, however, and allows normal cells to survive only until the toxic condition is alleviated. Even so, under conditions of prolonged stress, such as chronic inflammation, a mutation may actually "lock" in the cell, making these adaptive changes permanent. Not surprisingly, many of the cells and systems involved in inflammation (including abnormal cellular respiration and angiogenesis) are also found in a variety of tumors. In addition to DNA mutation, injuries to tissue may also cause increased cellular proliferation at the site of the injury. In such circumstances, sustained cellular proliferation may result from resultant chronic inflammation. When combined with the DNA mutations described above, enhanced proliferation can increase the number of cells at risk for mutations, leading to an environment that is conducive to the development of cancer.

Inflammation, Progression and Metastasis of Cancer

Inflammation is one major fuel that feeds the fire of stage 4 cancer growths and spread. The interaction between viruses, bacteria, environmental toxins (carcinogens) lead to DNA methylation and other changes in cellular metabolism. Inflammations from infections/toxins that can lead to cancer are major contributors in tumor genesis or progression. Patients often feel helpless, believing that their cancer was completely predetermined by their genetics, but there are options and likely sources of cancer. While genes may indicate a predisposition, they certainly do not dictate our fate.

The Important Role Oxygen Plays in Cancer Treatment

One of the most important things to remember about cancer is it is NOT a chemotherapy disease, it is NOT a radiation disease and it is not a Vitamin C disease. Cancer is actually a metabolic dysfunction tied to genetic mutations, and the first step in fighting it is on the metabolic level. Let's learn how oxygen plays a role in the development and treatment of cancer. Every cancer has a trigger: infections, chemical toxins or heavy metal toxins are a few of the

main ones. Early changes are seen through metabolic shifts that ultimately cause mutation, continually pushing genetic changes, growth and spread throughout the life of the cancer. Let's take a look at how changes in oxygen metabolism are some of the first metabolic signs of difficult cancers.

Oxygen's Important Role In Cell Metabolism and Cancer Growth

Cancer is a very difficult to understand disease and there are many misconceptions associated with it. But one of the main keys of understanding, treating and ultimately winning the raging war against cancer is none other than oxygen. Eighth on the periodic table, oxygen is responsible for the breathing of cells and are essential role in providing energy.[1] However, cancerous, mutated cells thrive in anaerobic, or oxygen-lacking environments. When growing,

Cancer is Fueled by Sugar and Destroyed by Oxygen

cancer cells show a change where they have lower levels of oxygen. This may stem from dysfunctions in the cell's mitochondria (known as cellular "factories" that play a major role in cell respiration). If these issues go unchecked, it leads to further complications and malfunctions in apoptosis (programmed cell death). You may remember from biology class, mitochondria have two main functions: energy creation and policing uncontrolled division of cells. Nobel Prize winner Dr. Otto Warburg famously hypothesized "**...the prime cause of cancer is the replacement of the respiration of oxygen in normal body cells by a fermentation of sugar,**" meaning, cancer is caused by a lack of oxygen. Today's modern cancer cell biology has shown he was on the right track as mitochondrial health and shifting to a more oxygen-rich environment may protect healthy cells and further neuter cancer cells. Furthermore, malignant, rapidly growing tumor cells typically have glycolytic rates up to 200 times higher than those of their normal tissues of origin. This means cancer has a much higher need for sugar than normal cells; this has been proven by the abnormally high level of insulin receptors found on all cancer cells. Because

cancer cells favor the lack of oxygen, they shift to glycolytic pathways; put simply they use glucose as their source of energy. Cancer cells average about 16 times more insulin receptor sites than normal cells. It's important to realize that the genetics of a cancer in its early stages and its late stages are completely different. This is what makes late-stage cancer so complex and difficult to treat – you're essentially trying to overcome these numerous advanced metabolic changes. Let's look at some of the gene pathways that make this issue even more compounded.

Angiogenesis and Genes That Fuel Cancer Growth

Angiogenesis is a normal, healthy cell process through which new blood vessels form from pre-existing vessels. However, **it's also the fuse which sets off unchecked growth, turning benign tumors into malignant steamrollers.** It's also what transitions the metabolism of the cancer, making it that much harder to kill. Hypoxia is when a portion of the body doesn't have adequate oxygen supply. Hypoxia-inducible factor 1-alpha, (HIF-1-alpha,) is a protein that is encoded by the HIF1A gene, playing an essential role in cellular and systemic responses to hypoxia. Cancer cells use this protein to grow their blood supply and spread. According to a study by the Liver Cancer Institute at Zhongshan Hospital and Shanghai Medical School in Shanghai, "HIF-1alpha in HCC [hepatocellular carcinoma, the most common form of liver cancer] plays an important role in predicting patient outcome. It may influence HCC biological behaviors and affect the tumor inflammation, angiogenesis and act in concert with the oncogene MYC [a gene found in many cancers]. Attaching importance to HIF-1alpha in HCC may improve the prognostic and therapeutic technique." [2] Epidermal Growth Factor Receptor (EGFR) is normally used to tell cells to grow. It is found in all cancer cells. However, EGFR over-expression has been linked to numerous cancers, such as lung, prostate, colon, breast, anal and others. This receptor is also associated with increased chemotherapy resistance, leading to tumors that are untreatable. Additionally, EGFR is linked to insulin, making it the metabolic gasoline that fuels changes and growth in the cell. This also links back to HIF-1 alpha. According to a study by the Department of

Pathology at the VU University Medical Centre in Amsterdam, "In invasive breast cancer, HIF-1alpha is associated with angiogenesis, and expression of growth factors [including] the receptor EGFR. Thus, agents targeting HIF-1 may combine different pathways of inhibiting breast cancer growth, including angiogenesis and growth factors." [3]

Discovery of Tumor M2-PK Proves Cancer Cells Shift From Oxygen to Glucose as Source of Energy

M2-PK (also known as PKM2) is an enzyme that is important in tumor metabolism, discovered in 2010 by Harvard Medical School. Tumor M2-PK helps cancer cells shift to greater glycolytic pathways. It is only found in cancer cells and not in normal healthy cells, making M2-PK an excellent marker for monitoring excelled growth or tracking improvement in treatment, depending if levels are high or low.

Reactive Oxygen Species and Chemotherapy

Chemotherapy and radiation therapy both rely on Reactive Oxygen Species (ROS) to work, augmenting ROS stress. ROS are essential toxic substances like hydrogen peroxide and others that can cause damage to cells in high concentrations. ROS are natural byproducts of the metabolism of oxygen, however, more resistant cancers actually produce their own antioxidants to fight these toxic substances. Earlier stage cancers do not appear to have the same defense mechanisms that are found in more resistant later stage cancers. This explains why chemotherapy and radiation therapy may not work in late-stage cancers. The answer may involve actually increasing ROS levels so therapy can kill cancer cells once again – this is the therapeutic aim of oxidative medicine, giving high doses of antioxidants and creating ROS instead of destroying it. Therefore, the dosing and delivery change the entire mechanism of action of integrative treatments. In this form of ROS, oxygen is what actually allows chemotherapy and radiation to work. Several types of DNA damage are caused by ROS-related oxidation. That is the goal of effective cancer treatment, to not only kill cancer cells but their genetics as well. In many cases, when oxidative therapy is combined with

correctly-tested chemotherapy you can improve overall treatment for patients. Everyone's metabolism is different and therefore, every cancer patient's tumor's metabolism is different. By using the oxygen metabolism and other signaling pathways like EGFR and M2-PK, doctors can find the specific metabolism and make the strongest push in their favor. To destroy cancer you must see cancer for what it is: a metabolic dysfunction pushing for constant genetic mutations, which aids its spread. The best part about these treatments is they are helpful for most, if not all cancers. Integrative medicine focusing on antioxidants, anti-inflammatory foods and nutritional supplements, holistic therapies that increase oxygen and decrease waste and inflammation, and controlling the body's alkalinity might help, please contact us today at (337) 896-4141 - LITEON Natural Health Center.

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