VIRAL SCORE
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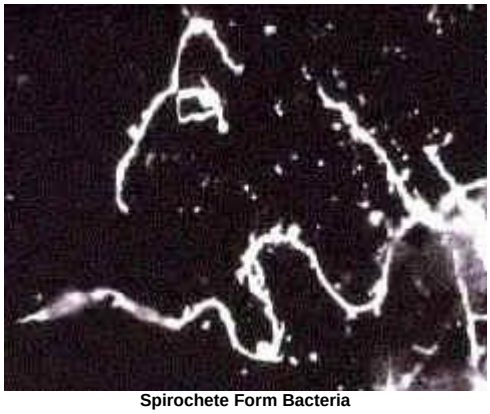
Understanding Lyme disease and Lyme disease as a causative factor in Cancer Links!

Borrelia Burgdorferi Cyst-Form Bacteria

Cell-Wall-Deficient Bacteria



Spirochete Form Bacteria

Borrelia Burgdorferi, the causative bacteria in Lyme Disease, is capable of transforming into three distinct bacterial forms: spirochete, cell-wall-deficient, and cyst. This transformation occurs for the purpose of bacterial survival and proliferation in the human body.

Each form has different characteristics and vulnerabilities.

The chart below summarizes the characteristics of each bacterial form, as well as their appropriate (and inappropriate) treatments.

When beginning any Lyme Disease alternative approach, it is imperative to understand Borrelia Burgdorferi and develop a alternative approach that targets not just the well-known spirochete form of the bacteria, but also the more elusive cyst and cell-wall-deficient forms.

Bacterial forms: characteristics and treatments

	Spirochete	Cell-Wall-Deficient (CWD)	Cyst
Primary bacterial activities and characteristics that facilitate survival	Very mobile. Spiral/drill capable shape allows penetration into dense tissue and bone. Capable of intracellular infection. Rapidly converts to CWD and cyst form when threatened.	Lack of cell wall makes targeting by immune system and antibiotics more difficult. Capable of intracellular infection. Converts Vitamin D to immunosuppressive hormone known as 1,25-D. Causes autoimmunity. Clumps together in dense colonies inner layers unreachable by antibiotics and immune system.	Dormant form bacteria are not mobile and do not cause symptoms. Can survive antibiotics, starvation, pH changes, hydrogen peroxide, temperature variation, and most other adverse conditions. Converts back to spirochete form when conditions are favorable.
Symptoms	Conventionally recognized Lyme Disease symptoms, i.e., bull's-eye rash, Bell's palsy, flu-like symptoms, fever.	Numerous syndromes and conditions not conventionally attributed to Lyme Disease, i.e., paralysis, multiple sclerosis, mental disorders, chronic fatigue syndrome, "post-Lyme syndrome," many more.	Does not cause symptoms
Common (yet not fully accurate) beliefs	Causative bacteria in Lyme Disease.	Not recognized or acknowledged; insignificant bacterial form.	Not recognized or acknowledged; insignificant bacterial form.
Correct beliefs	Causative bacteria in early-stage Lyme Disease, but not even close to the whole story.	Causative bacteria in many Lyme Disease symptoms and problems. As or more dangerous than spirochete form. Very difficult to treat.	Responsible for "relapsing and remitting" Lyme Disease. Can persist unrecognized and asymptomatic for many years.
Conventional treatment and success rates	Pharmaceutical antibiotics. Sometimes successful if infection is caught early. Often causes conversion to CWD and cyst form.	Often misdiagnosed as autoimmune or psychiatric disorders and mistreated with steroids, painkillers, antidepressants. Symptoms are often deemed idiopathic. Very low success rate.	Asymptomatic so conventionally not treated with anything. Frustration and confusion experienced by patient and practitioner upon inevitable relapses due to cysts.
Proper alternative approaches and success rates	Cold Laser therapy and a complete neurotoxin release protocol is the preferred alternative approach, and is the only known alternative approach that antiseptically kills spirochetes and does not induce conversion to CWD and cyst form. Highly successful.	ViralScore™ neurotoxin release protocol is a preferred alternative approach and is typically successful, often leading to remission of "incurable" diseases, ranging from autoimmune diseases to mental disorders to chronic fatigue syndrome.	Use the Immunecleanse formulation to strip the bio-film when spirochetes emerge from cysts. This allows a natural immune response to attack the spirochetes.

Cancer Viruses – Most labeled Lyme disease clients have the SV40 Virus

SV-40, a pro-cancer virus in vaccines

In 1955, Jonas Salk performed a medical miracle when he discovered how to mass produce polio vaccine by growing it on the kidneys of rhesus monkeys. While there is no question that thousands were saved from the ravages of polio by the Salk vaccine, by 1960 a problem had surfaced -- researchers had isolated a viral contaminate in the vaccine, Simian (monkey) Virus # 40. It seems that when the live polio virus grown on monkey tissues was extracted for vaccine production this SV-40 virus was extracted as well.

When SV-40 was injected into research animals it produced brain cancer. It appears our government didn't wish to create a public panic or discredit the public health service, because instead of recalling the tainted vaccines, it quietly ordered the manufacturers to find a monkey free of SV-40 and continue production. As of 1963, the rhesus monkey had been replaced with the African green monkey for production of a safer polio vaccine, but between the years of 1955 and 1963 as many as 98 million Americans had received doses of live polio virus vaccines tainted with SV-40.

Nowadays SV-40 has appeared in 61% of all new cancer patients -- patients too young to have received the contaminated vaccine being administered forty years ago who are now believed to have been infected by human to human transmission. Being a blood born organism, it is also suspected that SV-40 is transmissible from mother to child during pregnancy. The more this matter is researched the more startling the evidence. Senior epidemiologist at the National Institutes of Health, Dr. Howard Strickler, has plotted a geographic pattern to the cancers associated with SV-40 helping to confirm its link to the tainted vaccine. People who lived in Massachusetts and Illinois who received identified lot numbers of the contaminated vaccine administered in the 1950s are now demonstrating ten times the rate of the osteosarcoma bone tumors as those who received vaccine free of the SV-40 contaminate in other parts of the country.

DNA Polyoma Viruses

In 1964, studies were conducted on a polyoma virus (a tumor-producing DNA virus). It was discovered that the persistent genetic DNA material in the polyoma virus brought about malignant transformations in hamster embryo cell cultures. This was reported in the November 23, 1964 issue of the Journal of the American Medical Association.

SV-40 is one example of a DNA polyoma virus. Polyoma (many tumor-causing) viruses cause prolonged infection where tissue is destroyed, integrate into the hosts genetic material, are capable of mutating a cell, may reproduce after coming into contact with a 'helper' virus, enable the separate replication of the viral genome, can generate immune

responses, and they can induce malignancy. Scientists are amazed at how little genetic information these viruses carry in proportion to the damage they can cause.

The 'D' in DNA and the 'R' in RNA have characteristics which are dependent on the kind of sugar molecule associated with it. DNA exists predominantly in the nucleus, but is also represented in the cytoplasm and in the mitochondria. RNA is also present in the cytoplasm. When viral RNA or DNA combines with the genetic material in the cell itself, the viral genetic material can become part of the host cell genetic code, altering the genetic structure of the cell. When the altered cell duplicates, the encoded viral genetic material may affect cellular processes in such a way as to produce abnormal cells, which sometimes become malignant or cancerous.

Cancer-Causing RNA Viruses and DNA proviruses

The discovery in 1975 that viruses causing cancer in animals had a special enzyme called reverse transcriptase makes the problem even more interesting. These kind of viruses are called RNA viruses. When an RNA virus has the reverse transcriptase enzyme within its structure, it allows the virus to actually form strands of DNA which easily integrate with the DNA of the host cell which it infects. Studies by Dr. Robert Simpson of Rutgers University indicate that RNA viruses which do not cause cancer can also form DNA, even without the presence of reverse transcriptase. DNA formed in this way from an RNA virus is called a provirus. It is known that some non-cancerous viruses have a tendency to exist as proviruses for long periods of time in cells without causing any apparent disease. In other words, they remain latent. Some examples of common RNA viruses that do not cause cancer, per se, but have the capacity to form proviruses are influenza, measles, mumps and polio viruses.

Viruses as Catalysts for Cancer

An article in the January 6, 1962 Science Newsletter indicated that 'common human viruses act as carriers in causing cancer by interacting with cancer-causing chemicals; this has been indicated by experiments which show that cancer-causing substances that are present in too small a quantity by itself will become active and create tumors when combined with single doses of virus. Malignant tumors appeared in five type of injected mice.' The viruses mentioned were ECHO9, B-4, Coxsackie, and Polio virus 2. The article further indicated that 'viruses may also activate other cancer causing substances besides chemicals in the environment, such as DMBA, AF, and DBA.'

Even common non-tumor viruses, including those in smallpox vaccine and polio virus 2, can act as carcinogens. It was reported in Science on December 15, 1961 that these common viruses acted as catalysts in producing cancer when given to mice in combination with known organic carcinogens in amounts too small to induce tumors themselves. This means that some vaccinations will induce cancer, when combined with the growing problem of environmental pollution from toxic by-products of agriculture (pesticides on and in food) and industry.

A Listing of Cancer Causing Microbes

The July 14th 1997 issue of Business Week has an article in it about how many cancers are being linked to various viruses, bacteria, and parasites. Among the organisms now linked to cancer are as follows:

Microbe	Type of Cancer
Hepatitis B Virus	Liver Cancer
Human Papiloma Virus (HPV)	Cervical Cancer
Helicobacter Pylori	Stomach Cancer
HTLV-1	A type of Leukemia in Japan
Epstein- Barr virus (EBV)	Burketts Lymphoma, pharyngeal cancer
Kaposi's Sarcoma Herpes Virus (KSHV)	Kaposi's Sarcoma, and 100% of Myeloma cases.
Schistosomiasis	Bladder Cancer
Liver Flukes	Liver and biliary cancer
Helicobacter hepaticus	Liver cancer
Hepatitis C Virus	Liver cancer
Papillomaviruses (HPV-5,HPV-8,HPV-17)	Skin cancer
Polyomavirus (BK and JC)	Neural tumors? and insulinomas?
Retrovirus (HTLV-2)	Hairy-cell leukemia
Lyme disease bacteria B. Burgdorferi	Skin and Breast cancer
Epstein-Barr Virus	Majority of Non-Hogkins lymphoma (sp)
Granuloma type Virus	Skin Cancer (Not confirmed)

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More in depth info on RNA+DNA viruses and other types of viruses
Inherited virus may play role in breast cancer.

NEW YORK, Aug 12 (Reuters Health) -- An inherited virus may be one of the factors that triggers breast cancer in humans, researchers report. Scientists say that a primitive retrovirus, human mammary tumor virus (HMTV), has been identified in human breast cancer tissues. 'If a definitive link to this retrovirus is established, HMTV may become a target for a vaccine to prevent breast cancer and a target for new treatments for breast cancer,' explained study lead author Dr. Robert Garry of Tulane University in New Orleans, Louisiana.

A retrovirus similar to HMTV has already been linked to malignant breast tumors in mice. Speaking to attendees at the 11th International Congress of Virology in Sydney, Australia, Garry explained that vertebrate species other than mice -- including some humans -- carry similar viruses. He said that his team had identified the virus, dubbed HMTV, in breast cancer tissue and other organ issue from breast cancer patients, and also in tissues from individuals who did not have breast cancer.

The virus, he said, is likely to be 'a cofactor' for triggering breast cancer, along with other factors such as an individual's genetic makeup. Dr. Orli Etingin, an oncologist and assistant professor of medicine at New York Hospital/Cornell Medical Center in New York City, called the finding 'a very interesting new piece of the (cancer) puzzle.' Speaking with Reuters Health, she noted that 'retroviruses have (already) been implicated in certain kinds of lymphomas.' But she believes that 'a lot more research really has to be done in order to confirm the finding and also to establish what the relationship of the virus is to the development of tumors in humans.'

JC virus in the pathogenesis of colorectal cancer, an etiological agent or another component in a multistep process?

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Abstract

JCV infection occurs early in childhood and last throughout life. JCV has been associated to colorectal cancer and might contribute to the cancer phenotype by several mechanisms. Among JCV proteins, particularly two of them, large T-antigen and agnoprotein, can interfere with cell cycle control and genomic instability mechanisms, but other viral proteins might also contribute to the process. Part of viral DNA sequences are detected in carcinoma lesions, but less frequently in adenomas, and not in the normal surrounding tissue, suggesting they are integrated in the host cell genome and these integrations have been selected; in addition viral integration can cause a gene , or chromosomal damage. The inflammatory infiltration caused by a local chronic viral infection in the intestine can contribute to the selection and expansion of a tumor prone cell in a cytokine rich microenvironment. JCV may not be the cause of colorectal cancer, but it can be a relevant risk factor and able to facilitate progression at one or several stages in tumor progression. JCV transient effects might lead to selective expansion of tumor.