A REVIEW OF THE STUDY LINKING ORAL PATHOGENS AND BREAST CANCER

One five-year study by our Institute, involving more than 200 participants, led to the discovery of this cause of breast cancer, and, subsequently, to the methodology that will greatly reduce or eliminate breast-cancer deaths in the future. It was found in all research subjects, that specific and identifiable neurotoxic microorganisms that inhabit jawbone necrosis, root canal teeth, abscesses, dental caries, etc. are responsible for setting breast cancer into motion.

Another study involving more than 500 individuals, 200 of whom were in the study mentioned above, revealed that these same pathogens were involved in other cancers as well. The use of antibiotics appears to have no effect on these microorganisms due to the fact that they are initially harbored in areas of the mouth where there is unhealthy tissue that has little or no blood circulation. As the organisms multiply in the jawbone or other area of the mouth, such as a root canal tooth, they eventually find their way to tissue that has been damaged or traumatized, such as tissue in the breast that has been damaged from some activity or possible storage in fatty tissues of food-induced toxins. Since bacteria are programmed to attack unhealthy tissue, the dental or jawbone microorganisms find their way to these sites. The breast is one of the closest sites from the mouth and a likely area to have been damaged from some physical activity. In the overwhelming majority of cases, when the source of the pathogens was found on the right side of the jaw, the tumors appeared on the right side of the body, and vice versa. **In breast cancer, all cases in the study, with no exceptions, were on the same side of the body as the mouth pathology.** It is suspected that when the microorganisms find their target in the breast, be it a bruised area, bra irritation, or possibly a fat cell loaded with toxins, a cancer stem cell (recent Harvard studies have confirmed the existence of cancer stem cells) response to these neurotoxic organisms is turned on as a healing response, which stem cells are known to do, and the tumor cells begin to multiply. These oral neurotoxins can explain the presence of cancer stem cells that Harvard researchers have implicated in breast and central nervous system cancers and possibly in other cancers.

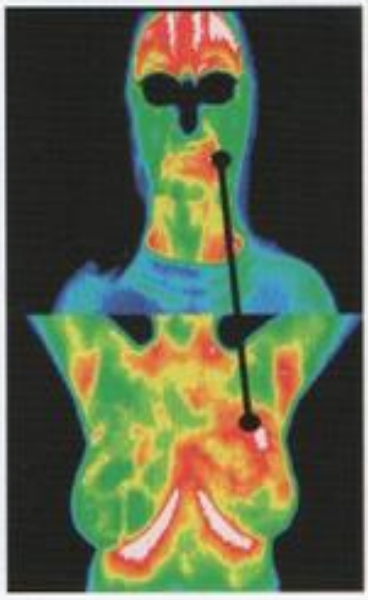
Our finding can also explain the issue of the return of breast cancer to those individuals who have had seemingly successful cancer treatments. It only makes sense that if the source of the cancer is still present, the cancer can reappear from a new infection of tissue in the breast, in the breast scar tissue after the breast is removed, or in some other area of the body.

It is interesting to note that the oral pathology found in our study, in most cases, is a silent disease. Without DITI, it can go unnoticed indefinitely, even by experienced dental professionals. Perhaps this is one reason doctors and scientists have had difficulty recognizing this condition.

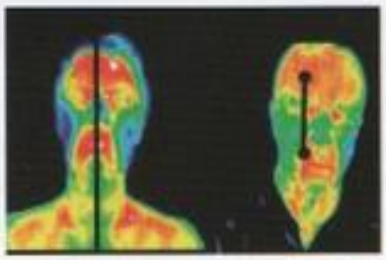
The series of infrared imaging photos that follow demonstrate the verified connection between an oral pathology and a cancer mass. The first photo demonstrates oral pathology in the upper front jaw area. Patient was found to have massive amounts of breast disease in both breasts and breasts were removed. However, the cancer had apparently metatasized to the backbone prior to the initial discovery of the primary site, which was the breast. Note the primary site and the metastasis sites were all in the midline directly below the verified dental pathology!

Noted Dental Pathology

The image below is of a female with a diagnosed breast tumor. Just above the breast tumor in the left breast is verified dental pathology in the left jaw. Traditional technology (mammography) had missed her tumor even though it was massive in size!



The image below is of a male with a diagnosed brain tumor. Just below the brain tumor is dental pathology in the right lower jaw. Again, the tumor and the verified oral pathology were on the same side of the body (black line).



Protein Studies Linking Oral Pathogens to Cancer

"If you want to find out how genes affect other genes, you have to find out how proteins affect other proteins."

— Roger Brent

"If you want to find out how proteins affect other proteins you have to study inhibition rates by chemical toxins."

— Robert J. Jones

It has become apparent that the real cause of cancer is genetic protein based. In other words, toxic inhibition of proteins within the cell structure allows or encourages a cell, or group of cells, to become malignant. The International Genome Project flatly states that **all cancers are genetic and are caused by one or more of three things:** (1) Radiation, (2) Chemical Toxins, and (3) Spontaneity.

Since we have no way to control or measure the rate of radiation exposure, his efforts were concentrated on measurable chemical toxins. I started on the conquest of exploring chemical toxicity and its relationship to protein inhibition several years ago (mid-late 1990s).

In the process of my research a huge amount of known carcinogens have been looked at. As we narrowed the search to a protocol of daily or chronic exposure, at the top of the list were compounds that are known carcinogens and neurotoxins. These toxins have been classified as thio ethers. The most specific of these thio ethers is dimethyl-sulfide. Although small in amounts of exposure

P53 AND ITS ASSOCIATES, P21, CDK2,P27,& METASTASIS OF CANCER

the average inhibition of ability to bind to the cellular membrane came to a startling conclusion of more than 90% inhibition as an average for all three proteins.

To distinguish the specific toxins, we tested 36 lanes on Affinity Labeling gels. Specifically, as we set the protocols for this research project, we used toxicity samples from over 900 extracted root canal teeth as a composite and over 4,000 bone fragments obtained from biopsy samples as a separate composite. Root canal toxins and cavitation toxins were tested separately to determine how each toxin individually inhibited the binding ability of the protein.

Establishing published cellular weights (amounts) of these proteins, we proceeded to inject Affinity Labeling gels with amounts of human protein as to the stated amount found in each individual cell. Therefore, using toxins extracted from human samples and human proteins, we were able to exhibit extreme or severe inhibition of these individual proteins by chronic exposure to these toxins. We then ran additional lanes on the same Affinity Labeling gels to determine the effects of glio toxins (fungi) and also the effects of mercury from dental amalgam.

As you will note during this presentation, the cavitation toxins from a composite of 100 or more cavitations was much more toxic than root canal toxins.

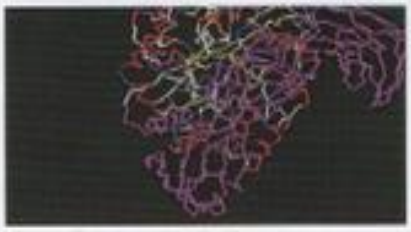
In our basic DNA, we have the chromosome ladder as illustrated in blue and protein in green with amino acids in yellow.

The inhibitions I will demonstrate will have serious effects on the protein's ability to function in the nucleus of the cell and the downstream effects of these inhibitions and how they affect the cellular functions will be noted.

The structure of the core domain of the p53 protein (light blue) bound to DNA (dark blue) The six most frequently mutated amino acids in human cancers are shown in yellow - all are residues important for p53 binding to DNA. Red ball: zinc atom. [Reproduced from Cho, Y., et al. (1994) Science, 265, 346-355, with kind permission.]

Protein Studies Linking Oral Pathogens to Cancer- *continued from page 4*

This is the G1S cell structure. We present this as published. All the proteins that make the nucleus of this oncogene cellular structure.



I'd like to talk to you about each protein individually. And about how each functions which has been established by the Science of

National Genome Research Project.
The Cancer Genome
Anatomy Project

The four proteins that we are going to look at in this presentation are P53, P21, CDK2 and then were going to look at a new publication from the University of Michigan published in the Journal of the National Cancer Institute. We will look at how the inability to bind P27 to P21 and demonstrate how it allows the cancers to metastasize.

P-53

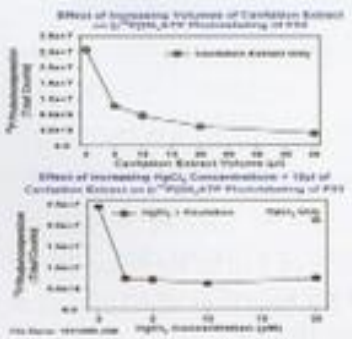
The anti-tumor protein and a major heart disease protein

What is P-53?

P53 is specifically a tumor suppressant protein. It has been defined as a normal function of P53 to be anti-oncogenic. While type P53 proteins introduced into cells were found to be growth suppressant. P53 is found rarely in a tumor cell, while it is very prolific in normal healthy cells. When it is found in a malignant tumor, it is found sparingly and in an inhibited state as we will demonstrate and therefore its ability to bind in the cellular membrane is greatly reduced. When P53 is normal or not inhibited, tumor growth or start is depressed.

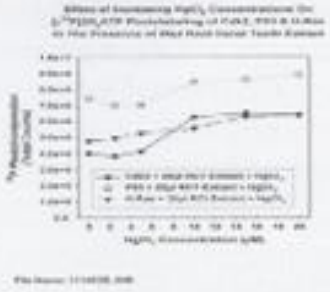
Amino acids are an important class of organic compounds that contain both the amino (_NH2) and carboxyl (_COOH) groups. Of these acids, 20 serve as the building blocks of proteins. These amino acids are inhibited from binding to the chromosome ladder and are just one of the examples of damage incurred by these oral/dental toxins.

This is the nucleotide protein before developing.



If you will note we have a graph that is printed out with a Hewlett Packard Phosphorescence measuring device as it measures radioactive light. This is non-subjective analyzation of inhibition of protein's ability to bind and function. To the left vertically is the scale of light reading. The bottom from left to right is the amount of toxin injected into each protein extract. They are shown in micro liters (ul).

Then we wanted to look at mercury amalgam vapors and what would they do to the inhibition aspects and we were surprised to find that mercury has very little effect in the binding ability to our DNA at this stage of toxicity. In fact, photo labeling increased very slightly when 10 ul added to the cavitation toxins. This is not to say that mercury by itself would not damage these proteins but did not provide a synergistic effect. So the condition was not exacerbated by the addition of the mercury.



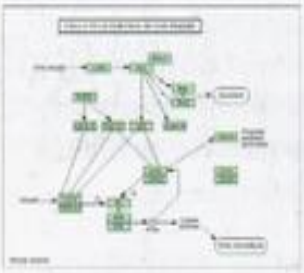
Now we are looking at a graph of mercury from amalgams added to the the Affinity Labeling gel. As you will note photo labeling of the protein P53 increased with increasing amounts of mercury. So mercury is not the cause of inhibition at this stage.

graph below where mercury from amalgams was added to the photo labeling gel. As you will note photo labeling of the protein P21 increased with increasing amounts of mercury. So mercury is not the inhibitor at this stage.

Now we have noted that all three proteins are greatly inhibited at even 5 ul from being able to function properly, and that we have produced three distinct markers for the start of cancer:

#1 – Inhibition of P53 due to these dental toxins is unable to suppress tumor start or growth.
#2 – Inhibition of P21 causes uncontrolled cell replication.
#3 – Inhibition of CDK2 creates uncontrolled cell growth.

These 3 markers positively identify the diagnosis of any cancer!



We want to present one more protein. This is not my research but it confirms my research. This research is from a team of scientists from the University of Michigan, published in the Journal of the National Cancer Institute. Their research into the GS1 pathway proteins as I have explored and presented in the past two years looked at the start of metastasis and prostate cancer and that P27 also known as RKIP1 or KIP1. When P27 is bound to P21 (H-Ras) it stops the migration of malignant cells into the vascular system and when P21 is inhibited from binding P27 it allows the start of metastasis not only on prostate cancer but on all malignant growths.

Conclusions of the Cancer Proteins

Inhibition of binding ability in all phases of GS1 of these three proteins expresses itself as the probable start of most if not all cancers. The chronic exposures of minute amounts of these toxins, which are also proven carcinogens, inhibit the binding ability of these proteins, which take on the form of a carcinogenic or mutant cell until the carcinogenic dose is reached. The inhibited forms of P21, P53, P27 and CDK2 cannot function in the glycolyzation, hydrolyzation and methylyzation pathways and exhibit other "downstream" effects such as production of free radicals that are introduced into the bloodstream, which can lead to the production of antibodies exhibited in other auto-immune diseases such as Lupus, Parkinson's, ALS and MS.

The Amazing DITI

Typically, DITI imaging of the breasts does not include the face or head. Since the early beginnings of Digital Infrared Imaging, it is estimated that more than 300,000 breast images have been performed globally. For research purposes, it has been unfortunate because of the obvious link between oral pathology and breast cancer that we have discovered. Perhaps if the early breast imaging had included the head, this link would have been discovered many years ago. When we began thermal imaging five years ago, we insisted that women have a full body imaging rather than just the breasts because we felt they were being cheated of the full benefits of DITI by not doing a full body scan. Because of our insistence on the full body scan, we were able to make this connection between oral pathology and breast cancer.

To help you better understand what we have discovered, a short explanation of how this technology works is in order.

Thermography measures differences in infrared heat emission from normal breast tissue, benign breast abnormalities—such as fibrocystic disease, cysts, infections and benign tumors—and from breast cancers. It does that with a high degree of sensitivity and accuracy. Breast thermography is a non-invasive measurement of the physiology of breast tissue. While breast cancer can only be diagnosed by tissue biopsy, breast thermography safely eliminates the need for most unnecessary biopsies, and it does so years sooner than any other test in modern medicine. With the recent verified discovery of the link between oral pathology and breast cancer, seeing a "hot spot" on the breast combined with a "hot spot" above it on the face helps us finally to have the upper hand on this disease. The statistics are grim and appear to be worsening each year. In the United States alone, there are 200,000 new cases of breast cancer and more than 40,000 deaths each year! With our verified discovery, we believe that the 40,000 deaths each year will be significantly reduced until such time, with screening of the masses, that death from breast cancer will be a thing of the past.

Today's high resolution digital Infrared Thermal Imaging has a thermal sensitivity of 0.05 degrees Centigrade. Because tumor tissue does not have an intact sympathetic nervous system, it cannot regulate heat loss. When the breast is cooled with small fans in a room kept at 68 degrees Fahrenheit, blood vessels of normal tissue respond by constricting to conserve heat while tumor tissue remains hot. Thus, tumors emit more heat than their surrounding tissues and are usually easily detected by heat-sensing infrared scanners. Over time, cancerous tissues stay hot or become even hotter—they do not cool down.

In sharp contrast, however, other possible conditions such as fibrocystic breasts, infections, and other benign disorders cool down as they resolve.

Advances in infrared technology, combined with data on 300,000 women with mammotherms, document that breast thermography is highly sensitive and accurate. Today, this means that more than 95% of breast cancers can be identified, and that this is done with 90-98% accuracy. In women under the age of 50, where there is the most devastating loss of life from breast cancer, mammography, MRIs and PET scans cannot come close to matching the combined sensitivity and specificity (accuracy) of breast thermography.

In conclusion, there is an abundance of scientific evidence supporting our claim that breast thermography is the most sensitive, sensible, accurate, comfortable, and inexpensive way to identify women with breast cancer. Breast thermography is clearly the most important adjunct to clinical breast examination.