Hidden Infections - The Silver Edge

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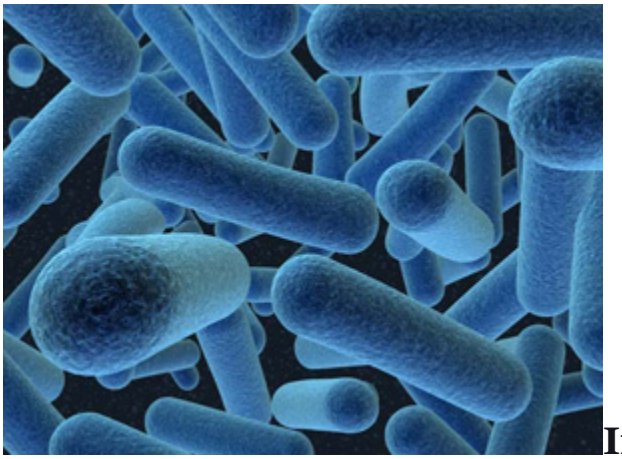

Infectious Microbes: the *Real* Culprits Behind Today's Most Debilitating Chronic Degenerative Diseases

A number of cutting edge medical doctors, researchers and scientists have slowly been coming to a startling conclusion: Many of today's most debilitating and sometimes deadly chronic degenerative diseases for which medical science cannot find an underlying cause, are actually being caused by hidden, long-term infections, or are otherwise being triggered by the long-term deleterious effects of pathogenic microorganisms on the human body.

This includes but is not limited to diseases such as cancer, heart disease and stroke, arthritis, multiple sclerosis (MS), lupus, fibromyalgia, chronic fatigue syndrome, Lou Gehrig's disease (ALS), diabetes, Alzheimer's, schizophrenia and other mental disorders, and *many* others.

You're Their Favorite Host

Pathogens always require a host. And the human body is one of the most convenient hosts of them all. And when pathogens `set up shop,' so to speak, in the human body, their #1 goal is to survive. To do so, they have a number of sneaky tricks in their survival repertoire. Some can crawl right into the cells of the human body and hide. Some can "hibernate" for decades, only to "awaken" when the body is weak and less able to defend itself. Some are *pleomorphic*, meaning they can literally change from one form into another and back again, as the situation for their survival requires. Some can even attach a piece of the human body to their own DNA much like putting on a mask or disguise — so that the immune system overlooks them even as they wreak havoc on the body.

Many of these insidious "stealth pathogens" work just below the level of immune system "awareness," ever so slowly and stealthily wearing down the body's defenses over long periods of time...hiding

when the immune system is triggered…and coming out to conquer more ground when the immune system has been weakened.

Well Worth Reading

Though it is unlikely you will ever hear about it from your doctor, the truth is, a great number of books have already been written on this fascinating subject. Many of the authors of these books have been reviled by the orthodox medical establishment for claiming that most, if not all, of the chronic degenerative diseases currently said to be rooted purely in genetics, environment or lifestyle, are actually being caused by bacterial, viral or fungal infections, and that much of what passes for "incurable" chronic degenerative disease can indeed now be cured or at least mitigated by either long-term antibiotic, anti-viral or anti-fungal treatments.

Fortunately, a small handful of these authors are now gaining a certain amount of notoriety even within orthodox medical circles, as the research behind their painstaking documentation becomes more widely known, and the power of their arguments becomes more widely accepted. Writers such as Professor Paul Ewald, Ph.D., a professor of biology at The University of Kentucky, and author of the controversial book *Plague Time: How Stealth Infections Cause Cancers, Heart Disease and Many Other Ailments*, have helped bring the concept of a microbial cause for most chronic degenerative diseases out of the darkness of medical disdain and into the spotlight. Ewald's impeccable scholarly credentials (he was the first recipient of the George E. Burch Fellowship in Theoretic Medicine and Affiliated Sciences), coupled with his persuasive documentation, has helped open the eyes of many other medical researchers who are now following his lead and blazing new trails in medical research.

Similarly, books like *The Virus Within: How Medical Detectives Are Tracking a Terrifying Virus that Hides in Almost All of Us* by long-time ABC World News Tonight and Nightline correspondent Nicholas Regush have helped bring mass public attention to the idea that hidden pathogens are in many cases wreaking havoc on our bodies, and that many of the diseases and disease complexes once thought to have "no known cause" are indeed being caused by microbial infections.

These popular works have in turn helped pave the way for new attention to be paid to some of the other outstanding books which have been published on this same subject over the course of the past two decades, but which were either ridiculed by the medical establishment, or completely overlooked when they first came out. Our favorites among the books on this subject published over the past few decades include:

Plague Time, by Professor Paul Ewald

The Virus Within, by Nicholas Regush

The Cancer Microbe, by Alan Cantwell, Jr. M.D.

Chlamydia pneumoniae Infection and Disease (Infectious Agents and Pathogenesis) by Herman

Friedman

Has Heart Disease Been Cured? By Douglass Mulhall and Katja Hansen

Four Women Against Cancer, by Alan Cantwell Jr., M.D.

The Persecution and Trial of Gaston Naessens, by Christopher Bird

Hidden Killers, by Erik Enby

Why Arthritis? by Harold W. Clark, Ph.D.

The Germ that Causes Cancer, by Doug Kaufmann

Infectious Diabetes, by Doug Kaufmann

The Fungal Link, by Doug Kaufmann

The Yeast Connection Handbook, by William G. Crook, M.D.

The Micro-Silver Bullet for Lyme Disease, AIDS Virus & Yeast Infection, by Dr. M. Paul Farber

How to Beat Multiple Sclerosis, by Nadine A. Wooley

Scleroderma: the Proven Therapy That Can Save Your Life, by Henry Scammell

The Arthritis Breakthrough (for RA, Lupus, Juvenile RA, Fibromyalgia, Scleroderma, Spondyloarthropathy and other forms of Arthritis), by Henry Scammell

Osler's Web: Inside the Labyrinth of the Chronic Fatigue Syndrome, by Hillary Johnson

These are just a few of the many great books which, taken together, help document the key role played by infectious microorganisms in almost every form of chronic degenerative disease known to man. We urge you to read these books, and see for yourself the clear evidence set forth within them.

In the meantime, here is a series of excerpts from articles that have been published in major medical publications, news sources, or on web sites around the world, discussing the vital topic of microbial infections and their direct relationship to chronic degenerative diseases such as cancer, heart disease, arthritis, diabetes, Alzheimer's, Multiple Sclerosis, lupus, Crohn's disease, Lou Gehrig's disease (ALS) and many others. Simply click on the links below to be taken directly to the article excerpts of your choice, or scroll down to view the articles one by one.

Click on a link below to go to the article of your choice...

Are Cancer, Heart Disease, Arthritis and Many Other Killer Diseases Secretly Being Caused by Hidden Infections?

What Makes You Sick? It Isn't What You Think

(Feature article from Popular Science magazine, 4-01; subscribe to Popular Science at 75% off the cover price: http://www.popsci.com/popsci/)

By Gunjan Sinha

Infectious microbes are to blame for cancer, heart disease, and most other ailments, says controversial biologist Paul Ewald.

On a blustery and frigid evening in early December, Professor Paul Ewald is huddled inside an auditorium with a group of 30 Amherst College students.

It's the last day of classes, and as part of their final project for "Seminar in Evolution," the students

are presenting data suggesting that almost every disease under the sun — including common killers such as heart disease and cancer — might be caused by bacteria, viruses, or other infectious organisms.

Ewald, an evolutionary biologist whose book "Plague Time" about this very subject had recently been released, sits attentively in the front row with his legs casually crossed…

One student points to the clustering of multiple sclerosis cases as evidence that the disease might be caused by something infectious. Another suggests the seasonal variation in births of autistic children is a reason to suspect that an infection during pregnancy induces the disease.

If I hadn't just spent the entire day with Ewald, I would have found the presentations almost laughable. Like most students of science, I was taught that statistical associations are soft science: They can't prove cause and effect. But that's exactly the kind of thinking that Ewald is out to dispel.

In "Plague Time," Ewald argues that the majority of chronic diseases — any disease that progresses gradually over time — are caused by infections. Just because you haven't proven cause and effect is no reason to ignore the data, he says.

To back up his arguments, Ewald goes beyond curious associations such as those found for multiple sclerosis and autism, and argues his case from an evolutionary point of view: Diseases such as cancer, heart disease, and even schizophrenia and obsessive-compulsive disorder are too common to be caused primarily by bad genes, Ewald claims.

Natural selection, he says, should have weeded those genes out of the population long ago. Instead, some genes might merely be making people more susceptible to infectious organisms, which are the true culprits of chronic disease …

But Ewald is more like a lone wolf than the leader of a pack. While some scientists, such as famed virologist Robert Gallo (the co-discoverer of the AIDS virus), agree in part with some of Ewald's ideas, others of equal stature are downright dismissive…

"Much of it is flagrant nonsense," says Robert Weinberg…a cancer researcher at the Whitehead Institute of Biomedical Research in Cambridge, Massachusetts…

As Weinberg points out, the average life expectancy of humans today is much higher than in the past. Until recently, most people didn't live long enough to get diseases like cancer and Alzheimer's. "Traditionally, human life was 30 or 40 years," Weinberg argues," and there was not any selective pressure against a genetic defect that manifested itself at the age of 60 or 70."

Ewald counters that there is no evidence proving that at least some of our ancestors didn't live to be old. "One must not look at average ages but rather at whether a substantial part of the population lived to be 60 or 70."

Besides, Ewald adds, similar skepticism was leveled against the now-famous Barry Marshall — an

Australian doctor who finally, despite much guffawing and finger-pointing from his peers, proved in 1984 that a bacterium called Helicobacter pylori causes most peptic ulcers.

Marshall's theory challenged widely held and seemingly unassailable notions that ulcers were primarily caused by stress.

[Worse than ridicule is the autoimmune disease disinformation campaign — perhaps sponsored by organizations who want to cut costs by denying treatment with antibiotics.

Any mammal would have a selective disadvantage if it tended to destroy itself when invaded by micro-organisms. The increasing number of people who are color blind to various degrees can be pointed to as an example of the survival of defects, but this does not apply to our response to parasites:

Humans have substantially reduced their chances of coming in contact with macro – predators such as tigers; and this security has been with us for at least 10,000 years — plenty of time for the effect to show itself of no penalty for not being able to distinguish green from yellow.

This is not the case with micro – predators. Polio, diphtheria, syphillis, tuberculosis, and HIV have wrecked havoc on us within the last hundred years. For every smallpox we put down, it seems that ten more species of pathogens rise in its place.

It was only in the 19th century that the germ theory of disease began to be accepted; and, even today, genetic tendencies and environmental stresses are the preferred explanations. In the 19th century, for example, doctors at the University of Vienna hospital had their collegue Semmelweis fired because he cut the maternity death rate of one mother for every eight admissions to one in thirty by having the staff on his ward wash their hands. (See "Plague Time" p. 17)]

However controversial, Ewald's ideas do have scientific legs to stand on. In the past few decades, a handful of cancers have been unquestionably linked to infections.

Take the case of cervical cancer and the human papilloma virus (HPV), for example. The Centers for Disease Control reports that the sexually transmitted virus is responsible for as much as 93 percent of all cases of cervical cancer.

But because most women infected with HPV never develop cervical cancer (the CDC estimates that 20 million Americans carry the virus), and the cancer takes years to kick in — characteristics very uncharacteristic of infectious disease — figuring out the connection took years.

"Over 100 years ago," Ewald explains, "people noticed that the frequency of cervical cancer was higher in prostitutes, and also that there were couples with penile and cervical cancer. But people didn't accept an infectious cause until the organism was identified over a century later."

Also pegged as cancer culprits are a herpes virus that causes Kaposi's sarcoma, a human T-cell leukemia virus (HTLV 1) responsible for a rare form of leukemia, and hepatitis viruses that cause liver cancer.

These and other viruses are responsible for between 15 and 20 percent of all cancers. The rest, say most scientists, can be blamed on rogue genes and non-infectious environmental factors such as diet and smoking.

Ewald, however, is convinced that evidence from other cancers, including those of the breast, will soon tip the scales in favor of infectious causes ...

Despite the lack of data proving a cause-and-effect relationship, scientists shouldn't disregard the possibility of an infectious cause, says Ewald.

Take the case of HTLV 1, which causes a rare form of leukemia prevalent in western Japan. The virus can be transmitted sexually or via mother's milk. People typically don't develop the cancer until 50 or 60 years after they've been infected, and some infected people never develop leukemia.

"It's not like chicken pox or some other organism that follows the established rules of infection," says Ewald, "and because it's transmitted in families, people could mistakenly assume it's hereditary."

Like HTLV 1, an infectious organism may be causing breast cancer but operating too cryptically to be detected with the tools available.

AIDS researcher Gallo, who also discovered HTLV 1, agrees: "Sure, there may be a breast cancer microbe, difficult to find, present rarely or occasionally, which in the right circumstances contributes to breast cancer, but that's still a very open issue."

Ewald argues that, from an evolutionary perspective, breast cancer is simply too common in the population for it to be caused by rogue genes.

His reasoning, backed up by mathematical calculations, goes something like this: Older people are still subject to natural selection after they stop reproducing because they pass on their care and wisdom to their children and grandchildren (as in some societies where grandmothers are responsible for preparing food). So a child without a grandparent will presumably be less "fit" than a child with a grandparent.

Over many generations, children with grandparents will prevail over children without grandparents (whose genes presumably made them more vulnerable to disease).

Ewald applies the same rationale to Alzheimer's and cardiovascular disease. Studies have shown that people with a gene called APO E4 appear to be more susceptible to both diseases.

But Ewald argues that a faulty gene is too prevalent (between 10 and 50 percent of a given population carries APO E4) to have been conserved through evolution, and that it is merely making people more susceptible to infection.

An airborne bacterium that infects the respiratory system, Chlamydia pneumoniae, may be one of the real culprits.

People with E4 are much more likely to be infected by Chlamydia, and several studies have confirmed the bug's presence in the fatty lesions associated with coronary artery disease.

But it's difficult to discern whether the organism initiates the disease, exacerbates the disease, or is even contributing at all.

[In "Plague Time", Professor Ewald, p. 109, writes concerning a Finnish study: "... 70 percent of the samples from heart attack patients had antibodies to Chlamydia [pneumoniae]. This percentage was significantly higher than the percentage in the control serums from people who had not had heart attacks."

Is it possible that all the deaths in recorded history from bubonic plague are miniscule compared to the people who are dying right now from heart disease due to chronic bacterial infection?]

Chlamydia's role in Alzheimer's disease is much more tenuous. In 1998, microbiologist Alan Hudson reported that he had found the bug in 22 out of 23 brains of Alzheimer's patients, whereas only one out of 25 normal brains tested positive.

"I couldn't believe my eyes, so I spent three years using every tool known to man confirming that the organism was really there, before going public with it," recalls Hudson.

Although Hudson and Ewald have become close collaborators, one can't help but wonder whether it was mutual professional interest that truly drew them together, or their shared experience of being treated like pariahs by the medical establishment.

A self-described "gene jockey," Hudson has spent his career studying how genes enable organisms to do what they do. He stumbled onto the Alzheimer's research through a former colleague when he was at the Philadelphia College of Osteopathic Medicine.

When Hudson submitted his Alzheimer's work to a journal for publication, the editors sent it out for peer review, as is standard practice among all journals. "The Chlamydia reviewers said `publish the thing,' the Alzheimer's people hated it," he says.

The journal initially rejected the paper, and it was only after some reviewers pressured the journal that they finally published it.

Hudson's experience is all too common in a field where arguments over scientific ideas can devolve into something akin to turf war ...

Just as the medical establishment ridiculed Barry Marshall and his germ theory of ulcers, says Ewald, scientists who have spent a lifetime studying the genetic basis of disease are of course going to reject alternative explanations. "They've simply got too much invested in existing theories," he argues.

Nevertheless, researchers are working to confirm Hudson's findings. As of this writing, a group based in the Netherlands has also found an association between Alzheimer's and C. pneumoniae, as has Chlamydia expert James Mahoney at McMaster University in Hamilton, Ontario.

One of the problems, says Hudson, is that identifying the organism is very difficult and the slightest deviation in established protocol can throw off results.

As for Ewald's evolutionary argument, most scientists agree that grandparents do contribute to fitness — the question is how much. Evolutionary arguments don't prove cause and effect.

To this, Ewald responds: "We always have to keep on the table the various hypotheses, even though we don't see direct evidence of them. One can never jump to the conclusion that infection is not important just because we find genes and environment are important."

"In most cases there's little evidence to exclude infectious causation of chronic disease. That kind of thinking has been responsible for a lot of death over the past 40 years."

Back to top

Does Alzheimer's Disease Have a Bacterial Link?

Alzheimer's Disease Related to Common Stomach Bacteria Disorder

Posted by: Dr. Kalish
www.mercola.com

September 26, 2006

Helicobacter pylori is a bacteria long associated with gastritis (inflammation of the stomach) and ulcers. Now scientists have further linked this common bacterial infection of the stomach to the development of Alzheimer's disease. Using histological analysis, the gold standard test for h. pylori infection, researchers determined:

46.7% of control group had h. pylori

88% of those subjects with Alzheimer's had h. pylori

In other words, h. pylori infection rates were nearly double in the group with Alzheimer's disease vs. their non-Alzheimer counterparts suggesting a relationship between this common stomach infection and the development of Alzheimer's disease.

Neurology. 2006 Mar 28;66(6):938-40.

Back to top

Could Amyloid-Producing Bacteria Be Causing Alzheimer's?

Bacteria produce fibers similar to those in Alzheimer's disease

By Edward R. Winstead

February 15, 2002

Common bacteria produce proteins that are remarkably similar to those found in the brains of Alzheimer's patients, according to a new study. Scientists found that E. coli bacteria generate protein fibers with the characteristics of amyloidsthe proteins that accumulate in the brain during debilitating human ailments such as Alzheimer's and prion diseases.

Bacteria use extracellular fibers, called curli, to colonize surfaces and mediate interactions with proteins in host cells. The discovery of bacterial amyloids gives researchers a new tool for investigating the details of how amyloids form in humans and for developing drugs to block their formation.

The discovery "also raises the intriguing possibility that bacterial amyloids could play a role in certain human neurodegenerative and amyloid-related diseases," the researchers write in Science. Scott J. Hultgren, of Washington University School of Medicine, St. Louis, Missouri, led the study.

The researchers used biochemical, biophysical, and imaging analyses to determine that the curli fibers produced by E. coli were in fact amyloid. They found differences as well as similarities between bacterial fibers and those associated with human disease. Amyloids in humans, for example, seem to assemble spontaneously, while bacteria have a specific machinery designed to assemble curli fibers.

[Chapman, M.R. et al. Role of Escherichia coli curli operons in directing amyloid fiber formation. Science 295, 851-855 (February 1, 2002).]

Back to top

Are Killer Microbes Causing Breast Cancer?

Suppressed and Forgotten Research Could Hold the Key to a Cure for this Dread Disease

Despite a century of cancer research the cause of breast cancer remains unknown. Age, diet, stress, hormone factors, genetic predisposition, and cancer viruses are all suspected as possible causative factors, but totally ignored are infectious bacteria which have been implicated in breast cancer and other forms of cancer.

A century ago when major diseases like tuberculosis, leprosy, and syphilis were discovered to be bacterial (not viral) infections, many physicians suspected bacteria might also cause cancer. At the close of the nineteenth century (when the science of microbiology was in its infancy), many different microbes were cultured from cancer. Variously called "cancer coccidia," "sporozoons" and "cancer parasites," a few of these microbes produced cancer tumours when injected into animals. But many did not, and most doctors finally assessed these cancer germs as laboratory "contaminants" or as "secondary invader microbes" that infect the tissue after the cancer is already formed.

The idea of a cancer parasite was finally dismissed in 1919 by noted American pathologist James

Ewing. In his popular textbook, Neoplastic Diseases, he declared: “Few competent observers consider it (the parasitic theory) as a possible explanation in cancer.” In Ewing’s opinion, cancer did not act like an infection. Therefore, he concluded that microbes couldn’t possibly cause it. He wrote: “The general facts of the genesis of tumours are strongly against the possibility of a parasitic origin.”1 Subsequently, few doctors dared to contradict Ewing by investigating bacteria in cancer.

Nevertheless, during the 1920s a few persistent physicians like pathologist John Nuzum of the University of Illinois College of Medicine; surgeon Michael Scott from Butte, Montana; and obstetrician James Young of Edinburgh, Scotland, continued to publish research showing that bacteria were implicated in breast cancer and other forms of cancer.

Working independently of one another, all three researchers cultured unusual bacteria from breast cancer, as well as from breast cancer tumours in mice. The peculiar growth of the “pleomorphic” cancer germ defied the established laws of microbiology by its ability to change shape and form, depending on how it was cultured in the laboratory, as well as the amount of oxygen supplied for growth and the age of the culture.

At first, the germ was barely visible as tiny round coccal forms. Later, these cocci enlarged into rod-shaped bacteria, which could connect together to form chains resembling a fungus. Small cocci could also enlarge into larger yeast and fungal-like spore forms.

Nuzum grew his “micrococcus” from 38 of 41 early breast cancers, and from the cancerous lymph nodes and metastatic tumours resulting from spread of the cancer to other parts of the body.2,3 During his 6 years of intensive bacteriological study, he learned the microbe could pass through a filter designed to hold back bacteria, indicating that some forms of the microbe were as small as the size of some viruses. With special stains he detected these small round coccoid forms within the breast cancer tumour cells. Although Nuzum couldn’t produce cancer tumours in mice, he was able to induce breast cancer tumours in 2 of 5 dogs injected with the microbe.

In a dangerous human experiment he injected the groin of a 70-year-old man with the bacteria he cultured from breast cancer. After 62 injections over an 18-week period, a skin cancer formed in the man’s groin. This experiment showed that breast cancer microbes were also capable of producing a different kind of cancer, such as skin cancer.

Young found his microbe in 16 cases of breast cancer, and in two mice with breast cancer. He identified “spore forms” and clumped “spore balls” in microscopic sections prepared from the mouse tumours.

Scott described three stages in the life cycle of his parasite: rod forms, spore or coccus-like forms, and large spore-sacs resembling a fungus.6,7 He treated cancer patients with an effective antiserum against these microbes, and spent the rest of his life trying to alert his colleagues to the infectious cause of cancer. But the antagonism of the medical profession to Scott’s cancer parasites and his antiserum was overwhelming, and he died a forgotten man.

During the last half of this century cancer microbe research was barely kept alive by a quartet of women, now all dead. The published research of Virginia Wuerthele-Caspe Livingston-Wheeler (a physician), Eleanor Alexander-Jackson (a microbiologist), Irene Diller (a cellular biologist) and Florence Seibert (a chemist) provides indisputable evidence that bacteria are implicated in cancer.

Livingston, who never let the male-dominated medical profession intimidate her, independently discovered the cancer microbe in the late 1940s and never stopped talking about it until her death in 1990, at the age of 84. Aided by Alexander-Jackson, who supplied the bacteriologic expertise, they became an unstoppable research team.8-12 The two women found a special stain (the acid-fast stain) that allowed the microbe to be recognised in culture and within the cancer tumour. Like the researchers back in the 1920s, they confirmed the microbe was filterable; and electron microscopic photos provided further proof that the filterable forms were indeed viral-size. Livingston named the microbe “Progenitor cryptocides” (Greek for the hidden-killer), which angered cancer experts, microbiologists, and American Cancer Society spokespersons, all of whom insisted the cancer microbe did not exist!

In the 1950s Irene Diller of the Institute for Cancer Research at Fox Chase, Philadelphia, discovered fungus-like microbes in cancer cells. Joining forces with the Livingston team, Diller worked with specially bred mice with a proven cancer incidence. By injecting them with microbes cultured from breast cancer and other tumours, she was able to more than double the cancer incidence of the mice.

She injected healthy animals with cancer bacteria. When cancer tumours developed she successfully cultured the microbe from the tumours – thus proving that these bacteria were implicated in the production of cancer. Utilising Livingston’s methods, Diller also grew the microbe from the blood of cancer patients.

In the early 1960s Florence Seibert became so impressed with Diller’s research that she quit retirement to help prove that bacteria cause cancer. Back in the 1920s Seibert devised a method to make intravenous transfusions safe by eliminating contaminating ubiquitous bacteria. Later, as one of the foremost authorities investigating the chemistry and immunology of the acid-fast bacteria that cause tuberculosis, she perfected the skin test for tuberculosis that has been used worldwide ever since. In 1938, she was awarded the famed Trudeau Medal, the highest prize given to tuberculosis research.

Experiments conducted by Seibert and her research team showed these acid-fast and TB-like cancer microbes were not laboratory contaminants because they were able to isolate bacteria from every piece of tumour (and every acute leukemic blood) they studied.

In her autobiography, Pebbles on the Hill of a Scientist, published privately in 1968, she wrote: “One of the most interesting properties of these bacteria is their great pleomorphism. For example, they readily change their shape from round cocci, to elongated rods, and even to thread-like filaments depending upon what medium they grow on and how long they grow. This may be one of the reasons

why they have been overlooked or considered to be heterogenous contaminants… And even more interesting than this is the fact that these bacteria have a filterable form in their life cycle; that is, that they can become so small that they pass through bacterial filters which hold back bacteria. This is what viruses do, and is one of the main criteria of a virus, separating them from bacteria. But the viruses also will not live on artificial media like these bacteria do. They need body tissue to grow on. Our filterable form, however, can be recovered again on ordinary artificial bacterial media and will grow on these. This should interest the virus workers very much and should cause them to ask themselves how many of the viruses may not be filterable forms of our bacteria."

Seibert's provocative papers, some emanating from the prestigious Annals of the New York Academy of Sciences, should have caused a stir. But with the quartet slowly closing in on the infectious cause of cancer, funds from previous supporters (like the American Cancer Society) suddenly dried up. All cancer microbe researchers eventually discovered that studying cancer bacteria was the kiss of death as far as funding was concerned. And without adequate funding, this type of cancer research was made more difficult.

But coming from thirty years of research into the acid-fast bacteria that cause tuberculosis, Seibert knew that the discovery of a pleomorphic and acid-fast microbe in cancer was tremendously important. She fervently believed that knowledge of this microbe would be instrumental in developing a possible vaccine and more effective antibiotic therapy against cancer. In Pebbles she confided: "It is very difficult to understand the lack of interest, instead of great enthusiasm, that should follow such results, a lack of certainty not in the tradition of good science. The contrast between the progress made in tuberculosis where we know the cause, where we have good general diagnostic tests, where we have a vaccine and effective antibiotic controls, and that made in cancer with the millions invested, is very striking. Some dedicated scientists should indeed find it rewarding to confirm or deny these painstaking and time-consuming experiments, for the sake of establishing the first necessary step in the important problem of the etiology of cancer."

Like the other women, Seibert observed the virus-like forms of the cancer microbe within the nucleus of the cancer cells. She theorised this infection could disrupt and transform nuclear genetic material that could lead to malignant change. Even though cancer microbes might appear to be simple and common microbes, their ability to infiltrate the nucleus of cells meant they were far from harmless.

In 1990, at the age of 92, Florence Seibert was inducted into the National Women's Hall of Fame, along with Barbara Jordan (Government), Billie Jean King (Athletics) and Margaret Bourke-White (Arts). When she died the following year her passage was noted in Time and People magazines, and in major newspapers like The Los Angeles Times. All the obituaries mentioned her contributions to the safety of intravenous fluids and her great achievement with the TB skin test. But not a word was written about her cancer microbe research, to which she devoted the last thirty years of her life.

Each year 190,000 American women are diagnosed with breast cancer. And the prognosis is still dismal for women whose breast cancer has spread to the lymph nodes and beyond. Yet the medical

establishment remains adamantly and irrationally opposed to cancer microbe research. It is perhaps understandable from an economic viewpoint that the medical profession would not welcome a proposed infectious cause of cancer that would challenge the highly lucrative multibillion-dollar cancer industry.

Physicians confidently ignore cancer bacteria because they have been carefully taught in medical school that there are no significant bacteria detectible in cancer. They still believe that cancer microbes represent contaminant bacteria or bacteria of no significance. Thus, published reports of cancer microbe research are rarely cited and the subject remains virtually unknown.

The idea of a microbe with virus, bacteria, and fungal-like stages is also anathema to most doctors. However, over the past several decades the study of cell-wall deficient bacteria and “mycoplasma-like” bacteria (which are both bacterial and viral-like) indicates that microbes indeed have a complex life cycle. In 1919, when Ewing offered his damning opinion of cancer parasites, none of these microbiologic peculiarities were even recognised!

In some instances, cancer microbe research appears to be deliberately suppressed. For example, the National Cancer Institute on its “cancer Facts” web page informs viewers about Virginia Livingston and states: “There is no scientific evidence to confirm her theories of cancer causation or to justify her treatments.” Obviously, this official judgement is a blatant lie because, as we have noted, Livingston’s discoveries have been confirmed by many competent scientists.

In addition, Livingston has written three books on the cancer microbe: Cancer: A New Breakthrough (1972), The Microbiology of Cancer (1977), and The Conquest of Cancer (1984).15-17 More recent books on bacteria in cancer include Alan Cantwell’s The Cancer Microbe (1990) and Can Bacteria Cause Cancer? (1997) by David J Hess.

Using acid-fast staining techniques, bacteria have been identified in breast cancer, lymphoma, Kaposi’s sarcoma (the so-called “gay cancer” of AIDS) and other forms of cancer.20-22 Figure 1 shows bacteria identified in breast cancer, indicating that such microbes are already present within the tumour and are not laboratory contaminants. Microbes have also been identified in “normal” and cancer-free breast tissue removed at the time of surgery. This suggests that the bacteria are not “secondary invaders” because they are identifiable in areas before the tissue has been invaded by cancer.20 Figure 2 shows the appearance of a microbe cultured from the same breast cancer. Note how the size and shape and appearance of the microbes within the tumour (Fig. 1) approximates the appearance of the bacteria cultured from the metatastic spread of the tumour to the skin (Fig. 2).

The current lack of knowledge about the cause of advanced breast cancer has resulted in the recommendation of some very expensive and death-defying treatments for this horrendous disease. Bone marrow transplants, which carry a 5% death rate, are being proposed as a routine treatment, at a minimal cost of $100,000 per patient.

As described in Karen Stabiner’s To Dance With the Devil: The New War on Breast Cancer (1997), the

procedure is not pretty.23 First, a catheter is placed in a woman's chest to deliver the drugs. A surgical treatment is then performed to scrape out bone marrow from her pelvis, followed by 7 days of growth hormone injections. Then starts days of intravenous chemotherapy that can cause kidney and bladder damage. A catheter is placed in the bladder, followed by a round of intravenous BCNU, or carmustine, a drug that makes a woman feel like she is falling down drunk. Patients become sleepy, sullen, disoriented, agitated, and angry. Loss of bowel control and vomiting are common. After all this, women are put into isolation because the white count drops precipitously, making her vulnerable to all sorts of infections. There may be inexplicable spiking fevers and rashes, and the inevitable loss of hair. After three weeks, patients are allowed to go home where they are told to watch for, "interstitial pneumonitis," a potentially fatal after-effect if not diagnosed and treated early.

Bone marrow transplant for breast cancer is not guaranteed, nor is it considered a cure. Women have been known to die of cancer three months after the procedure, proving that some patients do not respond to chemotherapy no matter how high the dose.

Even with radiation, chemotherapy and surgery, the cost of dying of cancer is not cheap. At the price patients are paying, physicians should not have the luxury of being ignorant about cancer microbe research, particularly when these microbes can be identified in cancer tumours.

With 40,000 American women dying annually from breast cancer, it is time medical science re-evaluated the parasite of cancer that James Ewing so casually dismissed in 1919. Perhaps if he hadn't been so adamant about cancer microbe research, his colleagues might have been able to do more to save him when he himself eventually died of "the Big C."

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(New Dawn No. 48 May-June 1998)

Back to top

Are Bacterial and Viruses Playing a Hidden Role in Just About Every Chronic Degenerative Disease Known to Man?

In a bold new book, evolutionist Paul Ewald argues that viruses and bacteria play a huge, hidden role in heart disease, cancer and other modern plagues

By Geoffrey Cowley, NEWSWEEK, 11-27-00

" Back in the 1880s, before tuberculosis had a known cause, experts attributed it to a combination of risk factors — things like depression, bad ventilation, insufficient food and "family predisposition". One standard textbook noted expansively that "the idea of infection being a cause... still prevails in the South of Europe".

FAST-FORWARD TO the 1980s, and you hear similar accounts of peptic ulcers. The highly touted risk factors were stress, smoking, alcohol and, of course, "genetic predisposition." Never mind that an Australian researcher named Barry Marshall was successfully giving himself ulcers by swilling beakers of bacteria — and curing them with antibiotics. The textbooks didn't even mention his work.

We now know that TB and ulcers are infectious conditions, caused by specific microbes and treatable with anti microbial drugs. Yet we're still laboring to explain most of our leading scourges — cancer, heart disease, mental illness, Alzheimer's– with long lists of risk factors.

In a compelling new book titled "Plague Time" (282 pages. Free Press $25), Amherst College biologist Paul Ewald argues that we're missing an obvious lesson here. Roughly translated: "It's the germs, stupid."

Though genes and lifestyle are no doubt important, Ewald says, the primary causes of today's "slow-burning plagues" are parasites –viruses, bacteria and other infectious microbes –whose long-term effects we have simply failed to recognize.

Ewald is not a virologist but a bold-minded evolutionist who, in past work, has created a whole new framework for thinking about infectious disease. To understand why microbes behave as they do, he considers their ecological incentives.

Cold viruses can't afford to be too virulent because they require mobile hosts. (A dying cold sufferer wouldn't get around enough to infect other people.)

Parasites that can survive outside their hosts don't have to be so considerate — especially if they can travel from host to host via mosquitoes or drinking water. A dying malaria sufferer is, if anything, preferable to a healthy one from the parasite's perspective. All the person has to do to spread infection is lie still and get bit.

In "Plague Time" he takes a similar approach. By his reasoning, our genes shouldn't cause much heart disease, Genes that impede our survival tend to die out over time, as their owners fail to reproduce.

By contrast, the parasites with the best tricks for exploiting us are the most likely to stay in the game. THERE IS NO QUESTION THAT VIRUSES AND BACTERIA CAN TAKE UP LONG-TERM RESIDENCE IN OUR BODIES. Some hide deep within our cells to avoid detection by the immune system, while others disguise themselves to resemble our own tissues.

We know the consequences can be serious. Suppose the immune system catches sight of a streptococcal bug that normally evades detection by masking itself as a heart cell. As the body attacks the invader, it may demolish the organ as well.

The question is whether these chronic infections are as pervasive as Ewald suspects. Some experts would scoff at the notion, but the recent findings are impressive. "Until the 1980s," he writes, "it was generally not appreciated that women who were suffering and dying from cervical cancer were the victims of a venereal disease epidemic".

Today it's undeniable. Epidemiologists have puzzled for more than a century over the link between sexual promiscuity and cervical cancer. But over the past 15 years, studies have revealed that human papillomaviruses, America's most common sexually transmitted pathogens, are present in some 93 percent of cervical tumors. Scientists have even identified the proteins that HPVs use to release the brakes on normal cell division.

TIP OF THE ICEBERG

Cervical cancer may be the tip of an iceberg. Less definitive studies have linked childhood strep infection to obsessive-compulsive disorder and Tourette's syndrome.

Traces of a virus that causes mammary cancer in mice have been recovered from human breast tumors.

Researchers in Japan and Germany have linked borna virus –a brain infection seen in horses, sheep and cats– to schizophrenia and bipolar disorder in people.

A growing body of evidence suggests that Chlamydia pneumoniae, a common respiratory bug, may play a key role in coronary artery disease, the leading cause of death throughout the Western world. Since 1988, researchers have consistently found the bacterium in clogged vessels but not in healthy ones. They've caused arterial lesions in rabbits by infecting them with the germ. They have even found hints that antibiotics can slow the progression of heart disease in infected patients.

As these connections are borne out, they could change medicine as profoundly during the 21st century as germ theory did in the 20th. The question is whether they'll get the attention they deserve.

As Ewald observes, "Those who control access to funding and the channels of scientific communication tend to be believers in the established views."

When Edward Jenner hit upon the notion of a smallpox vaccine in 1797, the Royal Society of London scolded him for risking his reputation on something "so much at variance with established knowledge, and withal so incredible."

When the Hungarian physician Ignaz Semmelweis figured out that physicians' unwashed hands were causing fatal infections among new mothers at the University of Vienna in the 1850s, he lost his own position there.

Though Barry Marshall first reported his findings on the infectious cause of ulcers in 1983, his peers ignored the discovery until 1990, when the National Enquirer got hold of the story and told the world. Let's hope the scientific community is less slow to notice this book. "

Back to top

Are Bacteria Playing a Major Role in Heart Disease?

Bacteria's Role in Heart Disease Discovered

Scientists believe they have discovered how a common bacterial infection can trigger heart disease – a controversial hypothesis that is generating enormous interest among heart researchers. The key finding, reported in this week's Science, is that chlamydia bacteria, which cause lung and eye infections and a sexually transmitted disease, have a protein on their outer coat that mimics a protein found in the heart muscle of mammals. Normally this molecular mimicry allows the bacterium to evade the chlamydia-infected individual's immune system. But sometimes the immune cells are not fooled; they mount an attack against the chlamydia germs that gets misdirected against heart cells as if they were the enemy. What we now see is that we don't have to have bacterial damage to the heart in order for bacteria to cause heart disease.

Chlamydia infections are very common; at least two out of three adults have antibody evidence of a past infection. Since heart disease is also common, it has been impossible to know from epidemiology alone if the two diseases were really linked. Boston University researchers reported earlier this month that British patients who had been treated with certain antibiotics were less likely to develop heart disease. Two large human studies are underway to see if antibiotic treatment will prevent heart attacks among individuals who have already survived one.

DR. MERCOLA COMMENT: It does appear that there is a strong link between heart disease and these infections. Antibiotic treatment would be one way to address the issue. I suspect the situation is very similar to the issue with rheumatoid arthritis. I have treated more than 2,500 patients with this illness

over the last ten years. (You can find my protocol at) However, I believe it is far better to boost the immune system with nutritional interventions. I believe that is why people who follow my diet recommendations seem to do better with the antibiotics. They do not generally experience a severe worsening of their symptoms and usually respond dramatically well to the treatment. I suspect that the situation is open to a more aggressive intervention of heart disease by screening individuals for antibodies to chlamydia and treating them with a low dose pulsed regimens similar to the one we use in rheumatoid arthritis. The Minocin in the protocol works wonderfully well for chlamydia.

(http://www.mercola.com Simply the best health newsletter on the internet. ED)

Back to top

Have Bacterial Been Linked Medically to Heart Disease And Stroke?

Bacterial Infections Linked to Heart Disease and Stroke

Common, chronic bacterial infections, including lung and urinary tract infections, as well as gum disease, may increase the risk of atherosclerosis, a build-up of fatty plaques in the arteries that could lead to heart attack, study findings suggest.

During the 5-year study, people with chronic bacterial infections were nearly three times more likely to develop new plaques in carotid arteries, which are the large arteries in the neck that deliver blood to the brain.

A buildup of fat in the neck arteries can increase the risk of stroke, and is a sign that heart arteries may be clogged as well.

But the researchers cautioned that widespread use of antibiotics to fight chronic infections — and hopefully prevent atherosclerosis — is not justified.

Previous research has suggested a link between infections and heart attacks, but few studies have examined the relationship between infections and atherosclerosis, which can lead to heart attack and stroke.

During the study, 41% of participants developed new plaques in their carotid arteries. People who had chronic infections were 2.78 times more likely to develop new plaques than people who did not have any infections.

But not all infections were linked to an increased risk of atherosclerosis. The study found that only bacterial infections, not infections caused by viruses like cytomegalovirus, the herpes zoster virus or hepatitis B or C, increased the risk of artery disease.

Researchers suspect that infection-related inflammation may play a role in the increased risk. Patients with infections who had high levels of inflammation tended to have a greater risk of atherosclerosis. Another possible explanation, according to the authors, is that bacterial infections

may trigger the immune system to turn against itself. This so-called autoimmune response may damage vessels, making it easier for fatty deposits to accumulate.

(Circulation February 27, 2001;103:1064-1070)

Back to top

Can Arthritis Be Caused by Hidden Infections?

Bacteria, Viruses Can Cause Infectious Arthritis

Most people are familiar with osteoarthritis the common “wear and tear” arthritis that occurs over years of musculoskeletal stress and injury, and the source of many of the aches and pains we attribute to aging. Another well-known ailment is rheumatoid arthritis, an autoimmune disease that usually affects several joints in the body at once. But there are actually about 100 types of arthritis and related disorders, many of which are caused by bacteria or viruses.

Arthritis is a condition in which the joints of the body become swollen, tender and inflamed. “With infectious arthritis, a bacterium, virus or other infection directly invades the joint and causes pus to form there,” says James J. Nocton, MD, Associate Professor of Pediatrics at the Medical College of Wisconsin. “While infectious arthritis itself is not contagious, the cause of the infection can be.” Dr. Nocton practices in the Division of Pediatric Rheumatology, Department of Pediatrics, at Children’s Hospital of Wisconsin, a Medical College affiliate.

Bacteria such as staphylococcus aureus (“staph”) and streptococcus (“strep”) are the most common causes of infectious arthritis. These bacteria can gain access to the body in various ways, for example through a cut or break in the skin or via an ear infection. The infectious agent travels through the bloodstream and settles in a joint, where the body’s own defense system the immune response fights against the intruder. The end result is inflammation.

Untreated Lyme disease, caused by a bacterium carried by the deer tick, can lead to infectious arthritis, as can untreated sexually transmitted diseases, particularly gonorrhea.

In addition to certain common bacteria, some viruses can also initiate arthritic disorders. Parvovirus, which causes the common childhood infection known as “fifth disease,” can cause infectious arthritis in children or adults. While illness from parvovirus generally resolves without treatment, anti-inflammatory medications may be prescribed to treat joint swelling and pain if infectious arthritis develops.

People whose immune systems are already compromised by health conditions such as diabetes or sickle cell anemia may be at even greater-than-average risk of developing infectious arthritis.

This article includes information from:
Medical College of Wisconsin Department of Pediatrics

Children's Hospital of Wisconsin, Pediatric Rheumatology

Back to top

Is Antibiotic Treatment for Rheumatoid Arthritis Effective?

From Carol & Richard Eustice, Your Guide to Arthritis
www.About.com

The History Of The Search For Cause And Cure

With no known cure for the more than 100 types of arthritis, treating and coping with the painful symptoms have become the core of concern for patients and physicians.

In the 1930's, a bacterial cause for rheumatoid arthritis was investigated but the research was short-lived except for distinct cases of acute infectious or septic arthritis. In 1939, the first real lead regarding an infectious cause for rheumatoid arthritis arose when mycoplasma, an atypical viral-like bacteria, was isolated from the exudate and tissue of rheumatic patients.

Investigators had already shown that mycoplasmas cause arthritis in mice, rats, chickens, goats, and cows.

They had found mycoplasmas in the genitourinary tracts of humans too, especially females.
In 1949 at the International Congress on Rheumatic Diseases the possible relationship between mycoplasmas and joint disease was reported. After obtaining one of the first National Institutes of Health (NIH) research grants in 1950, Thomas McPherson Brown, M.D. and colleagues at the arthritis research unit reported the following year that the rheumatoid disease mechanism was more of an immunologic reaction of antigen and antibody (with mycoplasma as the suspected antigen) rather than the infectious and transmissible type.

In 1955, the research unit reported that mycoplasmas, unlike bacteria and viruses could live in tissue cell cultures without destroying the tissue cells. To further support mycoplasmas as a causative agent/antigen, in 1964 a high incidence of mycoplasma antibodies in the blood of rheumatoid arthritis patients and lupus patients was found, indicating current or previous infection. Also recognized was a 4:1 higher incidence of mycoplasma antibodies in females suggesting a correlation with the higher incidences of rheumatoid arthritis in females.

Antibiotic Therapy

Efforts to demonstrate the effectiveness of tetracycline therapy were initiated and first reported over 40 years ago by Thomas McPherson Brown, M.D. Two weeks after Brown's death in 1989, NIH requested grant applications for the controlled clinical trials of tetracycline therapy for rheumatoid arthritis which he had been seeking. The preliminary results of the clinical trials, known now as MIRA

or Minocycline in Rheumatoid Arthritis, were promising and the NIH requested grant applications for studies of mycoplasma and other infectious agents as causes for rheumatoid diseases in 1993, and a pilot study for intravenous antibiotics for rheumatoid arthritis in 1994.

The result of the MIRA clinical trial stated, “Patients who suffer from mild to moderate RA now have the choice of another therapeutic agent. Not only did the antibiotic significantly reduce symptoms, but side effects were minimal and less severe than observed for most other common rheumatoid treatments”.

“Why Arthritis?”

Throughout the years, the theories that focus on mycoplasma as the responsible infectious agent and on tetracycline as the antibiotic treatment of choice have been hampered by lack of adequate funding for more research and from politics. “Why Arthritis?” by Harold W. Clark, Ph.D., one of Brown’s colleagues, assesses the rheumatoid diseases, decades of research, the search for a cure, and the frustration of researchers whose case for anti-mycoplasma therapy was overlooked for 40 years by the government and various arthritis organizations. Clark believes efforts were impeded because a safe, simple treatment threatens the medical establishment since patients would then require less medical intervention.

Many physicians remain skeptical and still do not suggest antibiotic treatment to their patients. The Arthritis Foundation was seemingly unimpressed even after antibiotic therapy was deemed as safe and effective. The foundation’s medical director reportedly said he did not view the treatment as a breakthrough and more study of dosages and long-term use of minocycline is needed.

According to the American College of Rheumatology, “Minocycline is prescribed for patients with symptoms of mild rheumatoid arthritis. It is sometimes combined with other medications to treat patients with persistent symptoms of this form of arthritis.”

Back to top

Are Mycoplasmas Bacterial Infections a Cause of Asthma?

ASTHMA ASSOCIATED WITH BACTERIAL INFECTION
From: Respiratory Reviews.com

The latest clinical information on respiratory medicine

Vol. 6 No. 7 July, 2001

DENVER Increasingly, microbes appear to be involved in the etiology of some cases of asthma. In addition to previous findings indicating that viral infection may exacerbate acute asthma, emerging evidence now implicates bacterial infection as a cause of chronic asthma. Richard Martin, MD, and colleagues report detecting infection with Mycoplasma pneumoniae or Chlamydia pneumoniae in 31

of 55 asthma patients using a combination of polymerase chain reaction (PCR), serology, and culture.[1] By contrast, PCR revealed mycoplasma infection in only one of 11 normal controls.

“Given that we detected infection in 56% of asthma patients [vs one in 11 control subjects], there may indeed be a link between bacteria in the airways and asthma in some patients,” said Dr. Martin, head of the Pulmonary Division and Vice Chair of the Department of Medicine at National Jewish Medical and Research Center (NJMRC) in Denver. The researchers did not, however, find an association between chronic stable asthma and viral infection.

“We were surprised to find these bacteria in the lower airways of a subset of stable asthmatics,” said coauthor Monica Kraft, MD, Associate Professor in the Department of Medicine and Division of Pulmonary Medicine at NJMRC. “The association raises an interesting `chicken versus egg’ issuedid the asthma allow microorganisms to `set up shop,’ or do the microorganisms actually cause chronic asthma?” Dr. Kraft told RESPIRATORY REVIEWS. If the latter is true, then antibiotics would be expected to help some patients with asthma.

LONG-TERM ANTIBIOTIC THERAPY?

The authors have garnered support for this idea from both clinical experience and research. “Using an empirical approach, we’ve tried clarithromycin with some of our clinical patients. Anecdotally speaking, we have steroid-dependent asthmatics who improved with clarithromycin,” said Dr. Kraft. “Initially, we tried a six-week course, but we found that respiratory function continued to improve in many patients [if the antibiotic was administered] over three to six months.”

Dr. Martin explained the reason for the lengthy course: “Unfortunately, mycoplasma is difficult to eradicate from the airways. In true pneumonias, even after chest X-rays show that mycoplasma infection is largely cleared from the lungs, some residual infection lingers.”

Dr. Martin and colleagues also back up their clinical experience with experimental evidence. “We’re submitting a manuscript shortly, describing a study demonstrating that asthmatics who are PCR-positive for chlamydia or mycoplasma infection show a 12% to 13% improvement in airway function following clarithromycin treatment,” he reported. Said Dr. Kraft, “Most antibiotics do have some anti-inflammatory quality, so it’s not clear how this works. Hopefully, animal studies will answer this question.”

While the researchers are encouraged by clinical and experimental success with antibiotic therapy in a subset of asthma patients, Dr. Kraft recognizes that long-term antibiotic treatment for asthma may be controversial. In light of concerns regarding general overuse of antibiotics and need to justify long-term administration under managed health care, this approach might be questioned. However, in the absence of a practical means of establishing the diagnosis, she said, “We haven’t found any other way.”

Reference

1. Martin RJ, Kraft M, Chu HW, et al. A link between chronic asthma and chronic infection. *J Allergy Clin Immunol.* 2001;107:595-601.

Back to top

Are Bacteria from Root Canals Secretly Causing Degenerative Diseases of the Heart, Eyes, Lungs, Kidneys, and other Organs?

An Interview with George Meinig, D.D.S.

Dr. Meinig brings a most curious perspective to an expose of latent dangers of root canal therapy – fifty years ago he was one of the founders of the American Association of Endodontists (root canal specialists)! So he's filled his share of root canals. And when he wasn't filling canals himself, he was teaching the technique to dentists across the country at weekend seminars and clinics.

About two years ago, having recently retired, he decided to read all 1174 pages of the detailed research of Dr. Weston Price, (D.D.S). Dr. Meinig was startled and shocked. Here was valid documentation of systemic illnesses resulting from latent infections lingering in filled roots. He has since written a book, Root Canal Cover Up Exposed – Many Illnesses Result", and is devoting himself to radio, TV, and personal appearances before groups in an attempt to blow the whistle and alert the public.

MJ Please explain what the problem is with root canal therapy.

GM First, let me note that my book is based on Dr. Weston Price's twenty-five years of careful, impeccable research. He led a 60-man team of researchers whose findings – suppressed until now rank right up there with the greatest medical discoveries of all time. This is not the usual medical story of a prolonged search for the difficult-to-find causative agent of some devastating disease. Rather, it's the story of how a "cast of millions" (of bacteria) become entrenched inside the structure of teeth and end up causing the largest number of diseases ever traced to a single source.

MJ What diseases? Can you give us some examples?

GM Yes, a high percentage of chronic degenerative diseases can originate from root filled teeth. The most frequent were heart and circulatory diseases and he found 16 different causative agents for these. The next most common diseases were those of the joints, arthritis and rheumatism. In third place – but almost tied for second – were diseases of the brain and nervous system. After that, any disease you can name might (and in some cases has) come from root filled teeth.

Let me tell you about the research itself. Dr. Price undertook his investigations in 1900. He continued until 1925, and published his work in two volumes in 1923. In 1915 the National Dental Association (which changed its name a few years later to The American Dental Association) was so impressed with his work that they appointed Dr. Price their first Research Director. His Advisory Board read like a Who's Who in medicine and dentistry for that era. They represented the fields of bacteriology, pathology, rheumatology, surgery, chemistry, and cardiology.

At one point in his writings Dr. Price made this observation: “Dr. Frank Billings (M.D.), probably more than any other American internist, is due credit for the early recognition of the importance of streptococcal focal infections in systemic involvements.”

What’s really unfortunate here is that very valuable information was covered up and totally buried some 70 years ago by a minority group of autocratic doctors who just didn’t believe or couldn’t grasp – the focal infection theory.

MJ What is the “focal infection” theory?

GM This states that germs from a central focal infection – such as teeth, teeth roots, inflamed gum tissues, or maybe tonsils – metastasize to hearts, eyes, lungs, kidneys, or other organs, glands and tissues, establishing new areas of the same infection. Hardly theory any more, this has been proven and demonstrated many times over. It’s 100% accepted today. But it was revolutionary thinking during World War I days, and the early 1920’s!

Today, both patients and physicians have been “brain washed” to think that infections are less serious because we now have antibiotics. Well, yes and no. In the case of root-filled teeth, the no longer-living tooth lacks a blood supply to its interior. So circulating antibiotics don’t faze the bacteria living there because they can’t get at them.

MJ You’re assuming that ALL root-filled teeth harbor bacteria and/or other infective agents?

GM Yes. No matter what material or technique is used – and this is just as true today – the root filling shrinks minutely, perhaps microscopically. Further and this is key – the bulk of solid appearing teeth, called the dentin, actually consists of miles of tiny tubules. Microscopic organisms lurking in the maze of tubules simply migrate into the interior of the tooth and set up housekeeping. A filled root seems to be a favorite spot to start a new colony.

One of the things that makes this difficult to understand is that large, relatively harmless bacteria common to the mouth, change and adapt to new conditions. They shrink in size to fit the cramped quarters and even learn how to exist (and thrive!) on very little food. Those that need oxygen mutate and become able to get along without it. In the process of adaptation these formerly friendly “normal” organisms become pathogenic (capable of producing disease) and more virulent (stronger) and they produce much more potent toxins.

Today’s bacteriologists are confirming the discoveries of the Price team of bacteriologists. Both isolated in root canals the same strains of streptococcus, staphylococcus and spirochetes.

MJ Is everyone who has ever had a root canal filled made ill by it?

GM No. We believe now that every root canal filling does leak and bacteria do invade the structure. But the variable factor is the strength of the person’s immune system. Some healthy people are able to control the germs that escape from their teeth into other areas of the body. We think this happens because their immune system lymphocytes (white blood cells) and other disease fighters aren’t

constantly compromised by other ailments. In other words, they are able to prevent those new colonies from taking hold in other tissues throughout the body. But over time, most people with root filled teeth do seem to develop some kinds of systemic symptoms they didn't have before.

MJ It's really difficult to grasp that bacteria are imbedded deep in the structure of seemingly-hard, solid looking teeth.

GM I know. Physicians and dentists have that same problem, too. You really have to visualize the tooth structure – all of those microscopic tubules running through the dentin. In a healthy tooth, those tubules transport a fluid that carries nourishment to the inside. For perspective, if the tubules of a front single-root tooth, were stretched out on the ground they'd stretch for three miles!

A root filled tooth no longer has any fluid circulating through it, but the maze of tubules remains. The anaerobic bacteria that live there seem remarkably safe from antibiotics. The bacteria can migrate out into surrounding tissue where they can "hitch hike" to other locations in the body via the bloodstream. The new location can be any organ or gland or tissue, and the new colony will be the next focus of infection in a body plagued by recurrent or chronic infections.

All of the "building up" done to try to enhance the patient's ability to fight infections – to strengthen their immune system – is only a holding action. Many patients won't be well until the source of infection – the root canal tooth – is removed.

MJ I don't doubt what you're saying, but can you tell us more about how Dr. Price could be sure that arthritis or other systemic conditions and illnesses really originated in the teeth – or in a single tooth?

GM Yes. Many investigations start with the researcher just being curious about something – and then being scientifically careful enough to discover an answer, and then prove it's so, many times over. Dr. Price's first case is very well documented. He removed an infected tooth from a woman who suffered from severe arthritis. As soon as he finished with the patient, he implanted the tooth beneath the skin of a healthy rabbit. Within 48 hours the rabbit was crippled with arthritis!

Further, once the tooth was removed the patient's arthritis improved dramatically. This clearly suggested that the presence of the infected tooth was a causative agent for both that patient's and the rabbit's – arthritis.

[Editor's Note – Here's the story of that first patient from Dr. Meinig's book: "(Dr. Price) had a sense that, even when (root canal therapy) appeared successful, teeth containing root fillings remained infected. That thought kept prying on his mind, haunting him each time a patient consulted him for relief from some severe debilitating disease for which the medical profession could find no answer. Then one day while treating a woman who had been confined to a wheelchair for six years from severe arthritis, he recalled how bacterial cultures were taken from patients who were ill and then inoculated into animals in an effort to reproduce the disease and test the effectiveness of drugs on the disease.

With this thought in mind, although her (root filled) tooth looked fine, he advised this arthritic

patient, to have it extracted. He told her he was going to find out what it was about this root filled tooth that was responsible for her suffering. “All dentists know that sometimes arthritis and other illnesses clear up if bad teeth are extracted. However, in this case, all of her teeth appeared in satisfactory condition and the one containing this root canal filling showed no evidence or symptoms of infection. Besides, it looked normal on x-ray pictures.

“Immediately after Dr. Price extracted the tooth he dismissed the patient and embedded her tooth under the skin of a rabbit. In two days the rabbit developed the same kind of crippling arthritis as the patient – and in ten days it died.

“..The patient made a successful recovery after the tooth’s removal! She could then walk without a cane and could even do fine needlework again. That success led Dr. Price to advise other patients, afflicted with a wide variety of treatment defying illnesses, to have any root filled teeth out.”]

In the years that followed, he repeated this procedure many hundreds of times. He later implanted only a portion of the tooth to see if that produced the same results. It did. He then dried the tooth, ground it into powder and injected a tiny bit into several rabbits. Same results, this time producing the same symptoms in multiple animals.

Dr. Price eventually grew cultures of the bacteria and injected them into the animals. Then he went a step further. He put the solution containing the bacteria through a filter small enough to catch the bacteria. So when he injected the resulting liquid it was free of any infecting bacteria. Did the test animals develop the illness? Yes. The only explanation was that the liquid had to contain toxins from the bacteria, and the toxins were also capable of causing disease.

Dr. Price became curious about which was the more potent infective agent, the bacteria or the toxin. He repeated that last experiment, injecting half the animals with the toxin-containing liquid and half of them with the bacteria from the filter. Both groups became ill, but the group injected with the toxins got sicker and died sooner than the bacteria injected animals.

MJ That’s amazing. Did the rabbits always develop the same disease the patient had?

GM Mostly, yes. If the patient had heart disease the rabbit got heart disease. If the patient had kidney disease the rabbit got kidney disease, and so on. Only occasionally did a rabbit develop a different disease – and then the pathology would be quite similar, in a different location.

MJ If extraction proves necessary for anyone reading this, do you want to summarize what’s special about the extraction technique?

GM Just pulling the tooth is not enough when removal proves necessary. Dr. Price found bacteria in the tissues and bone just adjacent to the tooth’s root. So we now recommend slow-speed drilling with a burr, to remove one millimeter of the entire bony socket. The purpose is to remove the periodontal ligament (which is always infected with toxins produced by streptococcus bacteria living in the dentin tubules) and the first millimeter of bone that lines the socket (which is usually infected).

There's a whole protocol involved, including irrigating with sterile saline to assure removal of the contaminated bone chips, and treating the socket to stimulate and encourage infection-free healing. I describe the procedure in detail, step by step, in my book [pages 185 and 186].

MJ Perhaps we should back up and talk about oral health – to PREVENT needing an extraction. Caries or inflamed gums seem much more common than root canals. Do they pose any threat?

GM Yes, they absolutely do. But let me point out that we can't talk about oral health apart from total health. The problem is that patients and dentists alike haven't come around to seeing that dental caries reflect systemic – meaning "whole body" – illness. Dentists have learned to restore teeth so expertly that both they and their patients have come to regard tooth decay as a trivial matter. It isn't.

Small cavities too often become big cavities. Big cavities too often lead to further destruction and the eventual need for root canal treatment.

MJ Then talk to us about prevention.

GM The only scientific way to prevent tooth decay is through diet and nutrition. Dr. Ralph Steinman did some outstanding, landmark research at Loma Linda University. He injected a glucose solutioninto mice – into their bodies, so the glucose didn't even touch their teeth. Then he observed the teeth for any changes. What he found was truly astonishing. The glucose reversed the normal flow of fluid in the dentin tubules, resulting in all of the test animals developing severe tooth decay! Dr. Steinman demonstrated dramatically what I said a minute ago: Dental caries reflect systemic illness.

Let's take a closer look to see how this might happen. Once a tooth gets infected and the cavity gets into the nerve and blood vessels, bacteria find their way into those tiny tubules of the dentin. Then no matter what we do by way of treatment, we're never going to completely eradicate the bacteria hiding in the miles of tubules. In time the bacteria can migrate through lateral canals into the surrounding bony socket that supports the tooth. Now the host not only has a cavity in a tooth, plus an underlying infection of supporting tissue to deal with, but the bacteria also exude potent systemic toxins. These toxins circulate throughout the body triggering activity by the immune system – and probably causing the host to feel less well. This host response can vary from just dragging around and feeling less energetic, to overt illness – of almost any kind. Certainly, such a person will be more vulnerable to whatever "bugs" are going around, because his/her body is already under constant challenge and the immune system continues to be "turned on" by either the infective agent or its toxins – or both.

MJ What a fascinating concept. Can you tell us more about the protective nutrition you mentioned?

GM Yes. Dr. Price traveled all over the world doing his research on primitive peoples who still lived in their native ways. He found fourteen cultural pockets scattered all over the globe where the natives had no access to "civilization" – and ate no refined foods.

Dr. Price studied their diets carefully. He found they varied greatly, but the one thing they had in common was that they ate whole, unrefined foods. With absolutely no access to tooth brushes, floss,

fluoridated water or tooth paste, the primitive peoples studied were almost 100% free of tooth decay. Further – and not unrelated – they were also almost 100% free of all the degenerative diseases we suffer – problems with the heart, lungs, kidneys, liver, joints, skin (allergies), and the whole gamut of illnesses that plague Mankind. No one food proved to be magic as a preventive food. I believe we can thrive best by eating a wide variety of whole foods.

MJ Amazing. So by "diet and nutrition" for oral (and total) health you meant eating a pretty basic diet of whole foods?

GM Exactly. And no sugar or white flour. These are (and always have been) the first culprits. Tragically, when the primitives were introduced to sugar and white flour their superior level of health deteriorated rapidly. This has been demonstrated time and again. During the last sixty or more years we have added in increasing amounts, highly refined and fabricated cereals and boxed mixes of all kinds, soft drinks, refined vegetable oils and a whole host of other foodless "foods". It is also during those same years that we as a nation have installed more and more root canal fillings – and degenerative diseases have become rampant. I believe – and Dr. Price certainly proved to my satisfaction – that these simultaneous factors are NOT coincidences.

MJ I certainly understand what you are saying. But I'm still a little shocked to talk with a dentist who doesn't stress oral hygiene.

GM Well, I'm not against oral hygiene. Of course, hygiene practices are preventive, and help minimize the destructive effect of our "civilized", refined diet. But the real issue is still diet. The natives Dr. Price tracked down and studied weren't free of cavities, inflamed gums, and degenerative diseases because they had better tooth brushes!

It's so easy to lose sight of the significance of what Dr. Price discovered. We tend to sweep it under the rug – we'd actually prefer to hear that if we would just brush better, longer, or more often, we too could be free of dental problems.

Certainly, part of the purpose of my book is to stimulate dental research into finding a way to sterilize dentin tubules. Only then can dentists really learn to save teeth for a lifetime. But the bottom line remains: A primitive diet of whole unrefined foods is the only thing that has been found to actually prevent both tooth decay and degenerative diseases.

(This article was excerpted from http://www.mercola.com, the best health newsletter on the interent. Dr. Meinig's book, The Root Canal Cover Up, is available on Amazon.com)

Back to top

Are Nanobacteria Making Us Ill?

www.Wired.com
By Amit Asaravala|

02:00 AM Mar, 14, 2005

Olavi Kajander didn't mean to discover the mysterious particles that have been called the most primitive organisms on Earth and that could be responsible for a series of painful and sometimes fatal illnesses.

He was simply trying to find out why certain cultures of mammalian cells in his lab would die no matter how carefully he prepared them.

So the Finnish biochemist and his colleagues slipped some of their old cultures under an electron microscope one day in 1988 and took a closer look. That's when they saw the particles. Like bacteria but an astonishing 100 times smaller, they seemed to be thriving inside the dying cells.

Believing them to be a possible new form of life, Kajander named the particles "nanobacteria," published a paper outlining his findings and spurred one of the biggest controversies in modern microbiology.

At the heart of the debate is the question of whether nanobacteria could actually be a new form of life. To this day, critics argue that a particle just 20 to 200 nanometers in diameter can't possibly harbor the components necessary to sustain life. The particles are also incredibly resistant to heat and other methods that would normally kill bacteria, which makes some scientists wonder if they might be an unusual form of crystal rather than organisms.

In 1998, Kajander tried to prove the skeptics wrong by turning up what he believed to be an example of nanobacteria's ribosomal RNA, something that only organisms have. But the claim was squashed two years later by a National Institutes of Health study, which found that the RNA was actually a remnant from a type of bacteria that often contaminates lab equipment.

The debate would have ended there, except for a steadily increasing number of studies linking nanobacteria to serious health problems, including kidney stones, aneurysms and ovarian cancer. The studies show that nanobacteria can infect humans, a find that has helped push nanobacteria back into the limelight. Now the pressure is on to resolve the controversy and expose how nanobacteria works — no matter what it is.

"It's all pretty exciting stuff," said David McKay, chief scientist for astrobiology at NASA's Johnson Space Center. "Whether these are bacteria or not — it doesn't matter at this point. What matters is if we can figure out the association between nanobacteria and kidney stones and develop some kind of countermeasure."

The link between nanobacteria and human diseases was first noticed by Kajander and microbiologist Neva Çiftçioglu in 1998. The researchers had observed, through an electron microscope, nanobacteria particles building shells of calcium phosphate around themselves. They began to investigate whether such particles played a role in causing kidney stones, which are also made of calcium compounds. Sure enough, at the center of several stones was a nanobacteria particle.

Another breakthrough came in 2003 when a team from the University of Vienna Medical Center discovered nanobacteria in the calcified debris found in tissue samples from ovarian cancer patients. Meanwhile, several other studies revealed nanobacteria in samples of calcified arteries.

Sensing a growing need for tools to detect and study nanobacteria, Kajander and Çiftçioglu formed a company called NanoBac in 1998. The decision was greatly criticized as a conflict of interest and is still brought up whenever either of the two publishes a new paper.

Fortunately for the researchers, a 2004 study by the esteemed Mayo Clinic supported many of their key findings and helped them regain some of their support. The Mayo study found that nanobacteria do indeed self-replicate, as Kajander had noticed, and endorsed the idea that the particles are life forms.

Kajander and Çiftçioglu were further vindicated this February when patients with chronic pelvic pain — thought to be linked to urinary stones and prostate calcification — reported "significant improvement" after using an experimental treatment provided by Nanobac Life Sciences, which now owns NanoBac. The study was conducted by a team at Cleveland Clinic Florida.

There's a lot riding on studies like these. Roughly 177,500 patients were discharged from U.S. hospitals with kidney stones and related problems in 2001, according to the NIH. More than 25,000 women in the United States are diagnosed with ovarian cancer each year. In the same period, 14,000 Americans die from complications caused by calcified arteries.

"It brings up a lot of questions," said John Lieske, who led the 2004 Mayo Clinic study. "How many kidney stones are caused by this? Are there other calcification-related diseases that are caused by nanobacteria? Is it infectious?"

Surprisingly, few groups are actually working on answering these questions. One would be hard-pressed to find more than a half-dozen research teams around the globe studying nanobacteria full time.

Lieske suggests it's because the field is still relatively young. But it's clear that there's an additional culprit: the often heated controversy over whether nanobacteria particles are, in fact, alive.

"There's a reluctance to get into controversial areas. It's hard to get proposals funded," said McKay. "Most people are waiting until there's a little more meat on the bones."

Even John Cisar, who led the 2000 NIH study that contradicted Kajander's initial findings, agrees that the issue has become muddled. Though he maintains his stance that nanobacteria are not alive, he said in a phone interview that he is not against further research.

"I'm not saying there's nothing there," said Cisar. "It's just that we were looking at it from a microbiologist's perspective. And when we didn't find any signs of life, we moved on."

Kajander stands by his original assertion that nanobacteria are life forms. However, he blames

himself for getting researchers hung up on the life question by using the name “nanobacteria.”

“Calcifying self-propagating nanoparticles would have been much better,” he wrote in an e-mail to Wired News.

But he added that his regrets about the name don’t change the fact that nanobacteria have “miraculous” properties. Those include a growth cycle that closely matches typical biological cycles, the ability to form a shell and the “presence of both mammalian and bacterial components.”

It’s these properties — and the potential to save lives — that keep researchers focused on nanobacteria.

In February, NASA’s McKay and Nanobac’s Çiftçioglu announced that they had observed nanobacteria growing at five times its normal rate after they placed it in an incubator that simulates the microgravity conditions of space. The findings mean astronauts may be at an elevated risk for kidney stones on long flights — something NASA is extremely worried about in light of its new plans to send humans to Mars.

The findings could also add fuel to nanobacteria research by giving scientists a way to grow cultures faster.

“The trouble with studying nanobacteria is that trying to get enough material is very hard,” said Lieske. “Trying to culture a lot of it takes time.”

Indeed, nanobacteria particles double about once every three days. In comparison, typical bacteria double about every 20 minutes.

Lieske’s group has continued to experiment with nanobacteria since its 2004 paper. Though he said the team is looking for evidence of DNA and RNA, he is cautious about saying whether he thinks the particles are alive or just an unknown form of crystal.

As a possibility, he offered a third option: The particles could be a form of archaea, a relatively new category of tiny organisms whose DNA is vastly different from that found in typical bacteria. Over the past two decades, archaea have surprised scientists by turning up in places where life was least expected, like in sulfurous lakes and hydrothermal sea vents.

Whatever the case, the Mayo Clinic team may publish a paper outlining new findings in about six months, according to Lieske.

The world may not be waiting, but a handful of faithful microbiologists certainly will.

Back to top

Do Clouds Harbor Infectious Nanobacteria?

Clouds May Harbor Nanobacteria

www.wired.com

By Amit Asaravala
02:00 AM Apr, 11, 2005

Tiny particles linked to a number of painful and sometimes deadly diseases may spread across the globe by hitching a ride in clouds, claim researchers in a recent issue of the Journal of Proteome Research.

The particles, known as nanobacteria, are 100 times smaller than typical bacteria and have been found in kidney stones, arterial plaques and ovarian cancers.

But scientists have yet to agree whether the particles actually cause the diseases or how they infect humans.

Also unknown is whether the particles are life forms or an unknown type of crystal — a rift that has sparked one of the biggest controversies in modern microbiology.

Now, a new theory by Andrei Sommer, of the University of Ulm, Germany, and N. Chandra Wickramasinghe, of Cardiff University in the United Kingdom, attempts to show how nanobacteria moves from humans to the environment and back.

In a letter in the February issue of the Journal of Proteome Research, the pair describe studies suggesting that nanobacteria exist in the atmosphere — at least above Hyderabad, India, where the researchers captured samples of the air with a specially designed balloon.

The nanobacteria particles closely resembled those found in humans when compared on seven key criteria, including size and shape — a finding that suggests humans can be infected through the atmosphere.

In the journal's introduction to the paper, Sommer theorizes that the particles may be introduced to the atmosphere through human urine, which enters waste-water streams and becomes aerosolized.

Once in the atmosphere, the nanobacteria can fall back to Earth in dry or wet form. The researchers think dry forms are relatively harmless, but wet forms, in raindrops, would be more likely to be infectious because the nanobacteria would still be "active."

"Inactive, transiently desiccated microorganisms, transported back from the dry atmosphere to the Earth by gravity, are likely to cause little harm, compared to those returning in rain drops, after having been incorporated for some time in long-lived clouds, where they would encounter better conditions for revitalization," wrote the researchers.

The researchers also suggested that nanobacteria could help clouds develop by clumping together at the perfect size to promote the collection of airborne water droplets.

Attempts to contact Sommer and Wickramasinghe after business hours Friday were unsuccessful.

Back to top

Is There Any *Real* Medical Evidence of Nanobacteria as a Cause of Chronic Disease?

New Evidence on Infection as Chronic Disease Trigger Published, Debated by World-Class Scientists

Cutting Edge Research Aimed at Redefining the Way Doctors Diagnose and Treat Disease

TAMPA, FL (Dec. 20, 2006) New evidence that may help solve one of the great puzzles of 21stcentury medicine was published today in a special section of the Journal of Investigative Medicine. It features leading scientists in the field brought together by The American Federation for Medical Research and the American Physiological Society.

Is chronic disease triggered by an infection? New evidence comes from scientists at NASA, Mayo Clinic, and Nanobac Pharmaceuticals who pioneered investigations into infectious calcifying particles. A condition known as calcification occurs in most diseases on the leading cause of death list and in illnesses such as kidney, gallbladder and prostate stones. Calcification is also linked to chronic inflammation in atherosclerosis and end-stage renal disease, but it is unclear how this occurs.

Half-a-dozen papers in the Journal resulted from a symposium that assembled, for the first time in medical history, experts from biology, medicine and geology to debate these possible causes of calcification: chemical crystallization; cell-mediated crystallization; and potentially infectious calcifying nanoparticles (CNPs) that generate calcification.

One conclusion by symposium organizers: Although infection is just one of three potential causes of calcification, it deserves focus as a chronic disease trigger, based on new evidence presented. “Increasingly, micro-organisms are being identified as an unexpected cause of disease…” writes symposium co-organizer and Mayo Clinic scientist Dr. Virginia Miller.

The symposium considered works by Dr. Neva Ciftcioglu and Dr. Olavi Kajander, lead scientists with Nanobac Pharmaceuticals, as well as two other works by Mayo researchers, which provide evidence that calcifying nanoparticles might be infectious and spark calcification in disease.

Although Dr. Miller says the idea of infectious nanoparticles is still controversial, she concludes that, “Nanoparticles might serve as an inflammatory stimulus that initiates cell transdifferentiation, stimulate the formation of matrix vesicles, or simply form a nidus for subsequent inorganic calcium accumulation.”

Participants in the symposium included scientists from such world-class institutions as UCLA and various institutes in Europe. Scientists from Nanobac Pharmaceuticals are the leading investigators on the issue of infection as a possible cause of calcification, and have published the most papers on this topic.

Participants list:

Neva Ciftcioglu, Ph.D., director of science at Nanobac Pharmaceuticals, is one of the scientists who originally isolated nanoparticles from mammalian blood and, working at NASA's Johnson Space Center, presented concepts of nanoparticles as a nidi for biomineralization.

Neal X. Chen, Ph.D., assistant professor in the Department of Medicine at Indiana University, reviews mechanisms of cell transdifferentiation and crystallization in renal cells.

John C. Lieske, MD, associate professor of Medicine, Mayo Clinic College of Medicine, continued the theme of calcification of renal tissue, in particular Randall's plaques, by presenting evidence for self-replicating, self-calcifying nanoparticles in renal stones.

Linda L. Demer, MD, Ph.D., professor of medicine, Division of Cardiology, The David Geffen School of Medicine at UCLA, reviewed transdifferentiation of vascular smooth muscle cells to a boneassociated phenotype and the importance of RANKL and receptor in that process.

Howard H.T. Hsu, Ph.D., associate professor, Department of Pathology, University of Kansas Medical Center, reviewed data supporting the contribution of matrix vesicles to vascular calcification and presents a provocative theory of the stimuli involved in their formation.

Karim Benzerara, Ph.D., Institut de Mineralogis et de Physique des Milieux Condenses and Institut de Physique du Globe of Paris, France, demonstrates how the state-of-the-art scanning transmission X-ray microscopy and near-edge x-ray absorption fine structure (NEXAFS) can be used to define the biochemical characteristics of nanoparticles isolated from the environment and human tissue.

Virginia M. Miller, PhD., Professor of Surgery, Mayo Clinic College of Medicine, symposium coorganizer.

For more information or to schedule a briefing/interview please contact Carson Chandler (202) 367-1625, cchandler@akerpartners.com or Matt Taylor (202) 367-1631, hyperlink.

About Nanobac Pharmaceuticals: Nanobac Pharmaceuticals Inc. ((OTCBB:NNBP)) is dedicated to the discovery and development of products and services to improve human health through the detection and treatment of calcifying nanoparticles (CNPs). The company's pioneering research is establishing the pathogenic role of CNPs in soft tissue calcification, particularly in coronary artery, prostate and vascular disease. Nanobac's drug discovery and development is focused on new and existing compounds that effectively inhibit, destroy or neutralize CNPs.

Back to top

Can Hidden Infections Be a Major Cause of Mental Illness?

The Infection Connection – how infections might be partly responsible for mental illnesses

Psychology Today, July, 1999

by Harriet Washington

PSYCHOLOGY HAS LONG HELD THAT MENTAL ILLNESS IS BORN OF ADVERSE EXPERIENCES. MORE RECENTLY, RESEARCH HAS POINTED THE FINGER AT FLAWED GENES. NOW A THIRD CULPRIT MAY BE EMERGING: INVASION BY BACTERIA AND VIRUSES.

Eight-year-old Seth broke from the grasp of Jane, his harries mother, for the third time in 10 minutes. Tering across the emergency room, he stopped short, transfixed by a piece of paper lying on the floor. His ref-rimed eyes seemed to bulge from their sockets and his mouth twitched violently, as if he were in pain. Indifferent to Jane's pleas to stop, he proceeded to pick up from the floor every piece of paper, no matter how filthy, with hands that were reddened and raw. It was the state of his hands that had precipitated the trip to the hospital: Seth had spent most of the night in the bathroom, washing them over and over.

With his head jerking spasmodically and his fingers pecking at pieces of paper and cigarette butts, the boy resembled some strange overgrown bird. Then, suddenly terrified, he flew back to Jane and began pulling on her arm. "Mommy, Mommy, let's leave!" he whimpered. "They're going to kill us. They're coming!"

Jane tried her best to calm him, but she too was beginning to panic. Two days before, Seth had been a perfectly normal little boy whose most serious health problems were the occasional cold or sore throat. He had become mentally ill overnight.

What caused Seth's anxiety, his tics, his obsessive-compulsive behavior? Astonishingly, it was probably that minor sore throat, his doctors concluded. Today, scientists are increasingly coming to recognize that the bacteria and viruses that frequently invade our bodies and cause sore throats and other minor ailments may also unleash a host of major mental and emotional illnesses, including anorexia, schizophrenia and obsessive-compulsive disorder.

It is a theory sharply at odds with earlier views of the genesis of psychological illness. Followers of Freud long held that mental and emotional trouble is primarily the result of poor parenting, especially by mothers. Indeed, until about 30 years ago, psychoanalysts frequently placed the blame for schizophrenia on "schizophrenogenic" mothers. Obsessive-compulsive disorder, also, was put at Mom's door. "It was thought to be the result of harsh toilet training," observes Susan Swedo, M.D., chief of pediatrics and developmental neuropsychiatry at the National Institutes of Mental Health. But such theories, which added immeasurable guilt to the burdens of parents with mentally ill offspring, have turned out to have little evidence to back them up, most experts now agree.

Instead, in recent years, the focus has shifted to genes as the main source of mental illness. Faulty DNA is thought to be at least partly responsible for, among other problems, anxiety and panic disorders, schizophrenia, manic depression and antisocial personality disorder, which is characterized

by impulsive, excessively emotional and erratic patterns of interpersonal behavior.

Yet genetics doesn't appear to wholly account for the occurrence of major psychiatric ailments. If heredity alone were to blame, identical twins would develop schizophrenia with a high degree of concordance, but in fact in only 40% of cases in which one identical twin has the disease does the other twin have it as well. Autism, though it has been observed to run in families, also strikes five of every 10,000 children apparently arbitrarily Nor can depression and other affective disorders be completely explained by damaged DNA. Says Ian Lipkin, Ph.D., a neuroscientist and microbiologist at the University of California at Irvine: "Genetics doesn't hold the key to understanding how to fit these square pegs into round holes."

Bacteria and viruses may be that key, but scientists have been slow to grasp the idea. Consider the case of syphilis, which is caused by the bacterium Treponema pallidum. In its final, or tertiary, stage, the disease can precipitate psychiatric problems like dementia, mania, depression, delusions and Tourette's like tics. Though some scientists' suspected a connection between infection with the bacterium and the mental disturbances that may take three to live decades to emerge, the link became widely accepted only in the 1940s after the introduction of the antibiotic penicillin as a treatment for syphilis. In the interim, patients with syphilis who later developed psychiatric problems were often institutionalized as crazy But even with the link established, Freud's theories were in ascendance and few scientists were willing to consider that microbes might be a common source of other mental illness.

Now, decades later, infection has emerged as a prime suspect in psychological illnesses. The inadequacy of genetic and experiential explanations has prompted scientists to look elsewhere–and their gaze has come to rest on physical ailments, such as heart disease, cancers and ulcers, that in some cases have an infectious origin. Could the same be true, they wonder, for mental and emotional ills?

Back to top

What Does Medical Science Know About the Infectious Basis of Mental Illness?

The Role of Infections in Mental Illness

by Frank Strick, Clinical Research Director

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In considering an infectious etiology to any chronic mental illness there are at least four categories to consider. First are those infections already recognized to induce psychiatric symptoms. These include

pneumonia, urinary tract infection, sepsis, malaria, Legionnaire's disease, syphilis, typhoid, diphtheria, HIV, rheumatic fever and herpes. (Ref: Chuang)

While the psychiatric effects of these infections are known to the medical field, they are rarely screened for if the initial presentation is made to a mental health professional. Moreover, the significance of some of these infections may date back to prenatal development. Research done at the John Hopkins Children's Center and published in the Archives of General Psychiatry in 2001 found that mothers with evidence of Herpes Simplex Type 2 infection at the time of pregnancy had children almost six times more likely to later develop schizophrenia. And in the US, Europe and Japan, birth clusters of individuals who develop schizophrenia later in life closely mirror the seasonal distribution of Ixodes ticks at the time of conception (Lyme disease).

Second are those parasitic infections such as neurocysticercosis where the brain is directly invaded by the infective agent through a well-established, imageable (visible on brain scan) mechanism (cysts, lesions, cerebral swelling etc.) Signs of psychiatric disease (depression and psychosis) were found in over 65% of neurocysticercosis cases (caused by a tapeworm whose incidence in the US is rising due to demographic increases in foreign immigrant populations.) [Ref: Forlenza] While the mechanisms for psychiatric manifestations are easy to demonstrate when brain tissue is directly affected, there are also multiple documented reports in the literature of psychiatric symptoms associated with other parasites like giardiasis, ascaris (roundworm), trichinae (cause of trichinosis), and Lyme borrelia and viruses like borna virus. Documentation also exists of these psychiatric symptoms resolving when the underlying hidden infection is treated.

Dr. J. Packman of Yale University wrote over ten years ago that "Patients with parasitic loads are more likely to exhibit mental status changes and there is an improvement in mental status of a subset of psychiatric patients following treatment for parasites." In fact, a review of 1300 human cases of trichinosis in Germany found CNS (central nervous system) involvement in up to 24% of the cases (Menningeal inflamation or encephalitis). [Ref: Froscher]

Clinically, in cases like neurocysticercosis, the problem is not the lack of a well-defined mechanism but the lack of mental health practitioners qualified to make such a diagnosis or even suspect it. Even infectious disease specialists tend to underestimate the scope of the problem, in part due to underreporting (neurocysticercosis is not a reportable condition in most states and the incidence of trichinosis is, we believe, vastly underestimated according to newly developed antibody assays only made available in 2003).

Next are those parasitic, bacterial and viral infections like toxoplasmosis and strep where a strong statistical link to mental illness has been demonstrated but research is underway to establish a causal connection. In humans acute infection with toxoplasmosis gondii can cause brain lesions, changes in personality and symptoms of psychosis including delusions and auditory hallucinations. Researchers at Rockefeller University and NIMH have suggested that after streptococcal infection some children may be at increased risk for Obsessive Compulsive Disorder. Toxoplasma gondii can alter behavior

and neurotransmitter function. Since 1953, eighteen out of nineteen studies of T. gondii antibodies in persons with schizophrenia and other severe psychiatric disorders have reported a higher percentage of T. gondii antibodies in the affected persons. (For example, in one large study toxoplasmosis infection was twice as common in mentally handicapped patients as in healthy controls and in a recent German study of "individuals with first episode schizophrenia compared to matched controls, 42% of the former compared to just 11% of the latter had antibodies to toxoplasma").

Two other studies found that exposure to cats (the primary carrier for toxoplasmosis transmission) in childhood is a risk factor for the development of schizophrenia. Furthermore, certain antipsychotic and mood-stabilizer drugs such as Halperidol and Valproic acid inhibited this parasite in vitro at a concentration below that found in the cerebrospinal fluid and blood of individuals being treated with this medication, suggesting that some medications used to treat schizophrenia and bipolar disorder may actually work by inhibiting the replication of toxoplasmosis gondii. (Ref: Jones-Brando, Torrey, Yolken)

Other studies have shown that antipsychotic drugs like Thorazine, Haldol and Clozapine inhibit viral replication and that the cerebrospinal fluid of patients with recent-onset schizophrenia shows a significant increase in reverse transcriptase (an enzyme) activity – which is an important component of infectious retroviruses (a type of virus). Furthermore, when the CSF (cerebral spinal fluid) from these patients was used to inoculate a New World monkey cell line there was a tenfold increase in reverse transcriptase activity which suggests the presence of a replicating virus. Malhotra has demonstrated the absence of CCR5-32 homozygotes (specific matching genetic codes) in over 200 schizophrenic patients – which dramatically increases susceptibility to retroviral infection. (Ref: F.Yee).

It is research like this that has led Johns Hopkins virologist Robert Yolken and psychiatry professor and former special assistant to the Director of the National Institute for Mental Health Dr. E. Fuller Torrey to believe that toxoplasmosis is one of several infectious agents that causes most cases of schizophrenia and bipolar disorder. The idea is not new. In fact, as far back as 1922 the famous psychiatrist Karl Menninger hypothesized that schizophrenia was "in most instances the byproduct of viral encephalitis." Torrey notes that in the late nineteenth century schizophrenia and bipolar disorder went from being rare diseases to relatively common ones at the same time that cat ownership became popular. And Yolken designed a retrospective study of twenty-five hundred families showing that mothers of children who later developed psychoses were 4.5 times more likely to have antibodies to toxoplasmosis than the mothers of healthy controls. Due to the frequency of cat ownership, a large percentage of the US population (up to 50%) has been exposed to toxoplasmosis but most immunocompetent carriers remain asymptomatic until another immunological burden such as HIV or a separate parasite weakens the host defenses and precipitates pathogenic expression. That is what makes interpretation of the chronic state so tricky and at the Research Institute for Infectious Mental Illness we make sure to try to identify any parasitic coinfections before deciding on an appropriate

course of treatment.

Finally, while toxoplasmosis gets a lot of attention due to Torrey's and Yolken's pioneering studies and the known mechanism of brain lesions, there are many other infective agents that may not target the brain specifically but can severely affect mental function through the cumulative downstream consequences of chronic infection. While the importance of this link in the etiopathogenesis of mental illness is rarely recognized, these focal and systemic infections are very common and their psychiatric effects often severe. (Parasites are the most common causes of mortality and morbidity in the world.) In this nonspecific category are scores of parasites, protozoa, helminths, bacteria, fungi and viruses which, if not directly invading and disabling brain tissue and neurotransmitter function, do so indirectly by depleting the host of essential nutrients, interfering with enzyme functions, and releasing a massive load of waste products – enteric poisons and toxins which disrupt brain metabolism. (A single mature adult tapeworm can lay a million eggs a day and roundworms, which infect about twenty-five per cent of the world's population, lay 200,000 daily).

Remember, the brain is your body's most energy-intensive organ. It represents only three percent of your body weight but utilizes twenty-five percent of your body's oxygen, nutrients and circulating glucose. Therefore any significant metabolic disruptions can impact brain function first. This link is borne out statistically. Mental patients have much higher rates of parasitic infection than the general population. Between 1995 and 1996 researchers at the University of Ancona did stool tests on 238 residents of four Italian psychiatric institutions and found parasites in 53.8 percent of the residents including all of those residents with behavioral aberrations(Ref: Giacometti). In our experience parasites are often implicated in cognitive dysfunction and chronic emotional stress disorders and, to the untrained eye, classic symptoms like apathy, exhaustion, confusion, appetite and memory loss, "nervous stomach," social withdrawal, lethargy and loss of sex drive and motivation are frequently assumed to signal a depressive disorder without an adequate differential diagnosis being made or even attempted. Adding to the confusion, classic indicators of acute infection such as fever or elevated antibodies often reverse themselves in chronic cases due to secondary hypothyroidism and immunodepression. Unfortunately, until Western psychiatry further recognizes that the mind/body connection goes in both directions patients will continue to suffer from a de facto lack of differential diagnostic criteria in clinically identical syndromes.

Even for those clinicians who recognize the devastating psychological effects that chronic intestinal, focal and even dental infections can have on normal brain function, accurate diagnosis presents formidable challenges. In fact some standard parasite stool test procedures identify less than ten percent of active infections and even the "politically correct" holistic specialty labs miss many infections that are nondetectable in fecal specimens, have inconsistent shedding patterns, are extra intestinal or otherwise hard to identify. For example, according to the World Health Organization, over two billion people are infected with worms, yet rarely will they show up in stool assays.

(These numbers are not surprising once you realize that the exposure vectors are potentially

everything you eat, drink, breathe and touch. If you think you have to leave the country to be exposed to exotic parasites, think again. In fact, try walking into the kitchen of your favorite restaurant and see if the cook speaks English.)

At the Research Institute for Infectious Mental Illness we use multiple labs with complementary strengths and a combination of advanced scientific diagnostic procedures including O & P microscopy, multifluid antigen and antibody detection, stool cultures, enzyme immunoassay, mucosal markers, inflammation assays, imaging techniques and other indirect laboratory indicators combined with extensive historical and clinical evaluations to identify chronic infectious stressors. (Patients previously diagnosed with "Chronic Candidiasis" often find that Candida was merely a cofactor or consequence of more significant infections and infestations which created obstacles to long-term cure.) "Mental" symptoms often improve dramatically when hidden neuroimmune infections are treated successfully and normal brain metabolism resumes, especially in "sudden-onset" syndromes. After identifying and treating the primary infections we focus on rebuilding the host's immunological defenses and mucosal integrity to prevent relapse. Premature nutritional supplementation, even in frank anemia, can be counterproductive since some vitamins and minerals (e.g., iron) can be growth factors for microorganisms which the body intentionally downregulates the uptake of during active infection. But individually formulated subsequent nutritional supplementation is usually essential for full recovery. We also screen patients for heavy metals, environmental chemicals, molds and electromagnetic stressors, "Brain allergies," food sensitivities, hormone disorders, diet and numerous other variables which can influence cognitive and affective function. To speed recovery, our evidence-based Integral Medicine approach may include appropriate treatments from consulting nutritionists, homeopaths, acupuncturists, herbalists and bodyworkers.

The erosion or loss of brain function is arguably the most frightening and disabling experience a person can have. Almost by definition, standard psychological or psychiatric intervention postulates a dichotomy between disorders of the body and those of the mind and has a long way to go in recognizing the importance of infectious etiologies in mental health care. The Research Institute for Infectious Mental Illness provides testing, clinical and consulting services to clients from all over the world and educates professionals in this critical area. Long distance phone consultations are also available.

Back to top

Is The "Russell Body" the Forgotten Clue to the Bacterial Cause of Cancer?

The twentieth century was indeed the century of Modern Medicine with tremendous strides made in the understanding and control of infectious diseases, as well as the introduction of life-saving antibiotics and vaccines. Unfortunately, along with these advances came the perils of genetic

engineering, the increasing threat of newly emerging viruses, bio-warfare, and bio-terrorism

Despite these scientific achievements, the cause of cancer remains a mystery. Scientists suspect genetic susceptibility, possible cancer-causing viruses, and environmental factors might play a role in some cancers, but none of these factors explain why millions of people die yearly from a variety of malignancies.

William Russell

How could scientists put men on the moon, but remain so ignorant about cancer and its origin? How can the infectious causes of tuberculosis, leprosy, syphilis, smallpox, polio, malaria, and other viral and bacterial and parasitic diseases be understood, but the cause of cancer be unknown? Could the cause of cancer conceivably be an infectious agent that has been overlooked, ignored, or unrecognized by medical doctors in the twentieth century? Could the germ of cancer be hidden in the Russell body? – a large microscopic form known to every pathologist for over a century!

On December 3, 1890 William Russell, a pathologist in the School of Medicine at the Royal Infirmary in Edinburgh, gave an address to the Pathological Society of London in which he outlined his histopathologic findings of “a characteristic organism of cancer” that he observed microscopically in fuchsine-stained tissue sections from all forms of cancer that he examined, as well as in certain cases of tuberculosis, syphilis and skin infection.

The parasite was seen within the tissue cells (intracellular) and outside the cells (extracellular). The size of Russell’s parasite ranged from barely visible, up to “half again as large as a red blood corpuscle.” The largest round forms were easily seen microscopically. The large size of some of these bodies suggested a fungal or yeast-like parasite. Russell provisionally classified the parasite as a possible “blastomycete” (a type of fungus); and called the forms “fuchsine bodies” because of their bluish-red staining qualities.

Microbiology was still in its infancy in Russell’s era, and it was generally thought that each microbe could only give rise to a single disease. Thus, the idea of a cancer germ (especially one that could also be identified in TB and syphilis) was received cautiously. Nine years later in 1899, in yet another report on “The parasite of cancer” appearing in The Lancet (April 29), Russell admitted that finding cancer parasites in diseases other than cancer was indeed a “stumbling block.” By this time a considerable number of scientists concluded that Russell bodies were merely the result of cellular degeneration of one kind or another. Furthermore, no consistent microbe was cultured from tumors; and the inoculation of these microbes into animals produced conflicting and often negative results.

Russell was trained as a pathologist, not as a microbiologist, and he avoided getting into the bacteriologic controversies regarding various microbes grown from cancer. He simply concluded, “It seems almost needless to add that there remains abundant work to be done in this important and attractive field.”

After three years' work at the New York State Pathological Laboratory of the University of Buffalo, Harvey Gaylord confirmed Russell's research in a 36 page report titled "The protozoon of cancer", published in May, 1901, in the American Journal of the Medical Sciences. Gaylord found the small forms and the large sacs characteristic of Russell bodies in every cancer he examined. Some large spherical bodies were four times the diameter of a leukocyte (white blood cell). Red blood cells measure about 7 micron in diameter and leukocytes are 2 to 3 times larger than red blood cells. Thus, some of the bodies that Gaylord observed attained the amazing size of around 50 micron in diameter. In addition, he found evidence of internal segmentation within the larger bodies "after the manner recognized in malarial parasites." The tiniest forms appeared the size of ordinary staphylococci.

Russell bodies in a lymph node of Hodgkin's disease. Gram's stain, magnified 1000 times, (in oil). Russell's 1899 paper ended his writings of a cancer parasite, but his discovery quickly became known to pathologists as Russell bodies. These bodies continue to fascinate researchers and physicians (like myself) up to the present time.

When Russell died at the age of 89 in 1940, the British Medical Journal published a large obituary noting that he was universally respected and embued with the dignity and highest ideals of his profession, and that he had served at one time as President of the Royal College of Physicians. No mention was made of his "parasites" or his "bodies", except to remark that "in his earlier years Russell devoted much time to the study of the cancer cell." Similarly, a large obituary appeared in the Edinburgh Medical Journal along with a full-page photo. His published books on Clinical Methods and widely read texts on circulation and gastro-intestinal diseases were cited, but not a word about his discovery in cancer.

The heresy of "the cancer microbe"

By the early part of the twentieth century the top cancer experts had all rejected so-called "cancer parasites" as the cause of cancer. The most influential physician to speak against it was James Ewing, an American pathologist and author of the widely-read textbook, Neoplastic Diseases. In 1919 Ewing wrote that "few competent observers consider it (the parasitic theory) as a possible explanation in cancer." According to Ewing and other authorities, cancer did not act like an infection. Therefore, microbes could not possibly cause cancer. He concluded, "The general facts of the genesis of tumors are strongly against the possibility of a parasitic origin."

As a result, the parasitic theory was totally discarded and few doctors dared to contradict Ewing's dogma by continuing to search for an infectious agent in cancer. Nevertheless, a few die-hard physicians remained convinced microbes were at the root cause of cancer and wrote about it convincingly in medical journals. The long history of this research is recorded in my book, The Cancer Microbe (1990) and anyone with internet access can do a Google search (type in "cancer microbe") and obtain a wealth of information on the microbiology of cancer. Another excellent history of cancer microbiology and the suppression of this controversial research is contained in David Hess' Can Bacteria Cause Cancer? (1997).

n the 1920s James Young, an obstetrician from Scotland, repeatedly grew pleomorphic (having many forms) bacteria from various cancers. The microbes had a “specific life cycle” and “spore stages” comprised of exceedingly tiny and barely visible spores. In the laboratory these tiny spores transformed into larger coccoid (round) forms, rod-forms and yeast-like forms (similar in size to Russell bodies). John Nuzum, a Chicago physician, reported a pleomorphic coccus he repeatedly isolated from breast cancer. The tiniest forms were virus-like and passed through a filter designed to hold back bacteria.

In 1925 Northwest Medicine published two papers by Michael Scott, a Montana surgeon who learned about the cancer microbe in TJ Glover’s lab in 1921. Scott’s microbe was similar to Young’s. The parasite had a life cycle composed of three stages: a coccus, a rod, and a “spore sac” stage. Scott believed cancer was an infection like tuberculosis and attempted a vaccine treatment, but his treatment methods were quickly suppressed by the medical establishment.

In the 1930s in Germany the controversial Wilhelm von Brehmer described microbes in the blood of cancer patients, evoking the wrath of his scientific colleagues and prompting an intervention by Adolf Hitler. (See Proctor’s The Nazi War on Cancer [1999]) Georges Mazet, a French physician, also found pleomorphic bacteria in Hodgkin’s disease in 1941. Hodgkin’s is a type of lymphoma cancer involving the lymphatic system. Mazet later reported similar acid-fast (red staining) bacteria in many different kinds of cancer, including leukemia.

In the 1950s, 60s, and 70s, a quartet of women further refined the microbiology of cancer, emphasizing the extreme pleomorphism of the organism and its detection in tissue with the acid-fast stain. The published research of Virginia Livingston, Eleanor Alexander-Jackson, Irene Diller and Florence Seibert, is essential reading for the most updated understanding of the microbiology of cancer.

In the late 1970s Guido Tedeschi and other Italian microbiologists at the University of Camerino discovered “granules” in the red blood cells of healthy and ill people that turned out to be bacteria that could be cultured in the laboratory. Some of the staphylococcal and corynebacteria-like bacteria cultured from the red blood cells were acid-fast and cell wall-deficient, a staining and growth characteristic shared with the cancer microbe. This research has been confirmed by newer studies suggesting that bacteria reside in blood from healthy as well as sick individuals. These findings of tiny blood bacteria (nanobacteria) provide further evidence to support the theory that microbes can cause cancer.

Some other well-known scientists in the field of cancer microbiology include Gunther Enderlein, Royal Raymond Rife, Gaston Naessens and Wilhelm Reich. All have web sites devoted to their cancer research.

Russell bodies and their Origin

More than a century has passed since Russell’s discovery and although electron microscopes (which

have been used since the 1950s) have the ability to magnify objects tens of thousands of times, the significance and function of his bodies still remains unknown.

What is well-known is that Russell bodies can be found, not only in cancer, but in the majority of inflamed tissues throughout the body. Distinguishing large Russell bodies from actual fungal forms of Blastomyces can still be difficult, particularly when a pathologist encounters a true case of fungal infection due to Blastomyces.

In 1954 RG White, in “Observations on the formation and nature of Russell bodies”, produced Russell bodies in animals by injecting them with different species of bacteria. He then studied the ensuing development of these bodies in the spleen, lymph nodes and plasma cells of the injected animals. Plasma cells are specialized forms of white blood cells that normally produce antibodies.

EM Schleicher, in his 1965 paper on “Giant Russell bodies”, discusses the various theories of origin. Possibilities include origin from the lymphocyte, origin in plasma cells with later degeneration, origin from the mitochondria of cells, and even an origin from a red blood cell (erythrocyte) swallowed up by a plasma cell.

Most researchers currently believe Russell bodies are essentially immunoglobulins (proteins that acts as antibodies), but an electron microscopic study by SM Hsu et al. in 1981 has cast some doubt on this belief.

None of these studies mention the possibility that Russell bodies might represent unusual large growth forms of bacteria. However, if Russell bodies prove to be tiny intracellular microbes that grow and enlarge within leukocytes, it would be natural to expect these white blood cells (especially the plasma cell) to produce an antibody attack against these invading organisms, resulting in the production of immunoglobulin-coated cells and organisms.

Bacterial transformation into Giant forms (L-form “large bodies’)

There are many different kinds of bacteria but only one type that has been consistently observed and studied in cancer for over a century. The cancer microbe has many forms, some of which appear as ordinary staphylococci or larger yeast-like forms that further enlarge to the size of Russell bodies. As mentioned, some Russell bodies enlarge to truly gigantic proportions, one hundred times the diameter of small cocci. One can liken this growth potential to an empty balloon that is then blown up to full-size. In addition, the microbe has exceedingly small filterable submicroscopic forms approaching the size of viruses, visible only by use of the electron microscope.

Scientists who have extensively studied the cancer microbe claim it most closely resembles the type bacteria that cause tuberculosis and leprosy- the so-called mycobacteria. Mycobacteria are closely related to fungi; and some microbiologists claim mycobacteria are essentially derived from the “higher” fungi. “Myco” in Greek means fungus. Ergo, mycobacteria are considered fungus-like bacteria.

During the 1960s microbiologist Louis Dienes popularized the terms “cell wall-deficient” and “L form” to encompass bacterial growth stages that exist at one extreme as small filterable virus-sized forms, and at the opposite extreme as large (50 micron or larger) spherical forms that he termed “large bodies.” These so-called large bodies are what I believe Russell bodies represent.

It must be understood that microbes are partially “classified” in microbiology according to size. Viruses are submicroscopic and cannot be visualized with an ordinary light microscope. Unlike bacteria, viruses can only replicate inside a cell. Bacteria can be seen microscopically, but smaller submicroscopic and filterable bacterial forms (now known as nanobacteria) are also known. Fungi and yeast forms are much larger than bacteria, and “mold” can obviously be seen with the naked eye.

Larger Russell bodies are indeed similar in size to certain spore forms of fungi. However, what is generally not appreciated is that bacteria can grow into fungal-sized large bodies, depending on certain laboratory conditions. Thus, bacteria in this form can easily be mistaken for fungi and yeast organisms.

Giant-sized L-forms greatly resemble large-sized Russell bodies. The century-old history of research into atypical growth forms of bacteria is reviewed in Lida Mattman’s seminal text, Cell Wall Deficient Forms: Stealth Pathogens (1993). A knowledge of this somewhat esoteric branch of microbiology is essential to understand the proposed microbiology of cancer.

The most impressive electron microscopic photographs I have ever observed of cell wall-deficient L-forms of mycobacteria were taken by the late C Xalabarder of Barcelona. In a series of papers and books (1953-1976) published in Spanish (with English-language summaries) by the Publicaciones del Instituto Antituberculoso “Francisco Moragas”, Xalabarder totally transformed my concept about how tuberculosis-causing mycobacteria reproduce and grow and drastically change their appearance. In medical school we were taught that “simple” bacteria simply divide in two equal halves by “binary fission”. However, nothing could be further from the truth, and it is only by a refutation of this simplistic concept that a serious study of the microbiology of cancer can be undertaken.

Tuberculosis and Cancer

Because cancer is produced by a microbe similar to the bacteria that cause TB, much can be learned from experiments like those performed by Xalabarder in 1967. Using “atypical mycobacteria” grown from TB patients who had taken long courses of drug therapy, Xalabarder then injected these bacteria into guinea-pigs and rabbits. Amazingly, he was able to experimentally produce lesions which microscopically resembled cancer! He also produced experimental lesions characteristic of so called “collagen disease”- a type of lesion seemingly unrelated to cancer.

During the 1960s I discovered unusual pleomorphic acid-fast bacteria in a collagen disease called scleroderma, and later in another collagen disease called lupus erythematosus. The germs I grew from these patients closely resembled scleroderma microbes that were reported by Virginia Livingston in 1947, and which subsequently led to her discovery of similar acid-fast microbes in cancer.

In 1969 Xalabarder manipulated different developmental stages of TB bacteria and inoculated them into one thousand guinea pigs. In the process, he produced the microscopic picture of sarcoidosis in the animals. Sarcoidosis is a human disease closely related to TB but one in which TB germs cannot be found. Xalabarder's most impressive sarcoid lesions were produced by inoculating sputum specimens from TB patients who "converted", meaning that their TB bacteria could no longer be cultured from their sputum. Controversy over the cause of sarcoidosis is still not settled, although I reported bacteria similar to cancer microbes in this disease in the 1980s.

The most spectacular electron microphotographs of cell wall-deficient mycobacteria are presented in Xalabarder's L-forms of mycobacteria and chronic nephritis (1970). In the earliest growth stages of mycobacteria in culture the smallest elements appear as tiny submicroscopic forms visualized only with the electron microscope. These filterable forms of tuberculosis bacteria – the so-called "tuberculosis virus"- have been known to cause cancer in animals since the 1920s. By adding antibiotics to the lab culture media Xalabarder was able to induce many unusual growth forms of tuberculosis bacteria. Using serial images, he was able to trace the development of these tiny submicroscopic forms up to the size of ordinary cocci – and then up to the size of "large body" forms reaching and even surpassing the size of red blood cells. Some of the large bodies of mycobacteria also exhibit internal structure, similar to what Gaylord noted in his Russell body research.

Cancer and Bacteria

Although the idea of a cancer microbe is medical heresy, there is ample data to show that cancer patients are highly prone to bacterial infection. A PubMed computer search (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi) of "bacteria + cancer" elicits 49, 244 citations contained within 2,463 web pages. According to a 2003 article by Vento and Cainelli, patients with cancer who are undergoing chemotherapy are highly susceptible to almost any type of bacterial or fungal infection.

Why are physicians, and especially pathologists and bacteriologists, so unaware, so disinterested, or so antagonistic to credible cancer microbe research? Why have pathologists failed to consider Russell bodies as large forms of bacteria?

For over 30 years I studied various forms of cancer and skin diseases "of unknown origin", as well as autopsy cases of cancer, lupus, scleroderma, and AIDS. In all these diseases I was able to detect bacteria, although pathologists would never mention bacteria in any of their official biopsy reports. In my experience, they simply could not conceive of cancer and collagen disease (and AIDS) as a bacterial infection, nor did they seem to be aware of bacteriology reports pertaining to "large bodies" and pathologic effects produced by the "tuberculosis virus." In short, they were trained to see and report only the typical rod-shaped acid-fast (red-stained) "typical" form of mycobacteria, , but they were not trained to look for or to recognize other growth forms of the same bacteria that might be hidden in their pathologic tissue specimens.

When objects like Russell bodies are observed in a wide variety of diseases and in “normal” tissue, the significance is lessened. Doctors expect “normal” tissue to be free of microbes. I suppose they also conclude that Russell bodies cannot be an infectious agent because it would be impossible for an infectious agent to appear in so many different kinds of diseases and in so many different forms of cancer.

For most of the last century stomach ulcers were thought to be non-infectious because pathologists could not identify bacteria in the ulcers and because doctors believed bacteria could not live in the acid environment of the stomach. This thinking all changed gradually after 1982 when Barry Marshall, an Australian physician, proved most stomach ulcers were caused by a microbe called Helicobacter pylori, which could be identified microscopically with special tissue staining techniques in ulcer tissue. On the other hand, many people normally carry this stomach microbe without any ill effects. Not surprisingly, pathologists are now reporting numerous Russell bodies in plasma cells in some ulcer patients, giving rise to a previously unrecognized tissue reaction called “Russell cell gastritis.”

Russell bodies and bacteria

When bacteria are threatened by the immune system or by antibiotics they may lose their cell-wall and assume a different growth form that renders them less susceptible to attack by the immune system. Some Russell bodies elicit little or no inflammatory cell response. This lack of cellular response is yet another reason why physicians have a hard time believing Russell bodies could be microbes.

I have observed the largest and most complex Russell bodies in tissue where there was almost a total lack of inflammation. My photographs of such “large bodies”, some with obvious internal structure, that I observed in patients with scleroderma and pseudoscleroderma, were published in the American Journal of Dermatopathology in 1980. The first case of fatal scleroderma I studied in 1963 had numerous “large bodies” in the fat layer of the diseased skin that were unlike anything ever seen in dermatology. The patient had been hospitalized for pulmonary tuberculosis 7 years before developing scleroderma. The mystery of these “yeast-like” bodies deep in his skin was solved years later when I first learned about the existence of “large body” forms of Mycobacterium tuberculosis. When this patient died, Mycobacterium fortuitum, an “atypical” form of mycobacteria was cultured from his scleroderma tissue.

Bacteria are vital for our survival. They are hardy and the bacteria we carry will surely outlive us. The bacteria that cause cancer are the “simple” bacteria we carry with us. The cancer microbe is not an exotic microbe nor a rare one. However, bacteria can change form as the environment in our bodies changes. There is indeed a delicate balance between our bacteria and our immune system which allows these bacteria to live in harmony with us.

But when dis-ease occurs these microbes become aggressive, giving rise to a host of diseases, some of

which are cancerous, and others that are inflammatory, degenerative, or simply transitory. Another reason for physicians to doubt that a single type of germ could cause such a variety of pathologic effects.

Bacteria are ubiquitous and so are Russell bodies. And if Russell bodies prove to be bacteria, the reason for this becomes obvious.

The Russell body and the origin of cancer

In 1981 King and Eisenberg's article on "Russell's fuchsin body: 'The characteristic organism of cancer' " appeared in the American Journal of Dermatopathology. They reconfirmed that "Russell bodies have now been shown to be immunoglobulins." They remarked that Russell was not the first to describe them; and that similar bodies were reported by Cornil and Alvarez in rhinoscleroma five years earlier in a French journal in 1885. Declaring it ironic that these "bodies should bear the name of a man who so thoroughly misunderstood them", the authors ended by stating: "Hence, when the term Russell body is used today, one should be aware that the eponym is as inaccurate as was Russell's perception of their significance."

Unlike King and Eisenberg, I believe Russell was right on the mark. There is a parasite in cancer. It has been studied and reported by various scientists throughout the world for many decades, and a wealth of scientific information on the cancer microbe is available in medical libraries. For those with Internet capability, the words "cancer microbe" typed into Google.com will give instant access to a treasure trove of information on the subject.

There is no secret to cancer. In my view, the cause is staring us right in the face in the form of the Russell body. William Russell understood very well in the nineteenth century what medical science in the twenty-first century has yet to discover.

Alan Cantwell, M.D. is a retired dermatologist and cancer researcher. A number of his full-length papers on the microbiology of cancer can be found on the net at the Journal of Independent Medical Research web site (http://www.joimr.org/). He is the author of The Cancer Microbe, and AIDS and the Doctors of Death (both published by Aries Rising Press, Los Angeles). Correspondence address: PO Box 29532, Los Angeles, CA 90029, USA. Dr. Cantwell's books, including The Cancer Microbe, are available at:http://ariesrisingpress.com/books/

Back to top

Are Chronic Hidden Infections Causing Clogged Arteries?

NEWS ARTICLE from THE LOS ANGELES TIMES, 4-2-01, By JANE E. ALLEN

"Chronic infections tied to clogged arteries

Recurrent infections of the sinuses, lungs and urinary tract increase the risk of clogged arteries, a new

study has found, bolstering the theory that heart attacks and strokes have infectious origins.

Of 826 Italian men and women involved in the five-year study, those who had suffered from common chronic infections were about three times more likely to develop new fatty deposits in the principal artery carrying blood to the brain.

“Our study provides further strong evidence that chronic infection increases the overall risk of blood vessel disease,” said lead author Dr. Stefan Kiechl, a neurology professor at Innsbruck University Clinic in Austria.

Kiechl and his colleagues from Austria and Italy focused on changes in the carotid artery but also detected similar changes in other blood vessels, such as those of the heart.

They think the same process is at work in arteries of the neck, heart and legs. Problems occur when pieces of the fatty deposits break off and lodge in a blood vessel, cutting off blood supply and causing a heart attack or stroke ...

In the study, blood levels of C-reactive protein, a key indicator of inflammation often used to assess heart-attack risk, were elevated among people who had suffered chronic infections. The subjects studied were 40 to 79 and received ultrasound scans of carotid artery in 1990 and 1995.

“This is yet another piece of evidence implicating the potential role of inflammation and infection in atherosclerosis,” said Dr. P.K. Shah, cardiology chairman at Cedars-Sinai Medical Center in Los Angeles. He noted that the study also found a worsening of existing plaque, not just accumulation of new plaque ...”

Back to top

Is Heart Disease Caused by a Virus?

NEWS ARTICLE from THE WALL STREET JOURNAL, 9-19-00, By SCOTT HENSLEY, Staff Reporter of THE WALL STREET JOURNAL

`Study Finds Heart-Muscle Viruses Cause Chronic-Fatigue Syndrome

TORONTO — Viruses that insidiously damage heart muscle may be the cause of chronic-fatigue syndrome, a mysterious malady that many physicians have written off as a psychological condition, according to a provocative new study by an infectious disease expert.

At a major scientific conference here, Martin Lerner, a doctor at William Beaumont Hospital in Royal Oak, Mich., presented data on a series of patients, including himself, who developed the debilitating condition and were later treated, with apparent success, with potent antiviral drug regimens.

“It’s an infectious disease,” that primarily attacks the heart, Dr. Lerner declared at a meeting of the American Society of Microbiology.

Dr. Lerner said daylong cardiac monitoring found that 95% of chronic-fatigue patients he and his research team tested in two separate small studies had abnormal electrocardiograms indicative of heart damage.

Dr. Lerner said he suspects the heart damage is caused by Epstein-Barr virus and cytomegalovirus, both long implicated in the condition.

The damage to the heart occurred, he believes, when the viruses were held in partial check by the patients' immune systems. Though the immune systems appear to have kept the viruses from reproducing, Dr. Lerner said partial bits of the viruses that were being produced appear to be causing heart damage ...

A test for the hard-to-diagnose syndrome would represent a significant clinical advance.

Chronic-fatigue syndrome now is diagnosed by a rough checklist of symptoms and a process of elimination. The key clinical finding is that patients have persistent or relapsing fatigue for six months or more.

In fact, doctors have argued whether the syndrome is a disease at all, and even if it is, exactly how prevalent it might be. By some estimates, the syndrome affects about six in every 100,000 people.

After implicating viruses as the cause of the syndrome, Dr. Lerner tested possible treatments.

Many of the patients, including Dr. Lerner, who were infected by Epstein-Barr virus regained cardiac function and returned to normal life after taking high doses of valacyclovir (brand name Valtrex), an antiviral drug made by Glaxo Wellcome PLC, for several months.

Patients with cytomegalovirus received ganciclovir (Cytovene), another antiviral drug made by Roche Holding AG.

Dr. Lerner became interested in chronic-fatigue syndrome when he fell ill in 1988 at age 58. He thought at first that he had heart disease, and an examination by doctors confirmed that his heart was weak.

Later, he suspected there was more to the picture. In 1996, he began antiviral drug therapy and his heart function returned to normal.

The smoking gun in Dr. Lerner's investigation came from patient samples of heart tissue. The viruses had weakened their hearts by scrambling the normally well-ordered muscle fibers ... "

Back to top

Is the Human Papillomavirus Causing Cancers of the Head and Neck?

"HPV-16 a Possible Risk Factor for Squamous-Cell Carcinoma of the Head and Neck

Results of a nested case-control study suggest that infection with human papillomavirus type 16

(HPV-16) may be a risk factor for squamous-cell carcinoma of the head and neck.

Dr. Jon Mork of National Hospital in Oslo, Norway, and a multicenter European team report the finding in the April 12th [2001] issue of the New England Journal of Medicine.

They analyzed serum samples for antibodies against HPV types 16, 18, 33 and 73 from 292 patients who developed squamous-cell carcinoma of the head and neck an average of 9.4 years after enrollment, as well as from 1568 matched control subjects.

The researchers also measured serum cotinine levels, a marker of smoking, and used polymerase chain reaction (PCR) analysis to look for HPV DNA in tumor tissue samples from 160 of the cancer patients.

Dr. Mork's team detected HPV-16 DNA in 50% of oropharyngeal cancers and 14% of tongue cancers. After adjustment for smoking, HPV-16 seropositivity was associated with a 2.2 excess risk of squamous-cell carcinoma of the head and neck, the team reports ... "

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Back to top

Is a Cancer-Causing Virus Injected Into Millions of Americans in the 1950's and '60's Now Quietly Wreaking Havoc On Our Bodies?

NEWS ARTICLE from THE SAN FRANCISCO CHRONICLE, 7-16-01

"Growing Medical Fear Over Possible Carcinogenic Virus

SAN FRANCISCO — A growing number of medical researchers fear that a monkey virus that contaminated polio vaccine given to tens of millions of Americans in the 1950s and '60s may be causing rare human cancers.

For four decades, government officials have insisted that there is no evidence the simian virus called SV40 is harmful to humans. But in recent years, dozens of scientific studies have found the virus in a steadily increasing number of rare brain, bone and lung-related tumors — the same malignant cancer SV40 causes in lab animals.

Even more troubling, the virus has been detected in tumors removed from people never inoculated with the contaminated vaccine, leading some to worry that those infected by the vaccine might be spreading SV40.

The discovery of SV40 in human tumors has generated intense debate, pitting government health officials, who are convinced that the virus is harmless, against researchers from Boston to China who now suspect SV40 may be a human carcinogen. At stake are millions of research dollars and potential medical treatments for those afflicted with the cancers SV40 may be causing.

In April, [2001] more than 60 scientists met in Chicago to discuss the controversial virus and how it

works to defeat certain cells' natural defenses against cancer.

"I believe that SV40 is carcinogenic (in humans)," said Dr. Michele Carbone of Loyola University Medical Center in Maywood, Ill. "We need to be creating therapies for people who have these cancers, and now we may be able to because we have a target – SV40."

But scientists at the National Cancer Institute [NCI] say their studies show almost no SV40 in human tumors and no cancer increase in people who received the contaminated vaccine.

"No one would dispute there's been a widespread, very scary exposure to the population of potentially cancer-causing virus," said Dr. Howard Strickler, NCI's chief investigator. "But none of our studies and other major analyses have shown an inkling of an effect on the population."

Critics charge, however, that the few studies done by the government are scientifically flawed and that health officials have downplayed the potential risks posed by SV40 ever since they learned in 1961 that the virus contaminated the polio vaccine and caused tumors in rodents.

"How long can the government ignore this?" asked Dr. Adi Gazdar, a University of Texas Southwestern Medical Center cancer researcher. "The government has not sponsored any real research. Here's something possibly affecting millions of Americans, and they're indifferent."

"Maybe they don't want to find out." The recent SV40 discoveries come at a time of growing concern over the dangers posed by a range of animal viruses that have crossed the species barrier to humans, including HIV, which scientists now believe came from chimpanzees and ultimately caused the AIDS epidemic ...

During the first half of the 20th century, polio struck down hundreds of thousands of people, leaving many paralyzed – some in iron lung machines – and killing others. The worst year was 1952, when more than 57,000 polio cases were reported in the United States. Three thousand died.

Then on April 12, 1955, Dr. Jonas Salk, a slightly built, soft-spoken researcher from Pittsburgh, mounted the podium at the University of Michigan and announced that he had developed a vaccine. That afternoon, the government licensed the vaccine for distribution.

Salk's vaccine was made by growing live polio virus on kidney tissue from Asian rhesus monkeys. The virus was then killed with formaldehyde. When the vaccine was injected in humans, the dead virus generated antibodies capable of fending off live polio ...

Four years later, Bernice Eddy, a researcher at the National Institutes of Health, noticed something strange while looking through her microscope. Monkey kidney cells – the same kind used to make the vaccine – were dying without apparent cause.

So she tried an experiment. She prepared kidney extracts from eight to 10 rhesus monkeys and injected tiny amounts under the skin of 23 newborn hamsters. Within nine months, "large, malignant, subcutaneous tumors" appeared on 20 of the animals.

On July 6, 1960, concerned that a monkey virus might be contaminating the polio vaccine, Eddy took her findings to Dr. Joseph Smadel, chief of the NIH's biologics division. Smadel dismissed the tumors as harmless "lumps."

The following year, however, at a Merck laboratory in Pennsylvania, Dr. Maurice Hilleman and Dr. Ben Sweet isolated the virus. They called it simian virus 40, or SV40, because it was the 40th virus found in rhesus kidney tissue ...

At the same time, an oral polio vaccine developed by virologist Albert Sabin was in final trials in Russia and Eastern Europe, where tens of millions had been inoculated, and it was about to be licensed in the United States. Unlike the Salk vaccine, the oral version contained a live but weakened form of polio virus and promised lifelong immunity.

But U.S. Public Health Service officials were worried. Tests had found SV40 in both the Sabin and Salk vaccines – it was later estimated that as much as a third of the Salk vaccine was tainted – and that SV40 was causing cancer in lab animals.

In the spring of 1961, they quietly met with the agency's top vaccine advisers. The agency found no evidence that the virus had been harmful to humans, but in May, the officials ordered manufacturers to eliminate SV40 from all future vaccine.

New procedures were adopted to neutralize the tainted polio virus seed stock and SV40-free African green monkeys were used to produce the bulk vaccine instead of rhesus monkeys.

But officials did not recall contaminated Salk vaccine – more than a year's supply – still in the hands of the nation's doctors.

And they did not notify the public of the contamination and SV40's carcinogenic effect on newborn hamsters ...

The first public disclosure that the Salk vaccine was contaminated came in the New York Times on July 26, 1961. A story on Page 33 reported that Merck and other manufacturers had halted production until they could get a monkey virus out of the vaccine.

When asked to comment, the U.S. Public Health Service "stressed" there was no evidence the virus was dangerous ...

In Boston, two researchers stumbled on something disturbing. Dr. Robert Garcea and his assistant, Dr. John Bergsagel, were using a powerful new tool called polymerase chain reaction, or PCR, to look for a pair of common human viruses in children's brain tumors.

But a different DNA footprint kept popping up in more than half the tumors. They finally realized they were seeing SV40.

For more than a decade, scientists had reported sporadic findings of SV40-like proteins in human tumors. But the earlier tests were primitive and the results suspect. PCR, however, is capable of

amplifying infinitesimal fragments of DNA, which makes detections far more credible.

The findings were troubling. The researchers noted in their published report that the children were too young to have received the contaminated vaccine. But somehow the virus had infected them and embedded itself in their tumors.

That same year, Michele Carbone was surprised to find a milky, rindlike tumor in a laboratory hamster at the National Institutes of Health in Bethesda, Md.

The animal was one of a group given an SV40 injection directly into their hearts. Sixty percent of those hamsters developed the fatal cancer called mesothelioma.

Carbone, a postdoctoral fellow at the institute, knew that SV40 caused tumors in hamsters but only in specific locations where large doses of virus were injected. Here the mesothelial membrane lining the lungs apparently became cancerous from minuscule amounts of SV40 shed by the tip of the needle on the way to the hamsters' hearts.

So he tried another experiment, this time injecting SV40 directly into the thin mesothelial walls of another group of hamsters. Within six months, every animal developed mesothelioma.

Carbone was puzzled. Mesothelioma is a rare cancer. Few human cases were reported before the 1950s, but its incidence had been increasing steadily, reaching several thousand cases a year in the United States by 1988.

Studies had linked mesothelioma to asbestos exposure – with tumors usually appearing many decades later. Yet 20 percent of victims had no asbestos exposure.

Carbone decided to use PCR to test 48 human mesotheliomas stored at the NIH. He was stunned: 28 of them contained SV40.

PCR unleashed a wave of SV40 discoveries. By the end of 1996, dozens of scientists reported finding SV40 in a variety of bone cancers and a wide range of brain cancers, which had risen 30 percent over the previous 20 years.

Then, Italian researchers reported finding SV40 in 45 percent of the seminal fluid and 23 percent of the blood samples they had taken from healthy donors.

That meant SV40 could have been spreading through sexual activity, from mother to child, or by other means, which could explain how those never inoculated with the contaminated vaccine, such as the Boston children, were being infected …

Dr. Howard Strickler … led a study using PCR on 50 mesotheliomas from Armed Forces hospitals across the country. And he found no SV40.

Although the findings bolstered the government's long- standing position that SV40 did not appear to be a health risk, federal officials decided to convene a conference on the virus.

In January 1997, 30 scientists gathered at the National Institutes of Health in Maryland. Garcea, Carbone and others presented their evidence showing SV40 in tumors and pleaded for research funding.

Strickler presented his mesothelioma study, as well as new research he had just completed ...

This new study compared 20 years of cancer rates of people born between 1947 and 1963, and therefore likely to have been exposed to the contaminated polio vaccine, with people born after 1963, whom they believed weren't exposed.

The study found no significant difference between the two groups.

But when Susan Fisher read Strickler's ... study in the Journal of the American Medical Association, she fired off a letter of protest to the publication.

An epidemiologist at Loyola University Medical Center in Maywood, Ill., Fisher challenged the study's methodology, calling it "an error in judgment" and misleading.

Using the same 20-year national cancer database for the two groups, Fisher compared people of the same age – "because these cancers are highly correlated with age" – and she came up with very different results.

Studying 18- to 26-year-olds who probably had been exposed to the contaminated vaccine, Fisher found a 19.6 percent greater incidence of the two major brain cancers linked to SV40 when compared with the incidence in people the same age who were not exposed. She also found 16.6 percent more bone cancers and 178 percent more mesotheliomas among those exposed to the vaccine.

But Fisher cautioned against comparing the two groups. She argued that if SV40 is being transmitted and circulating in the population, then many people in the "unexposed" group would also be carrying the virus and that would undermine the comparison.

For years, researchers had believed that all SV40-contaminated Salk vaccine made between 1955 and 1963 had been used or discarded.

Then in 1999, Carbone was contacted by a former public health director in Oak Park, Ill., who said he had seven sealed vials of vaccine dated October 1955 in a refrigerator in his basement.

Carbone, who had left the NIH and joined the faculty at Loyola University Medical Center, ran tests on the vaccine and made a startling discovery: Not only was the vaccine contaminated, it contained a second form of the virus – an "archetypal" SV40 strain.

Although manufacturers switched from rhesus monkeys to SV40-free green African monkeys to grow the bulk vaccine in 1961, they have continued to use potentially contaminated polio seed stock grown on the rhesus monkeys tissue to start the bulk vaccine process.

Manufacturers checked the purity of their vaccine with a series of 14- day tests to detect whether any

SV40 slipped through.

But when Carbone replicated the tests, he found that the second, slower-growing "archetypal" strain took 19 days to emerge.

It was possible, Carbone noted in a published report, that this second strain of SV40 had been evading manufacturers' screening procedures for years – and infecting vaccine recipients after 1962.

Meanwhile, a new study led by Strickler had bogged down in bitter internal conflict.

After the NIH's 1997 conference, nine laboratories were recruited to participate in a government-sponsored study to determine if tests were really finding SV40 in tumors or whether earlier detections were the result of laboratory contamination.

Carbone and other researchers considered the study unnecessary. A similar multilab study led by Dr. Joseph Testa of Philadelphia had just been completed, and it virtually eliminated the contamination theory. The prestigious journal Cancer Research published Testa's findings in 1998.

But Strickler pressed on. An independent laboratory in Maryland prepared mesothelioma samples for nine participants.

When tests revealed almost no SV40 in the tumor samples, some participants questioned the preparation methods used by the Maryland lab. They also challenged Strickler's written conclusion implying that contamination had caused the earlier findings of SV40 in tumors.

If Strickler was right, the earlier SV40 detections were probably the result of stray SV40 in the labs. But critics argued that the study was scientifically flawed and should be scrapped.

The dispute became so contentious that FDA officials were forced to intervene and a neutral arbitrator assigned to mediate.

Finally, in early 2000, more than two years after the study was initiated, a carefully rewritten report emerged for publication.

It concluded that contamination was an unlikely explanation for earlier SV40 findings. Then it struggled to explain the discrepancy between earlier detections of SV40 in about half of all mesotheliomas tested and the fact that the nine labs found the virus in only slightly more than 1 percent of the study's tumor specimens.

The report noted that discrepancy might be because of the inefficiency of the method used by the Maryland lab to recover viral DNA – like the genetic sequences of SV40 – from the mesothelial tissue to create the test samples.

The Maryland lab also had inadvertently contaminated some of the laboratory controls and "theoretically" could have contaminated others.

The report concluded by calling for further research. Despite the study's ambivalent conclusions and

technical problems, the NCI submitted it to Cancer Research, the journal that had published Testa's study.

It was rejected. In laboratories around the world, researchers continued to find SV40 in a widening range of tumors that now included pulmonary, pituitary and thyroid cancers and some lymphomas.

Meanwhile, an NCI investigator named Dr. David Schrump was able to gut a common respiratory virus and use it to deliver genetic material called "antisense" into SV40-infected mesothelial cells and stop the cells' malignant growth.

His discovery, which was patented by the government, strongly suggested that SV40 contributed to mesothelioma and that a treatment might be possible.

Then in August, Carbone and several colleagues published a major study providing a "mechanistic" explanation of how SV40 contributes to the uncontrolled growth of mesothelial cells. The key, they found, was the large number of "tumor suppressor" proteins found in the mesothelial cells that makes them unusually susceptible to SV40.

In most human cells, they said, the virus reproduces itself and kills the infected cell in the process. But in mesothelial cells, SV40 is especially attracted to the "tumor suppressor" proteins and binds to them, knocking them out of action. The virus then lives on in the cell.

The result, they said, is a rate of malignant cell transformation in tissue cultures 1,000 times higher than has ever been observed.

In a paper published in the Proceedings of the National Academy of Science, Carbone further explained that asbestos fibers appear to act as a co-carcinogen in mesothelioma by somehow suppressing the immune system's response, which is designed to kill the infected cells.

Carbone and others believed that the time had come for another conference on the virus he calls "a perfect little war machine."

In April, [2001] more than 60 scientists gathered on a warm weekend at the University of Chicago's downtown conference center. Despite numerous faxes and certified letters inviting him, Strickler declined to attend.

Carbone opened the conference by confronting the question of whether SV40 is present in humans.

"Sixty-two papers from 30 laboratories from around the world have reported SV40 in human tissues and tumors," he said. "It is very difficult to believe that all of these papers, all of the techniques used and all of the people around the world are wrong."

For two days, scientists from as far away as China and New Zealand presented the results of their studies, with almost every speaker concluding that SV40 was present in the tissues they examined.

One of the newest discoveries came from Dr. Jeffrey Kopp, an NIH scientist who reported finding

SV40 in a high percentage of patients with kidney disease. The virus was also present, he said, in 60 percent of a new "collapsing" type of renal disease that was unknown before 1980 but has since increased rapidly in incidence.

There were also reports on efforts to develop a vaccine, recently funded by the NCI, that would allow the immune system to target and eliminate SV40.

At times, the meeting took on almost revivalist overtones as scientist after scientist said he or she was initially very skeptical of SV40's presence in human tumors but was now a believer.

"I was a hard sell," said Testa, the Philadelphia geneticist who conducted the first multilaboratory tests, noting that the study had convinced him.

Gazdar, the cancer researcher from Texas, showed a slide describing his own transformation: "Nonbeliever (arrow) Believer (arrow) Zealot."

The conference concluded with a consensus among the leading scientists that SV40's presence in human tumors was no longer in question. They were more circumspect about the virus's possible role in causing cancer.

If SV40 is a human carcinogen, they said, the virus probably requires interaction with other cancer-causing substances like asbestos.

Dr. Janet Butel from Baylor Medical College in Houston said that it simply might be too soon to make a determination, citing the many years it has taken to establish that other viruses cause cancer.

But even renowned tumor biologist George Klein from Sweden said he was impressed by Carbone and Schrump's work.

"This strongly suggests that the virus plays a role (in causing tumors)," said Klein, a former chairman of the Nobel Assembly.

In May, shortly after the conference, Strickler's multilab study was published in a small journal called Cancer Epidemiology, Biomarkers & Prevention.

Carbone and other SV40 experts dismissed the study. "A garbage paper in a garbage journal," said Garcea, now on the faculty at the University of Colorado School of Medicine.

But Strickler strongly defends the study. He said it was the first to use strict controls not used in other studies. He acknowledged, however, that the study "doesn't prove that SV40 is not out there."

Strickler, who now teaches at Albert Einstein School of Medicine in New York, said he remains skeptical about whether SV40 has infected humans, a suspicion he says that is shared by the broader scientific community.

But a recent NCI statement acknowledges that there is evidence to suggest that SV40 "may be associated with human cancer." The statement, released last month, also said that SV40's interaction

with “tumor suppressor proteins” indicates “possible mechanisms that could contribute to the development of cancer.”

Top NCI officials declined to be interviewed on the record for this report … ”

Back to top

Can a Common Bacteria Found in Ticks Cause Cancer in Humans?

NEWS ARTICLE from SCOTLAND ON SUNDAY, 9-25-00, By Tom Peterkin, Health Correspondent

“Sheep tick can pass rare skin cancer to people

SCOTTISH doctors have discovered that the humble sheep tick is responsible for passing a rare but deadly form of skin cancer to humans.

The new research has heralded the prospect of the cancer being tackled by antibiotics instead of more aggressive therapies.

Researchers at Raigmore Hospital in Inverness have identified a link between a B-cell lymphoma skin cancer and bacteria transmitted by the parasitic blood-suckers.

It has been known for some time that the bacterium Borrelia burgdorferi is responsible for passing on Lyme disease to humans. But doctors in Inverness are the first to identify a significant relationship between the tick-borne bacterium, which is a distant cousin of syphilis, and cancer.

Dr John Goodlad of the department of pathology at Raigmore Hospital said: “We knew that because there was a high incidence of Lyme disease in the Highlands if there was going to be a relationship between B-cell lymphoma and cancer, we were going to find it here.

“We did a large number of controls, so we know that it was not just a chance result. Our findings showed a statistically significant association and gave us evidence that the organism was present. This is the first time that the link has been shown in the UK and it is certainly the first British study of this.”

It is estimated that the Scottish Highlands has one of the highest rates of Lyme disease in Northern Europe. In the north of Scotland, there are 16 cases per 100,000 people each year.

“But that is probably a gross underestimation,” said Goodlad. “It is probably much higher than that, because it is not a notifiable disease. Not all cases are reported and no one has done an epidemiological study to look at it thoroughly.”

Borrelia burgdorferi triggers the unpleasant symptoms of Lyme disease such as arthritis, inflammation of the heart and brain, and skin rashes.

The disease took its name from the town of Lyme in Connecticut, where it was first recognised in 1975. Since then it has been recorded all over Europe and North America.

But the new link with skin cancer was only revealed when 20 B-cell lymphoma patients were compared with other skin cancer victims at Raigmore.

The researchers found that a significant number of those with lymphoma carried burgdorferi-specific DNA.

The research … has been published in this month's edition of the American Journal of Surgical Pathology … "

Scotland on Sunday

Back to top

Are 1.2 Million New Cases of Cancer a Year Being Caused by Infections?

Infections and Human Cancer Microbes and Malignancy: Infection as a cause of human cancers

New England Journal of Medicine Volume 341:1628-1629 November 18, 1999 Number 21

(Cancer Surveys. Vol. 33.) Edited by R. Newton, V. Beral, and R.A. Weiss. 396 pp. Plainview, N.Y., Cold Spring Harbor Laboratory Press, 1999. $93. ISBN 0-87969-549-8. Edited by Julie Parsonnet. 465 pp., illustrated. New York, Oxford University Press, 1999. $75. ISBN 0-19-510401-3.

In the opening chapter of Infections and Human Cancer, Parkin and colleagues estimate that in 1990 there were 1.2 million new cases of cancer worldwide that were due to infectious agents. This figure accounts for about 15 percent of all cancers. These cases plus the sum of all neoplasms caused by tobacco account for the largest number of preventable cancers in the world. Infectious agents cause almost one fourth of all cancers in developing countries, a reflection of the high carrier rates in these regions for the four agents that cause 90 percent of cancers due to persistent infection

Back to top

Does This Common Virus Explain the High Rates of Breast Cancer in Europe and America?

"Common virus might explain high rates of breast cancer in developed countries"

Prof. Ann Richardson and colleagues at Otago University's Christchurch School of Medicine are exploring the link between breast cancer and two viruses that thrive in modern civilization: Cytomegalovirus (CMV) and epstein-barr virus (EBV). Once thought to be a relatively benign germ, EBV is the cause of mononucleosis, the "kissing disease" contracted by many teens.

More than a dozen viruses are now known to be the direct cause of malignant tumors. The U.S. government has just added viruses and bacteria to its list of known carcinogens.

It is beginning to look like a new and different "DaVinci Code" has just been discovered and it is just

as hot, just as controversial… but this one explains why you may get cancer or Alzheimer's disease. And how you will die.

An intellect of incredible power, Leonardo DaVinci desperately sought the secret code of life, and, in turn, the way to defy early death from disease. It was his good fortune that his contemporary, Paracelsus, provided the key to this code: Germs.

He had discovered that diseases and most of the disabilities of old age are caused not by angry spirits, foul vapors or ill will, but by invading biological agents: What we now call bacteria and viruses.

The real DaVinci Code secret may be inside you right now. It is a bloodline of microbes as old as mankind itself. It is not the flu, it is not the West Nile bug, it something more sinister and final. It is the contagious seed of cancer. It will ultimately kill half of everyone living today.

A grand unified germ theory, thanks to the genius of people like Leonardo DaVinci, has begun to explain what cancer really is, why it spreads, and why we haven't been able to cure it despite our best efforts. More than a mystic code — it is a common thread that ties together hundreds of brand new medical studies. The evidence is now in, and it is stunning:

Cancer is no accident of nature. It is nature itself at work. It is caused by a stealthy infection that grows slowly. Cancer can take years, decades, to flare up — but you can pick up a tumor germ in roughly the same way that you catch a cold: From another person.

What is a virus anyway? One can look at it as just a seed. A cancer spore: What a tumor uses to create another tumor. Me to you, you to me.

A tumor is a living foreign organism within your body. A separate species. A parasite in every sense of the word. It communicates with its offspring as it spreads.

The body has evolved unusual ways to fight these parasites; your body is fighting them right now. But you can defeat your own defenses by doing something stupid. (Like taking certain vitamin pills!)

New research shows how the code has been unlocked to reveal the actual cause for chronic diseases formerly blamed on the failings of our own bodies. As a result, actual cures may be coming for ailments never before thought to be contagious: Crohn's disease, Alzheimer's, multiple sclerosis, leukemia, and others.

The original definition of an "autoimmune disease" like rheumatoid arthritis or lupus went something like this: "The body must be attacking itself, for we have found no germ at the center of the inflammation…" Well, it may be time to seriously question that autoimmune concept. We have now found the germs.

Back to top

Is Coxsackievirus B a Secret Cause of Insulin-dependent Diabetes Mellitus?

Is insulin-dependent diabetes mellitus caused by coxsackievirus B infection? A review of the epidemiologic evidence.

Barrett-Connor E.

The evidence that coxsackievirus B plays a causal role in the etiology of insulin-dependent diabetes mellitus (IDDM) is reviewed. This hypothesis is biologically plausible; one variant of another picornavirus, encephalomyocarditis virus, causes diabetes in genetically susceptible mice, but prior infection with another, serologically indistinguishable variant prevents this. The seasonal distributions of infection due to coxsackievirus B and of IDDM are similar. Case reports document coxsackievirus B infection coincident with the onset of IDDM. More than one-third of patients with recent-onset IDDM have antibody to coxsackievirus B but have not necessarily had recent infection. Case-control studies show no consistent association, with odds ratios ranging from 0.7 to 20. In two prospective studies, IDDM did not follow coxsackievirus B epidemics, but the sample sizes were small. Although epidemiologic data are too inconsistent to allow one to conclude that coxsackievirus B is a frequent cause of IDDM, methodologic difficulties–illustrated in the animal model–suggest that it would be premature to discard this hypothesis.

PMID: 2988099 [PubMed – indexed for MEDLINE]

Back to top

Is Cytomegalovirus Infection Causing Post-Surgical Diabetes in Transplant Patients?

Several types of viral infections have been associated with increased risk of diabetes mellitus [1,2]. Enteroviruses are among the most studied environmental triggers of type 1 diabetes, but also other viruses such as the rubella virus, mumps virus, EpsteinBarr virus, varicella zoster virus and cytomegalovirus (CMV) have been suggested to be associated with type 1 diabetes [1].

Recently, hepatitis C virus (HCV) has emerged as an important risk factor of type 2 diabetes and new-onset posttransplantation diabetes mellitus (PTDM) [2]. Insulin resistance secondary to hepatic steatosis, or elevated levels of pro-inflammatory cytokines such as TNF- [2], may explain the latter relationship. Others have argued that both insulin resistance and insulinopaenia are involved in the pathogenesis of HCV-associated glucose intolerance [3].

In the present article we address a possible relation between CMV infection and new-onset PTDM in renal transplant recipients, and potential pathogenetic mechanisms will be discussed in detail.

[NDT Advance Access originally published online on July 26, 2005
Nephrology Dialysis Transplantation 2005 20(11):2311-2315; doi:10.1093/ndt/gfi033]

Back to top

Is More Than 52% of Stomach Cancer Being Caused by H. Pyloria Bacterial Infection?

The authors acknowledge that Pisani et al.'s estimate that 52.6 to 59.5% of stomach cancer is caused by Helicobacter pylori is too low because "recent data indicate that the risk associated with the bacteria may have been underestimated due to spontaneous eradication in the precancerous stomach, potentially increasing the fraction of gastric cancer attributable to H. pylori to more than 75%.

[Infections as a major preventable cause of human cancer. H Kuper, HO Adams, D Trichopoulos. J Intern Med 2000 Sep;248(3):171-183.)]

Back to top

Can Mosquitoes and Biting Flies Be Passing Infectious Microorganisms to Humans and Causing Chronic Disease?

The article is titled "The Etiologic Agent of Lyme Disease in Deer Flies, Horse flies, and Mosquitoes"

DISCUSSION:

This is the first report of B. burgdorferi in horse flies, deer flies and mosquitoes.

....the number of infected deer flies and horse flies varied with the species and sampling areas....Also, like mosquitoes and other biting insects, the blood- feeding behavior of female tabanids differs within and between species, and infection may be correlated, in part, with the quantities of blood ingested.

... Serological studies of mammals and identification of B. burgdorferi have established that this agent is widely distributed within given habitats in the United States and that closely related strains exist in Europe. The presence of this bacterium in tabanids and mosquitoes increases the risk of Lyme disease in tick infested areas.

These and other blood sucking arthropods should recieve further consideration in ecological and epidemiological studies of this disease and of related disorders. "

(The Journal of Infectious Diseases. Vol. 154, No. 2 . August 1986. Louis A. Magnarelli, John F. Anderson, Alan G. Barbour)

Back to top

Can Urinary Tract Infections Cause Kidney Stones?

What causes kidney stones?

Doctors do not always know what causes a stone to form. While certain foods may promote stone formation in people who are susceptible, scientists do not believe that eating any specific food causes stones to form in people who are not susceptible.

A person with a family history of kidney stones may be more likely to develop stones. Urinary tract infections, kidney disorders such as cystic kidney diseases, and certain metabolic disorders such as hyperparathyroidism are also linked to stone formation.

(National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health; https://www.niddk.nih.gov/)

Back to top

Is a Herpes Virus Triggering Multiple Sclerosis in Many People?

ARTICLE from ViraCor Diagnostic Labs, By Dr. Donald Carrigan, Director of the Laboratory, and Dr. Konstance Knox, Director of Research,

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HHV-6 and Multiple Sclerosis

BACKGROUND

Multiple sclerosis (MS) is a chronic disease of the central nervous system, i.e. brain and spinal cord, that is caused by progressive loss of myelin, the main insulating material for the nerves that are responsible for the functioning of the Central Nervous System (CNS).

The reason for the destruction of the myelin in the CNS tissues of patients with MS is unknown. However, risk for developing MS is related to the major histocompatibility complex, and a virus infection of the CNS has long been suspected to be involved as a trigger of the disease process.

Over the past few years evidence has been obtained by our laboratory and other laboratories in the United States and Europe that suggests involvement of a specific member of the herpesvirus family, human herpesvirus six or HHV-6, in MS.

HHV-6 is a frequent cause of severe and often fatal CNS infections in immunologically normal people as well as in patients with various forms of immunodeficiency. In those cases in which it has been examined, the damage to the CNS tissues associated with active HHV-6 infections has been diffuse or focal demyelination, similar to that seen in MS.

STUDIES BY ViraCor Diagnostic Labs

In studies performed by our laboratory (Abstract Number 1960; 39th Interscience Conference on Antimicrobial Agents and Chemotherapy and manuscript in press), we have documented that at least 59% (36/61) of patients with MS have active HHV-6 infections compared to 2% (2/89) of healthy control subjects and patients with other forms of neurologic disease. This difference is highly

statistically significant (p less than 0.0001 by two-sided Fisher's Exact Test).

HHV-6 in MS CNS Tissues

More specifically, using an immunohistochemical staining procedure with brain tissues from patients with MS, we documented that active HHV-6 infections are not only present in the brains of most such patients, but that they are very closely involved in the actual damage being done to the brain tissue.

Only two other neurologic disease controls had cells actively infected with HHV-6 in their CNS tissues, and both of these were diagnosed as having HHV-6 encephalitis. The vast majority of the MS CNS tissues containing active demyelinative disease contained cells actively infected with HHV-6 compared to only a few of the tissues free of active demyelination (p less than 0.0001 by two-sided Fisher's Exact Test).

HHV-6 in MS LYMPHOID TISSUES AND BLOOD

Somewhat surprisingly, we also found that the active HHV-6 infections were present in the lymphoid tissues such as spleen and lymph nodes in most patients with MS but not in the lymphoid tissues of normal controls (p less than 0.015).

Consistent with this observation, we have also documented by means of a rapid HHV-6 culture assay that active HHV-6 infections are present in the blood of most patients with MS, but not in the blood of normal controls (p less than 0.0001). There was no significant difference between the incidence of active HHV-6 infection in the bloods of MS patient with chronic progressive and relapsing / remitting MS.

CRITICAL REVIEW of HHV-6/MS Scientific Literature

In order to evaluate the results of these studies and place them in the context of other published reports on the role of HHV-6 in the pathogenesis of MS, a MEDLINE search of the National Library of Medicine for two MESH headings [(1) Multiple Sclerosis and (2) Herpesvirus Six, Human] was performed. A total of 39 articles were identified by this search, and these were classified as follows:

(1) 15 review articles/commentaries,

(2) 2 case reports,

(3) 5 basic science reports and

(4) 17 diagnostic technique applications.

The articles describing diagnostic technique applications were retrieved and analyzed with respect to the methodologies used and the conclusions drawn. A manuscript describing these findings is currently in press, [10-17-00].

POLYMERASE CHAIN REACTION (PCR) OF CELLULAR SAMPLES

In studies using diagnostic technologies that could not distinguish between active and latent HHV-6

infections, i.e. PCR analysis of blood leukocytes, CSF containing cells or CNS tissue, essentially no differences were found between samples from patients with MS and control individuals.

Interpretation of these negative data with respect to the pathogenesis of MS is unclear and is made even more uncertain by the wide range of positive results seen with normal cells and tissues, e.g. normal leukocyte positivity rates of 5% to 95% and normal CNS tissue positivity rates of 15% to 85%.

CLASSICAL SEROLOGY

Studies in which classical serological methods were used, i.e. viral specific serum IgG titers and viral specific serum IgM detection, suggest a special role for HHV-6 in MS.

Six of eight studies showed either increased HHV-6 IgG titers or a higher rate of positive HHV-6 IgM in MS patients compared to controls. An additional study detected HHV-6 IgG in samples of CSF from patients with MS but not in controls, supporting the idea of a special role for the virus.

It should be noted that, while the serological methods used in these studies suggest a special role for HHV-6 in MS, results obtained for any one individual must be interpreted with caution since healthy individuals can have high titers of HHV-6 IgG and can occasionally be positive for HHV-6 IgM. Conversely, patients with MS who have an active HHV-6 viremia may have low titers of HHV-6 IgG and may be negative for HHV-6 IgM.

POLYMERASE CHAIN REACTION (PCR) of ACELLULAR SAMPLES (SERUM and CSF)

PCR analysis of serum samples yielded mixed results with some studies suggesting a special role for HHV-6 in MS and others failing to show such an association.

Importantly, the two negative serum PCR studies failed to include positive serum controls, raising the question of whether their procedures could detect HHV-6 DNA in serum. Also, neither of these two studies controlled for inhibition of the PCR reaction by substances in the patient specimens.

Three of four studies using PCR analysis of acellular CSF samples demonstrated a clearly increased positivity of the MS patients compared to other neurologic disease (OND) controls.

While the negative acellular CSF study used a control for PCR inhibition, the control DNA used was 500 times higher than the lower limit of sensitivity of the PCR technique. Significant inhibition of the PCR reaction could have occurred that would not have been detected.

The three positive investigations using PCR analysis of acellular CSF samples deserve special comment. In the three studies, 14% (3/21), 11% (4/36) and 17% (2/12) of CSF samples from patients with MS were positive for HHV-6 DNA demonstrating active infection within their CNS tissues.

This reduced rate of positivity compared to that observed with immunohistochemical staining of CNS tissues probably reflects the relatively low level of active infection present in the CNS tissues of MS patients compared to that seen in the brains of immunocompromised patients with HHV-6 encephalitis.

It is well known that analysis of CSF samples by PCR in cases of herpes simplex encephalitis can give false negative results if the sample is obtained too early in the disease course when the infection is focal and limited in size.

IMMUNOHISTOCHEMICAL STAINING of MS CNS TISSUES

All three independent studies using immunohistochemical staining of CNS tissues from patients with MS and controls, demonstrated active HHV-6 infections only in the MS patients. Also striking is the fact that the percentage of MS patients who were positive in the three studies were quite similar, i.e. 50% (16/32), 73% (8/11) and 47% (7/15).

CONCLUSION

Thus, when appropriate diagnostic technologies are used, i.e. those that detect only active HHV-6 infections, a strong relationship between HHV-6 and the pathogenesis of MS is reproducibly observed.

In summary, this work from our laboratory, in combination with previous work by other investigators, demonstrates that a sizable proportion of patients with MS suffer from active, disseminated infections of HHV-6, including infection of their CNS. We believe that this infection is the fundamental cause of the disease which raises the possibility that MS may be amenable to effective treatment with pharmacologic agents capable of suppressing the replication of HHV-6.

Back to top

Interferon Is An Anti-Viral Drug. So Why Does It Work Against Multiple Sclerosis?

NEWS ARTICLE from REUTERS HEALTH, 12-4-00

[Interferon is an anti-viral. Why does it work against MS?]

“WESTPORT, CT, Interferon-beta-1a treatment can significantly slow declines in memory and information processing ability in patients with relapsing multiple sclerosis (MS), according to a report by the Multiple Sclerosis Collaborative Research Group.

“This is the first study of any treatment showing we can decrease the chances of the patients becoming cognitively impaired,” Dr. Dennis N. Bourdette, of the Oregon Health Sciences University, in Portland, told Reuters Health.

“The untreated natural history of MS is that 10 to 15 years after onset, over 50% of patients are unemployed because of the MS,” Dr. Bourdette pointed out. “The biggest single factor in becoming unemployed is cognitive impairment…By instituting treatment early and decreasing the risk of developing cognitive impairment, we’ll keep more patients employed longer. ”

A total of 166 patients who had symptoms for at least 1 year and at least 2 documented exacerbations

were treated with placebo or interferon-beta-1a (Avonex) 30 mcg intramuscularly once weekly for 2 years. Dr. Bourdette and associates report their results in the Annals of Neurology for December.

“It’s interesting,” Dr. Bourdette commented, “that the treatment effect on delaying cognitive impairment was more robust than the effects on physical impairment.”

After 2 years, results on the information processing and memory component of the comprehensive neuropsychological battery were significantly better in the treatment group than in the placebo group …”

https://www.medscape.com/pharmacists

Back to top

Is There Even *More* Evidence Linking Viral Infection With Risk of MS?

NEWS ARTICLE from REUTERS, 5-8-01

More Evidence Links Viral Infection With Risk of MS

WESTPORT, CT (Reuters Health) May 08 -, 2001 Late infection with common viruses is associated with an increased risk of multiple sclerosis, according to findings from a case-control study nested within the Nurses’ Health Study II.

Dr. Miguel A. Hernan, of the Harvard School of PubliDr. Miguel A. Hernan, of the Harvard School of Public Health in Boston, and associates identified 301 women with MS and matched them with 1416 healthy controls. The subjects completed questionnaires regarding such issues as lifetime history of various viral diseases and exposure to pets.

The odds ratio of a subject with a history of infectious mononucleosis developing MS was 2.1, the researchers report in the May issue of Epidemiology. For mumps or measles after age 15, the odds ratios were 2.3 and 2.8, respectively.

“Whether these viruses cause the elevated risk or are only surrogates for the actual etiologic exposure cannot be determined from our findings,” Dr. Hernan’s group cautions.

The investigators observed no association of MS with other common viral diseases, exposure to canine distemper virus, cat ownership, birth order or number of siblings. The risk of MS was moderately increased among dog owners, “but the 95% confidence interval was wide.”

Epidemiology 2001;12:301-306.

Back to top

Is Anti-Biotic Resistant E. Coli Slowly Infecting Humans Through the Food Chain?

NEWS ARTICLE from REUTERS, 7-17-01

“Spread of Antibiotic-Resistant E. coli From Animals to People May Be Common

LONDON — Antibiotic-resistant Escherichia coli is commonly spread from animals to people in the Netherlands, according to a report in the June [2001] issue of the Journal of Antimicrobial Chemotherapy.

Dr. E. E. Stobberingh and colleagues from the University Hospital Maastricht in the Netherlands analyzed the prevalence of resistance in faecal E. coli in 47 turkeys and 50 broilers commonly given antibiotics and in 25 laying hens that were infrequently treated with antibiotics.

To examine the “possible dissemination of resistant E. coli or resistance genes from these poultry populations to humans,” the researchers examined faecal samples from individuals who had contact with these animals. This included 47 turkey farmers, 51 broiler farmers, 25 laying-hen farmers, 47 turkey slaughterers and 46 broiler slaughterers ...

Turkeys and broilers had a significantly higher prevalence of resistant E. coli than the laying-hen population. Turkey and broiler farmers and turkey and broiler slaughterers had a higher resistance to nearly all antibiotics than laying-hen farmers ... ”

(J Antimicrob Chemother 2001;47:763-771)

Back to top

Is a Retro-Virus Causing Schizophrenia?

NEWS ARTICLE from REUTERS, 4-9-01

“Retroviruses Implicated in the Pathogenesis of Schizophrenia

Retroviruses may play a role in the development of schizophrenia in some individuals, scientists from the US and Germany report in the April 10th issue of Proceedings of the National Academy of Sciences.

In the report, Dr. Robert H. Yolken, of Johns Hopkins School of Medicine, in Baltimore, and colleagues say that they have identified retroviral RNA in the cerebrospinal fluid from 29% of patients with recent-onset schizophrenia and from 5% of patients with chronic schizophrenia.

The majority of the nucleotide sequences present in the schizophrenic patients belonged either to the human endogenous retroviral (HERV)-W family or to the murine leukemia retrovirus family. Frontal cortex tissue obtained postmortem from schizophrenics also showed differential up-regulation of the HERV-W family of retroviruses.

In contrast, no retroviral sequences were present in CSF in any of the normal control subjects or in controls with noninflammatory neurologic illnesses ...

“... most retroviruses can be activated by a number of environmental factors, including infection with

other viruses such as herpesviruses," Dr. Yolken [said].

In a clinical trial about to begin in the Baltimore area, Dr. Yolken and his colleagues will study the treatment of herpesvirus infections as a way of suppressing retroviral transcription and hence the symptoms of schizophrenia ... "

(Proc Natl Acad Sci USA, 2001;98:4293-4294,4634-4639)

Back to top

Can Psychiatric Illnesses Be Caused by Hidden Infections?

FEATURE ARTICLE from The Washingtonian, 1-91, By Neil Raven

"Bicycle Boy — His Behavior Was Compulsive, It's Origins Unknown; Then a Good Doctor Seemed to Make a Miracle Happen

He was 12 years old, and every day he pedaled furiously on his stationary bicycle for as many hours as they would allow him. He was so absorbed in his effort that it was all they could do to get him to stop for meals.

In fact, before he was hospitalized at a psychiatric institution he had been unwilling to stop for meals, for school work, for the simple exchanges of ordinary life. At age 12, he had lost almost 30 pounds. He looked, in the language of the ward, cachetic, or in the language of his friends, as if he had been an inmate in a concentration camp.

His parents, after all the agonizing, had coaxed him into a car and driven him out to the facility , where they had carried his suitcase as they walked him to the ward. And they had handed their son over to the care of others, out of desperation, convinced that he was now beyond their help – their son who wanted only to pedal, to exert himself and withdraw from the world he had once embraced with such sunny exuberance.

The psychiatrists questioned the parents and the boy – the skeletal, restless boy, who not so long before had been a good student, a healthy, happy son. He had been a wonderful athlete, an exciting soccer player, but he had had some knee problems. Over two years he had had four episodes in which his right knee swelled enough to require treatment.

It was after the last episode that he had withdrawn. He spent most of his time alone in his room, fiddling with a ham radio, not talking to his friends or his parents. He stopped doing his homework. And then came the exercising, the disinterest in food, the weight loss.

At a glance, the boy reminded the psychiatrists of the young women who suffered from that dreaded and potentially lethal psychiatric condition, anorexia nervosa. He had that bony look, that restless hyperactivity.

But he was male, which is unusual for anorexia nervosa patients. And he was only 12 — most patients with anorexia nervosa are older. It could be a working diagnosis. But when things don't quite fit the pattern, you ask questions. You call in more opinions. They called in Andrew Pachner.

Andrew Pachner looks over to the framed photograph on the wall of his office at the Georgetown university Hospital's neurology department. The photo is a blowup of a single Borrelia burgdorferi spirochete – a microorganism that bears a striking resemblance to the organism that causes syphilis …

[Dr. Pachner] recalls the day he first laid eyes on the 12-year-old bicycle boy. Pachner was then a junior faculty member in the Department of Neurology at the Yale School of Medicine, living on a salary that didn't even approach subsistence level. While the university looked the other way, all the junior faculty members moonlighted to pay the rent. Among Pachner's stints was a job evaluating patients at the psychiatric institute.

Not all patients were selected by the psychiatrists for Pachner's review. But the bicycle boy was. For one thing, there were those swelling episodes and the probable history of arthritis.

While he was still in training, Pachner had drifted down to the Yale arthritis clinic. Diseases of the joints might seem an unlikely source of fascination for a doctor specializing in diseases of the nervous system, but there was a vital connection. Diseases of both are often [sometimes?] caused by mistakes that cause the immune system to turn against itself – autoimmune diseases.

The doctors studying arthritis were happy to have Pachner around. Many of their arthritis patients were suffering from autoimmune diseases, such as systemic lupus erythematosus, which have neurologic complications. Pachner's neurology expertise was welcomed.

While Pachner was examining patients in the arthritis clinic, he became an interested bystander to one of the most celebrated moments in medicine – the identification of a new disease.

… a group of children in Old Lyme, Connecticut, not far from Yale … had a curious form of arthritis that followed the appearance of a peculiar and characteristic skin rash called erythema chronicum migrans, or ECM. [first described in this country by Dr. Scrimenti in Wisconsin, in 1970.]

… In 1982, Drs. Willy Burgdorfer and Alan Barbour, working at the Rocky Mountain Laboratory in Montana, pinpointed the cause of the disease.

… [The] young patients had an arthritis caused by a spirochete. Unlike bacteria, spirochetes are not easily grown in the laboratory. The standard way to study a microorganism is to grow it on a special broth, a culture plate.

But spirochetes, like exotic zoo animals, do not live long outside their native habitats. Once outside the body, they die. The human body makes antibodies to the organism , which makes diagnosis possible, but the antibody tests can be tricky, and occasionally misleading.

The world's best-known spirochete is Treponema pallidum, which causes syphilis. The one that causes Lyme disease would prove to be an even bigger problem than syphilis in some ways, because people could not avoid it by abstemious behavior.

It was a spirochete that awaited children as they ran through the Connecticut woods, doing what their parents thought was healthy and good. The spirochete was carried by forest animals, and it waited for the unsuspecting, anyone who cared to enjoy the great outdoors: hikers, pregnant women toting little kids, fishermen, gardeners, and farm workers. It was the tick-borne spirochete that causes Lyme disease.

The bicycle boy had had his first attack of Lyme arthritis in 1982, two years before Pachner discovered him pedaling away on the psychiatric ward.

Pachner was aware that syndromes similar to Lyme arthritis, syndromes suspected to be caused by an infectious agent, had been described in Europe, and he knew these syndromes often included some neurological features, usually a form of radicular pain , which radiates down an arm or a leg. Radiculitis meant the trouble was in peripheral nerves, which flow to and from the spinal cord out to the extremities.

But none of the these arthritis-related European syndromes involved the central nervous system. None of these European syndromes caused complex behavioral changes, and no connection had ever been drawn between an infectious arthritis and any sort of neurological disease that might affect a person's behavior.

In order to cause a behavioral change, a disease has to affect the brain directly and in a widespread fashion. Various forms of vasculitis – inflammation of the small blood vessels – can do this. Autoimmune diseases can do this.

But none of the infectious – arthritis group of diseases were known to be capable of involving the whole brain. Focal lesions can "stroke out" particular functions, causing paralysis, speech deficits, or sensory loss, but the entire brain must be involved for memory deficits, disorientation, or obsessive behavior to occur.

Clearly, what was going on in the bicycle boy was amore than a simple radiculitis: in which only a single nerve root would be affected.

By 1982, physicians in Connecticut had been alerted to the possibility of Lyme arthritis, and the boy's first attack of knee pain had been treated with a form of tetracycline. But two years later, when the boy started to withdraw from life, started to become a behavior problem, his physicians made no connection between his psychiatric symptoms and his earlier episodes of arthritis.

"Lyme arthritis" was a disease of the joints or, at most, of the skin and the joints: nobody had any basis for suspecting a connection between the knee and brain disease – except perhaps for Andrew Pachner.

… Pachner had begun to uncover neurological symptoms and findings in his Lyme arthritis patients. Another neurologist, Louis Reik, who had preceded Pachner in the arthritis clinic, had passed on his suspicions that the Lyme patients might have more than simple radiculitis complaints. But it was up to Pachner to push ahead with his observations.

Pachner connected the symptoms of the European patients to the new, more diverse symptoms he was seeing in the Yale clinic. Reading through the chart of that 12-year-old boy, Pachner began to get excited.

Could this boy have an infection that affected not just his knee but his brain as well? The organism identified as causing Lyme arthritis was a spirochete. Syphilis was a spirochete, and what syphilis could do to a brain was well-known. It could cause dementia, bizarre pain syndromes, a whole variety of symptoms so diverse that medical students are taught to think of syphilis as the “great imitator”.

Syphilis mimics many diseases because it can affect so many organs: heart, brain, joints, nerve, eye. Wherever blood goes, syphilis can go. Syphilis can cause a vasculitis of the small blood vessels in the brain, the eye, almost anywhere. Could this new spirochete, this borrelia burgdorferi, be as strange and protean in its manifestations as the “great imitator” itself?

Could it be, thought Pachner, that this bicycle boy has Borrelia in his brain?

If the spirochete that causes syphilis can enter the body through genital tissues, multiply, migrate to small branches of the vascular tree, migrate through the thin blood-vessel walls, and set up house in the brain and nervous tissue, and in heart tissue and aorta, was it so farfetched to believe that the Lyme spirochete might do something similar?

Might it enter the body through a break in the skin caused by an insect bite, the way malaria does, enter the blood stream, and multiply first in a knee joint causing arthritis, and then wreak havoc years later in the brain, as syphilis has been known to do?

Not having an answer, not having solid evidence or similar cases, Pachner could not voice his suspicions to the boy’s parents. He spoke instead to the psychiatrists and asked them to transfer the boy to Yale – New Haven Hospital. The parents were told simply that there was a chance the therapy at Yale could help their son. They were willing to try anything.

When the boy arrived at the hospital, he was taken to the neurological ward . Pachner met his parents and explained that he believed there might be a connection between their son’s previous bouts of arthritis and the problems that had landed him on the psychiatric ward. But Pachner could make no promises–they were in uncharted waters.

The boy’s parents did not know what to say. Their son’s strange course had been so baffling, their odyssey through the psychiatric wards so bizarre, they could accept anything. They had no choice but to hope that Andrew Pachner was correct.

On the neurology ward, Pachner did a lumbar puncture on the boy, inserting a needle into the midline

of his back, passing it between the vertebral bones to the fluid-filled sac called in which the spinal cord floats. Examining the fluid, called cerebrospinal fluid, or CSF, Pachner noted a profusion of immune cells called lymphocytes.

Now he knew he had something. Patients in Europe who had neurological symptoms following arthritic disease showed similar findings in their cerebrospinal fluid.

Those lymphocytes might be the marker for the presence of the Borrelia spirochete. Pachner ordered an intravenous line started on the boy and 20 million units of penicillin to be infused daily for fourteen days.

There was no reason to expect sudden response or improvement. If Pachner was right, if the boy's current depression and compulsive behavior were attributable to a brain infection with the spirochete Borrelia, then the initial infection dated back two years, to his first episode of arthritis. A long standing, deep-seated infection like that could not be expected to be resolved overnight.

But the response was dramatic. Within days of the initiation of therapy, Pachner recalls, "his behavior changed."

The parents were speechless . Even now, Pachner finds it difficult to describe the sensation of watching those first changes in the boy.

"It was like-" Pachner searches for a word, shakes his head, then finally says, "a fairy tale. That's all you can say ."

The boy was discharged. Pachner watched him leave with his parents. Two weeks later, the boy arrived with his parents at Pachner's clinic. He had gained weight, but more important, he was talking again, was more outgoing, and had gone back to school.

Within months the boy was back playing soccer and he was doing his homework. The transformation, or the reclamation, was complete. He was back to normal.

In the process, the understanding of the disease that had been called Lyme arthritis had expanded. The disease was no longer limited to the joints. It would henceforth be called Lyme disease, a disease of many organs, including the brain. IT WAS THE NEW GEAT IMITATOR.

Pachner has reported this new disease in many guises:

A 21-year-old man with a history of violent outbursts, confusion, and wild laughing was thought to have a herpes-virus infection of his brain; treated for Lyme disease, he returned to normal.

A 55-year-old woman who had gone to her doctor with a facial droop was cured after a diagnosis of Lyme disease led to early treatment with intravenous penicillin.

A 37-year-old man with fatigue, a sore throat, joint and muscle pains, and facial-muscle paralysis who was thought to have multiple sclerosis was found to have Lyme disease, and all symptoms resolved.

A 61-year-old man with double vision who was thought to have a brain tumor was treated for Lyme disease with only partial improvement, probably because his disease was too advanced to be cured.

And a 6-year-old girl suffering from headaches, knee pain, and tingling in her toes — and later from vertigo and staggering — was apparently [!!!] cured after treatment for Lyme disease followed positive studies of her blood and cerebrospinal fluid.

Pachner thinks about the bicycle boy and says he was just one of many cases. His eyes widen: "There are so many ways it can present. And there are so many ways it presents that look like bad diseases, that when you identify it and your reverse it – YOU FEEL LIKE GOD!" [a dangerous feeling] ...

[Dr. Pachner] speaks of the subtle differences among the various strains of the spirochete that may cause subtle differences in the damage, the signs, and symptoms of the disease. In his laboratory, he is getting to know the spirochete, or the "bug," as he calls it. He is fascinated by the mysteries:

Deer, for example, do not get sick, although they harbor large numbers of Borrelia organisms. Why? "Host defenses," Pachner says. [How do we know that the deer do not have long-term problems, if they live long enough?] ...

He seems driven by the will to know. He was working on his studies of the Lyme disease patients while he was living the impoverished life of a neurology resident, moonlighting like mad ...

Pachner left Yale for Georgetown in 1987, following Johnathan Pincus, the Yale professor of neurology who had been appointed chairman of the neurology department at Georgetown. Pinicus, author of the classic textbook Behavioral Neurology, was able to attract Pachner offering lab space and freedom to pursue his research interests.

Pachner shows me around his laboratory, of which he is proud. "I remember how scarce lab space was at Yale, how people doubled up and scraped by."

The lab Pachner has at Georgetown would have been considered a land of milk and honey at Yale. Several technicians work for him, and they are busy with lab chores. He has set up an assay for the Lyme antibodies, and a technician shows him some "runs."

The blood samples are sent in from local physicians, and some test positive: There is Lyme disease in the Washington area.

Although Lyme disease is known to occur in may countries, particularly in Europe, and in 45 states in this county, the Mid-Atlantic and New England states have an especially high infestation rate.

The tick that carries the disease, Ixodes dammini, [Ixodes scapularis] clings to deer, field mice, and even dogs. Because the ticks are so small, their human victims are often unaware of having played host to this blood sucker, which may cling for four to six days to an unsuspecting body.

In endemic areas such as certain parts of New England and Washington, any patient who walks into the doctor's office with one side of his face drooping in the classic manner of Bell's palsy should be

suspected of Lyme disease. And Bell's palsy is only one common neurologic complication.

Since Pachner's studies called attention to the many sites that may be inhabited by the spirochete, attention has also been focused on heart lesions, which vary from direct attack on the heart-muscle wall – myocarditis – to an attack penetrating every layer of the heart from the inner lining through the heart walls to its coverings -pancarditis.

Patients with Lyme disease can show up at the doctor's office with anything from severe chronic fatigue to arm pain to a variety of palsies to arthritis and skin rashes. Erroneous diagnoses of dementia, multiple sclerosis, psychiatric disease, and arthritis are common, so closely can the great imitator mimic the symptoms of other illnesses.

The diagnosis can be difficult even when the physician suspects Lyme disease. In Pachner's laboratory at Georgetown, blood, spinal fluid, or joint fluid from patients with Lyme disease often fails to yield positive cultures for the spirochete, which is difficult to keep alive outside the body.

While Pachner's laboratory has the highest-quality technicians and antiserums, only about half the patients are positive for the antibody to the B. burgdorferi spirochete early in the course of the disease. And, if the patient happens to be treated with an antibiotic before the diagnosis is made, the antibody test may turn negative while living spirochetes are still reproducing inside the body.

Making matters worse, antibody tests for Lyme disease may be falsely positive in patients who have no Lyme spirochetes but who have instead syphilis or other disease. Special antibody tests have to be done to be sure the doctor is not dealing with a "false positive," in which the test is positive but the patient has no Lyme disease.

Questions have been raised about the wisdom of any pregnant woman in an endemic area such as Washington venturing into wooded areas during tick season. Late spring and early summer are the peak times for the bites that leave the hallmark skin rash, but patients can be infected on any warm day of any month.

There is still no clear evidence about how much risk Lyme disease poses to a developing fetus, but in the absence of hard data, may physicians point to the concept that Andrew Pachner's studies implied: This spirochete behaves in many ways like syphilis, infiltrating along blood vessels. With syphilis as a model, few physicians feel comfortable about the risks for mother and child infected with Lyme disease.

With its many parks running through the heart of the city, with the C&O-Canal running into the heart of Georgetown, Washington is an area in which the country laps up to the front door of suburban and urban dwellers. Deer are common along the canals far into town as Glen Echo and Brookmont on the Maryland-District line and, in Virginia, along the George Washinton Parkway almost to Rosslyn.

Over the coming years, as Washington physicians become more aware of its many guises, more and more cases of Bell's palsy, dementia, fatigue, and arthritis will prove to be Lyme disease.

And there may even be a few boys who have withdrawn from friends and families-boys who are languishing on psychiatric wards-whose blood or spinal fluid will wind up in Andrew Pachner's lab, registering positive. [What about those who "register" negative on the ELISA or Western Blot?]"

Back to top

Are Slow Acting Bacteria and Viruses Simply Wearing Us Down?

DISCOVER Vol. 21 No. 11 (11-00), BOOK REVIEW by Annie Murphy Paul

Plague Time: How Stealth Infections Cause Cancer, Heart Disease, and Other Deadly Ailments

By Paul W. Ewald, The Free Press, $25.

Reviewed by Annie Murphy Paul

" "Humans have never escaped plagues of infectious disease," declares Amherst College biologist Paul Ewald. Despite antibiotics, exterminators, and indoor plumbing, our era is no exception: AIDS, West Nile encephalitis, tuberculosis, and other illnesses regularly threaten.

They may be just the tip of the infection iceberg, however. With an argument certain to stir controversy, Ewald asserts that germs are the culprits for almost every serious ailment plaguing humans today, including cancer, heart disease, diabetes, Alzheimer's, schizophrenia, and arthritis.

We may believe that our bodies are failing because of faulty genes or risky lifestyles or "just falling apart from the wear and tear of life," Ewald says, but really they're suffering the ravages wrought by slow-acting viruses, bacteria, and other pathogens.

A cautious and conventional medical establishment has always been slow to recognize the role of infection, Ewald says, citing the histories of syphilis and, most recently, peptic ulcers.

Moreover, scientists have been laggard in appreciating microbes' ability to evolve and evade our attempts to eradicate them. It's the protean nature of the human immunodeficiency virus, for example, that has made a cure for AIDS so elusive.

To redress these failings, Ewald urges researchers to relax the standards of proof when it comes to identifying a causative agent, accepting "a compelling body of evidence" in place of a definitive demonstration, and granting greater weight to anecdotal evidence.

As for combating pathogens, he advocates finding new ways to piggyback on the powers of the human immune system.

He also proposes some broad policy measures. Strict standards of hygiene, relaxed after the introduction of antibiotics, should be reinstated at hospitals.

Employees who are sick should be urged to stay at home.

In developing countries, where diseases like cholera and malaria still thrive, the emphasis should be on ensuring clean water supplies, adequate waste disposal, and mosquito-proof housing, instead of sophisticated medicine ... ”

Back to top

Is a Bacteria in Milk Causing Some Forms of Crohn’s Disease?

FEATURE ARTICLE from THE CLEVELAND FREE TIMES, 6-17-99, BY LISA CHAMBERLAIN

”THE CROHN’S CONNECTION

A scientific debate rages over an unproven theory linking a bacterium in milk to Crohn’s disease — a debilitating intestinal disorder affecting at least four million people worldwide.

Dr. Rodrick Chiodini went on a treasure hunt. Like most people who embark on an improbable journey, the hunt took over his life, changing it irrevocably. Unlike most treasure hunters, he found what he was looking for.

While working on his Ph.D. in microbiology at the University of Connecticut, Chiodini developed an expertise on a bacterium, Mycobacterium paratuberculosis (Mp), that causes a debilitating intestinal disorder in cattle. The disease in cows, identified more than a century ago by Heinrich Johne, is characterized by diarrhea, excessive weight loss, reduced milk production and ultimately death.

Named after its identifier, Johne’s disease (pronounced YO-neez) in cattle is similar to Crohn’s disease in humans (pronounced kronz). This chronic inflammatory disease of the gastrointestinal tract also results in severe diarrhea, excessive weight loss and — for humans, who live a lot longer than cows — debilitating abdominal pain, rectal bleeding, bowel obstruction, fistulas and abscesses. Chronic Crohn’s will likely lead to surgery for removal of inflamed intestine, as well as a lifetime of harsh drug therapy that often doesn’t work.

The possibility of a connection between Johne’s disease in cattle and a similar gastrointestinal condition in humans had been suggested as early as 1913, but for 70 years, every attempt to locate the bug in human Crohn’s patients failed. Despite the negative results, the similarities between Johne’s in cattle and Crohn’s in humans proved too compelling to ignore.

So in 1981, Chiodini went looking for the elusive bacterium, successfully isolating Mp, the cause of Johne’s disease in cattle. in the intestine of six people suffering from Crohn’s disease. Chiodini’s discovery — seemingly the microbiologist’s equivalent of unearthing a buried treasure — turned out to be a Pandora’s box. For the first time, Chiodini’s research implicated contaminated milk as a possible cause of a debilitating, sometimes fatal, gastrointestinal disease.

Back to top

Is a Mycobacterium Causing Other Forms of Crohn's Disease?

Jul 24, 2000 (Reuters Health) – Researchers in California have discovered a bacterial sequence that is found often in active Crohn's disease lesions but rarely in uninvolved tissues in Crohn's disease patients.

The discovery, if confirmed, could lead to a better understanding of the pathogenesis of Crohn's disease and the identification of new therapeutic targets for this disorder, according to Dr. Jonathan Braun, of the University of California, Los Angeles, and colleagues.

Using representational difference analysis techniques, the investigators isolated two novel microbial sequences, named I1 and I2, from patients with Crohn's disease. The I2 sequence appeared to be selectively expressed in histologically involved colonic mucosa, but not in mucosa cells from controls, they report in the July, 2000, issue of Gastroenterology …

The authors explain that "it is possible that the I2 sequence is derived from a novel bacteria, or from well-known enteric bacteria not yet characterized for this gene segment." Alternatively, there is evidence that the sequence may be derived from a Mycobacterium species, they add.

The new findings merely link the novel bacterial sequence with Crohn's disease; the data are not sufficient to determine causality. However, if further studies confirm a causal link between the I2-expressing bacterium and Crohn's disease pathogenesis, the sequence may become a new target for anti-Crohn's disease therapies, Dr. Braun and colleagues conclude …

Gastroenterol 2000;119:23-31,254-257.

Back to top

Can a Virus Make You Fat?

BBC NEWS, 7-28-00, By BBC News Online's Matt McGrath

"Could a virus make you fat?

A common cold-like virus may increase body fat

Researchers in the United States say some people's weight problems might be caused by a common cold virus.

Experiments with animals suggest that AD-36, a virus which causes coughing, sneezing and cold-like symptoms, interferes with the normal process of absorbing food energy and converts far more of it into fat.

There have been five other viruses to date that have shown an impact on obesity but this is first human one

Dr. Nikhil Dhurandhar and colleagues from the University of Wisconsin injected chickens and mice with AD-36.

Despite being fed the same amounts of food as a control group, the test subjects gained small amounts of extra weight.

But the scientists found that the animals had put on large amounts of body fat, almost 2.5 times more than normal.

This work has been published in the INTERNATIONAL JOURNAL OF OBESITY.

Dr. Dhurandhar told BBC News Online that in unpublished research he had also used the same virus to cause obesity in Marmoset monkeys.

And he said that while the research was not definitive proof, it indicated that this virus might have a similar impact on humans.

“There have been five other viruses to date that have shown an impact on obesity but this is the first human one. It’s suggestive of a direct effect in people,” he said.

Screening blood samples for signs of the virus, Dr Dhurandhar said that about 30% of obese people had contracted AD-36 compared with 5% for lean people.

“We’re not saying that all obesity is due to this virus – but there might be a percentage of people in whom this virus might be contributing to their obesity,” he said.

Other scientists called for more definitive research. Professor Nick Finer, director of the Centre for Obesity Research, Luton and Dunstable Hospital in Luton, UK, said the work was very interesting ...

Dr Dhurandhar said ethical considerations meant that virus experiments on people were not possible, but he would keep on collecting indirect evidence.

“We want to look at people who have the virus, and look at them again in two to five years,” he said.

Vaccines against this virus might one day be possible said Dr Dhurandhar, but he believed that the biggest impact of his work would be to increase the acceptance of obesity as a disease.”

Back to top

Is It Really Possible All of These Degenerative Diseases Are Caused, Triggered or Exacerbated by Infectious Microorganisms?

Annals of Internal Medicine. 15 November 1996. 125:844-851.

Bennett Lorber, MD, DSc

The complex interactions between microorganisms and human hosts include the well-known, traditional infectious diseases and the symbiotic relation we have with our normal flora. The media

have brought to the public's attention many newly described infectious diseases, such as Ebola virus hemorrhagic fever, that were not part of common medical parlance a decade ago.

While flooding us with interesting and often dramatic reports of so-called emerging infectious diseases, the media have largely ignored a more fundamental change in our appreciation of human-microorganism interactions: the discovery that transmissible agents may play important roles in diseases not suspected of being infectious in origin.

A well-known example is ulcer disease; other examples include neurodegenerative disease, inflammatory disease, and cancer. These fascinating instances of host-pathogen interaction open new prospects for the prevention of disease through immunization ...

The interactions between microorganisms and humans are complex and wondrous. On the one hand, they may result in human illness or death, as in bacterial meningitis or viral hemorrhagic fever. On the other hand, they may be truly symbiotic, as in the case of the flora that coat our mucous membranes to produce such nutrients as vitamin K and suppress colonization by pathogens.

Extraordinary changes have occurred in our appreciation of human-microorganism interactions (1). Not the least of these changes is the recognition of new infectious diseases. With just a few moments' reflection, one can call to mind an impressive list of diseases and pathogens that were not part of common medical parlance a decade ago.

Since smallpox was consigned to history almost 20 years ago, the weight of new afflictions has tipped the balance. National news magazines have eagerly and excitedly reported on many of these so-called emerging diseases, and these diseases have been the subject of bestselling books, movies, and television docudramas. Last year saw the advent of a new medical journal devoted to emerging infectious diseases (2).

In almost every instance, these newly recognized diseases were thought to be of infectious origin before they were proven to be so, because the clinical picture suggested that likelihood. Almost without exception, these newly described entities are acute self-limited illnesses that have fever as a hallmark, and many mimic known infectious diseases. For example, human ehrlichiosis is a febrile illness that follows a tick bite and is similar to Rocky Mountain spotted fever (3).

Although we have been flooded with interesting and sometimes dramatic reports of emerging infectious diseases and antimicrobial-resistant bacteria, the media have largely ignored a quieter revolution that has been taking place in our understanding of human-microorganism interactions: the discovery that transmissible agents are responsible for diseases that were never suspected of being infectious in origin.

Examples include ulcers, neurodegenerative diseases, vasculitides, and cancer ... In some instances, the pathogen is truly causal, a sine qua non for disease development. In other cases, it may trigger an immune reaction that leads to illness or function as a risk factor for disease.

How Do We Know a Disease Is Infectious?

The first to prove that a disease was caused by a particular organism was Robert Koch (46), who showed the bacterial origin of anthrax in 1876. Koch found that mice could be infected with matter obtained from diseased domestic animals, that disease could be transmitted from mouse to mouse through a series of inoculations, and that features of the disease were seen at each transfer.

He cultivated bacteria by putting infected pieces of spleen into drops of sterile serum, and he observed and photographed bacteria in the culture medium through his microscope. After serially transferring cultures eight times, he inoculated a healthy animal.

This inoculation produced the characteristic disease in the animal, from which Koch reisolated the organism. These experiments fulfilled criteria proposed 36 years earlier by Henle (46) as necessary to establish a causal relation between a specific agent and a specific disease. These criteria are now known as the Koch postulates ...

Because routine methods sometimes fail to grow a pathogen, the Koch postulates may not be fulfilled; an infectious origin is then presumed because a given disease responds to antimicrobial agents. This leap of logic is fraught with hazards.

For example, many will recall that antibiotic-related colitis due to Clostridium difficile toxin (47) was originally thought to be caused by Staphylococcus aureus because 1) uncontrolled studies during the peak era of staphylococcal nosocomial infections found S. aureus in stool specimens and 2) patients responded to oral vancomycin, a nonabsorbable, antistaphylococcal agent (48).

Modern technology has made it possible to establish the association of a specific infectious agent with a disease without fulfilling the Koch postulates. Whipple disease is a case in point. Diagnosis of this disorder is usually established by small-bowel biopsy tissue that shows pathognomonic periodic acid-Schiff staining matter in macrophages. Electron microscopy has clearly shown bacteria in these macrophages, and the disease responds to antimicrobial agents. However, attempts to grow the organism in the laboratory have failed.

The answer to this problem came from molecular biology. In 1992, Relman and associates (49), building on the work of Wilson and coworkers (50, 51) from a year earlier, used a POLYMERASE CHAIN REACTION (PCR)-based detection method to amplify 16S ribosomal RNA sequences from tissue. They identified the Whipple bacillus as a bacterium that was related to actinomycetes but was genetically distinct enough to be given its own genus designation. Relman and associates proposed the name Tropheryma whippelii for this organism.

This PCR technique has subsequently been used as a diagnostic tool (52) to identify the Whipple bacillus in such extraintestinal sites as the eye (53) and the mononuclear cells of the peripheral blood (54). It has also been used to establish a novel Bartonella species as the etiologic agent of cat scratch disease, bacillary angiomatosis, and peliosis hepatis (55).

Peptic Ulcer

The best known example of an infection that was not suspected of being one is that of ulcers. In 1983, at the Royal Perth Hospital in Australia, Marshall and Warren successfully cultured a spiral bacterium from human gastric mucosa and showed an association between the presence of this organism and gastric inflammation (5).

Their discovery forced revision of the view that bacteria could not survive in stomach acid and revolutionized our understanding of upper gastrointestinal illnesses. The bacterium, Helicobacter pylori, lives on gastric epithelium and attaches to specific receptors. It causes one of the most common of all bacterial infections (4, 5). About 40% of persons in developed countries are infected with H. pylori by adulthood and once acquired, infection persists for life if untreated.

The evidence that H. pylori is an important cause of gastritis and peptic ulcer disease and is a risk factor for gastric carcinoma is now overwhelming (4, 5). Helicobacter pylori also appears to play a role in the development of the so-called maltomas, which are low-grade B-cell lymphomas of lymphoid tissue associated with gastric mucosa (5, 42). It is detected in more than 90% of gastric maltomas, and eradication of it results in regression of these tumors (42).

Our understanding of ulcer pathogenesis is incomplete. The role of acid must be elucidated, as must the reason why some persons infected with H. pylori develop ulcers and some do not. Nevertheless, discovery of H. pylori has revolutionized ulcer treatment through the realization that antimicrobial agents can cure ulcers related to the organism and, by eradicating it, prevent ulcer recurrence.

Dementia and Paralysis

Scrapie, a disease of sheep and goats named for the unique tendency of afflicted animals to scrape off their coats by rubbing against inanimate objects, is one of a group of diseases called “spongiform encephalopathies.” This term derives from the histologic findings in the brain that are characteristic of these conditions. All of these diseases are fatal.

They were originally thought to be neurodegenerative, and some were thought to be hereditary, but they are now known to be caused by transmissible agents (7, 9). The other animal diseases in this group include transmissible mink encephalopathy, chronic wasting disease of mule deer and elk, feline spongiform encephalopathy, and bovine spongiform encephalopathy (also known as mad cow disease).

Mad cow disease was first identified in 1986 when cows in Great Britain began to acquire an illness that was characterized by apprehension and lack of coordination and eventually led to death (9). The source of this new epidemic was traced to a food supplement that included bone meal and neural tissue taken from dead sheep. More than 130 000 cattle have been stricken to date. There is great concern that humans may have fallen ill from having eaten tainted beef (56).

The first human spongiform encephalopathy to be described was kuru, which has been seen only

among the Fore highlanders of Papua, New Guinea. Many will remember that kuru was first described in 1957 by Gajdusek (57), who noted that afflicted Fore tribesmen developed a strange illness marked first by ataxia, then by dementia, and eventually by death. Evidence suggested that kuru was acquired through ritual cannibalism; the Fore tribe honored their dead by eating their brains. With the cessation of cannibalism, kuru has disappeared (57).

Four types of human spongiform encephalopathy are currently recognized (9): Creutzfeldt-Jakob disease, kuru, Gerstmann-Sträussler disease, and fatal familial insomnia. The most important of these, Creutzfeldt-Jakob disease, occurs worldwide, typically as a sporadic disease with onset in the seventh decade of life; 10% to 15% of cases appear to be inherited.

A few cases are iatrogenic, and the disease has been transmitted through dura mater grafts, corneal transplants, neurosurgical instruments, and the injection of cadaveric-derived human growth hormone. Experimental studies have shown that scrapie, Creutzfeldt-Jakob disease, and kuru can be transmitted by injecting extracts of diseased brains into the brains of healthy animals. The incubation period is measured in years and even decades.

In the early 1970s, Prusiner (7, 9) began a series of laboratory investigations showing that the agents of spongiform encephalopathies are tiny bits of protein that contain no nucleic acids. This observation was initially met with much skepticism because it was contrary to the main dogma of molecular biology: that all reproducing and transmissible agents require genetic material made of nucleic acids. Prusiner coined the term “prion” for these “proteinaceous infectious particles” that are responsible for transmissible and inherited disorders as well as sporadic disease.

This discovery marked a revolutionary change. It was newly recognized that neurodegenerative diseases with no evidence of inflammation could be caused by transmissible agents, and a new construct for thinking about the molecular requirements for transmissibility was developed.

The Guillain-Barré syndrome, the most common cause of acute neuromuscular paralysis, was associated with antecedent gastrointestinal and respiratory illnesses through case-control studies in the 1960s and 1970s. Clinically associated illnesses have included mononucleosis, chickenpox, mumps, hepatitis, and mycoplasmal infection; many viruses have been implicated through serologic studies.

An association between the Guillain-Barré syndrome and infection with the bacterium Campylobacter jejuni was recently described, and more than 20 reports and case series have documented this association in the past 10 years (10, 11).

At the onset of symptoms of the Guillain-Barré syndrome, 20% to 30% of patients have documented positive results on stool cultures for C. jejuni (10). Serologic evidence of C. jejuni infection is often documented in many patients with negative cultures, and antiganglioside antibodies have been associated with campylobacteriosis even in the absence of the Guillain-Barré syndrome (11). The Guillain-Barré syndrome subsequent to C. jejuni infection is associated with axonal degeneration,

slow recovery, and severe residual disability (58).

Another acute neuroparalytic problem, acute facial nerve paralysis, has been associated with infectious diseases, including syphilis and the Ramsay Hunt syndrome due to varicella-zoster virus. We recently learned that the spirochete Borrelia burgdorferi can produce acute facial paralysis (Bell palsy) as a common neurologic manifestation of early Lyme disease (12).

However, the cause of most cases of Bell palsy remained obscure until early this year (13), when a strong association with herpes simplex virus type 1 (HSV-1) was made. With PCR analysis, HSV-1 genomic sequences were found in endoneurial fluid from the facial nerve or from muscle innervated by the facial nerve in patients with Bell palsy; the virus was not present in controls.

Acute Renal Failure

That infectious agents could be among the many causes of acute renal failure was brought home to U.S. physicians when some 3000 cases of a febrile illness with renal failure occurred among United Nations troops involved in the Korean Conflict. Hemorrhagic fever with the renal syndrome, as it is now called, is caused by infection with one of several hantaviruses (15). These viruses produce asymptomatic, lifelong infections in rodents and are transmitted to humans through inhaled rodent excreta.

Most cases of the hemolytic uremic syndrome occur in children younger than 10 years of age; in this group, it is the most common cause of acute renal failure (17). In 1982, two outbreaks of acute bloody diarrhea were linked to ingestion of poorly cooked hamburgers by patrons of fast-food restaurants.

These outbreaks were ultimately shown to have been caused by Escherichia coli O157:H7, an uncommon organism that produces a Shiga-like verotoxin (16-18). Many cases of bloody diarrhea caused by this E. coli serotype have subsequently been reported, and most are linked to ingestion of poorly cooked ground beef. Most interesting is that about 10% of infected persons younger than 10 years of age develop the hemolytic uremic syndrome, and it appears that as many as 75% of cases of the syndrome in the United States are complications of intestinal infection with E. coli O157:H7 (16-18).

Arthritis

Physicians have long known that so-called reactive arthritis (spondyloarthropathy) follows intestinal infection with Salmonella typhimurium and Yersinia enterocolitica or urethral infection with Chlamydia trachomatis (28). Although live bacteria have not been convincingly shown in inflamed joints, recent studies have reported finding bacterial antigens and nucleic acid in intrasynovial cells by immunofluorescence and molecular hybridization (28).

The cause of rheumatoid arthritis remains unknown. In the past, a causal role for several agents, including mycoplasmas, was suggested, but convincing data are lacking. In September 1995, Tilley and colleagues (29) reported a 48-week, double-blind, placebo-controlled trial that showed a

beneficial effect for the antibiotic minocycline in rheumatoid arthritis.

This intriguing study harks back to an earlier observation. In 1947, Brown treated patients who had rheumatoid arthritis with crystalline chlortetracycline hydrochloride and, in 1949, reported favorable results at the 7th International Congress on Rheumatic Diseases (59).

Unfortunately for Brown and the progress of tetracyclines as antiarthritic agents, the beneficial effects of cortisone in the treatment of arthritis were introduced at the same meeting. The effect of tetracycline paled beside that of steroids, and the salutary effects of antibiotics on rheumatoid arthritis were largely ignored for almost 50 years.

Vasculitis

It has been known for some time that patients occasionally develop polyarteritis nodosa a few months after having had hepatitis B infection. About 15% to 25% of patients with polyarteritis nodosa (even those without a history of hepatitis B infection) can be shown to have immune complexes containing hepatitis B antigens in the circulation and in involved tissues (19).

In 1985, a group from the Mayo Clinic (23) reported that trimethoprim-sulfamethoxazole had a beneficial effect in patients with Wegener granulomatosis. Reports of efficacy followed during the next few years from at least three groups.

For example, a 1988 report (24) stated that 10 patients who had been treated with trimethoprim-sulfamethoxazole alone starting in 1984 had had an excellent response and that 9 of the 10 were in complete remission. In contrast to these glowing reports, workers from the National Institutes of Health (60) claimed that only 1 of 9 patients treated with trimethoprim-sulfamethoxazole in a so-called ongoing, prospective, open study had prolonged improvement. Therefore, the jury is still out on the effect of trimethoprim-sulfamethoxazole for Wegener granulomatosis.

In 1994, a group from the Netherlands (61) studied a cohort of 71 patients with Wegener granulomatosis and found that those who were chronic nasal carriers of S. aureus were more likely than those who were not nasal carriers to have relapses. Relapses were not related to diagnosed infections, and investigators are now studying whether eradication of carriage affects relapse rates.

Mixed cryoglobulinemia has recently been linked to a viral origin. When serologic testing for hepatitis C virus (HCV) and the ability to test for the HCV genome using PCR technology became available, the role of HCV infection in mixed cryoglobulinemia became clear.

The first report (21) came from an Italian group in 1991. This group found that 91% of 45 patients with mixed cryoglobulinemia had antibodies to HCV. Subsequent studies in Italy (20), France (22), and the United States (21) showed evidence of HCV infection in a large percentage of patients.

Other investigators showed that the HCV RNA levels of some antibody-negative patients were higher in cryoprecipitates than in plasma (62), suggesting that some immune complexes contained HCV antigens and that HCV plays a part in the pathogenesis of cryoglobulinemia. Antiviral treatment may

have a role in the treatment of mixed cryoglobulinemia. A 1992 report (62) showed that 2 patients had a dramatic clinical response to interferon-a, characterized of the disappearance of palpable purpura and of renal dysfunction.

The cause of giant cell arteritis remains unknown. A study published in 1995 (25) reported the incidence of giant cell arteritis in Olmstead County, Minnesota, over a 40-year period. This study documented that the incidence of giant cell arteritis had a cyclic pattern that peaked about every 7 years and an overall frequency that increased steadily over time. This was a totally new observation. Although the observed periodicity does not prove an infectious origin, it is compatible with one. The data need to be confirmed in other populations.

Inflammatory Bowel Disease

Because its clinical and histopathologic features are similar to those of ileocecal tuberculosis, investigators have long suspected that Crohn disease has an infectious origin. Sporadic reports of case clustering of Crohn disease among family members and close friends support this idea.

Mycobacterium paratuberculosis causes Johne disease, a chronic, granulomatous enterocolitis of ruminants that does not respond to antimycobacterial treatment. In 1984, Chiodini (6) recovered M. paratuberculosis from the tissues of three patients with Crohn disease; he later cultured identical isolates from Crohn disease tissues at five centers on three continents.

Mycobacterium paratuberculosis has been isolated from fewer than 15% of patients with Crohn disease, but it is rarely found in tissue from patients with ulcerative colitis or from controls. Interest in M. paratuberculosis as an etiologic agent has recently been renewed because the organism has been detected more frequently using PCR.

In 1992, Sanderson (6) found M. paratuberculosis DNA in 65% of Crohn disease samples, 4% of ulcerative colitis samples, and 13% of control samples. An even higher percentage of samples positive for M. paratuberculosis has been found in children with Crohn disease.

It was recently suggested that Crohn disease is caused by a persistent infection of vascular endothelial cells due to a viral-induced, focal granulomatous vasculitis. Wakefield (6) visualized paramyxovirus-like structures in nine of nine patients with Crohn disease. Measles antigen and messenger RNA were localized to granulomas and endothelial cells by immunohistochemistry and in situ hybridization.

Diabetes

Several observations support a link between infection and insulin-dependent diabetes mellitus (26). Enteroviruses have been found in the pancreata of patients with recent-onset insulin-dependent diabetes. After infection with certain enterovirus strains, susceptible mice develop insulin-dependent diabetes. Insulin-dependent diabetes also has a seasonal incidence similar to that of enteroviral infections: Enteroviral infections peak in the late summer and early autumn, and insulin-dependent diabetes peaks in autumn and early winter.

Several seroepidemiologic studies have linked insulin-dependent diabetes with antibodies to enteroviruses. For example, a 1995 report (27) of a case-control study supported an association between IgM antibodies to enteroviruses and new onset of insulin-dependent diabetes in older children.

Coronary Artery Disease

Implication of an infectious origin of atherosclerosis would open the potential for preventing heart attacks with a vaccine. Several reports have shown a relation between cytomegalovirus infection in transplant recipients and early coronary artery vasculopathy (30, 31). Cytomegalovirus antigens have been shown in atheromatous plaques in these patients.

Another infectious agent has been implicated in the pathogenesis of coronary artery disease in persons with normal immune function. In 1988, Saikku and colleagues (32) showed that men with acute myocardial infarctions were more likely than age-matched controls to have elevated serum antibody levels to Chlamydia pneumoniae.

In a follow-up study that was part of a large prospective study of risk reduction for coronary artery disease involving more than 4000 patients (32), these authors showed that elevated serum levels of antibodies to C. pneumoniae were associated with the development of coronary artery disease, as were circulating immune complexes containing chlamydial lipopolysaccharides. The study addressed the concern that myocardial infarction may have activated a latent chlamydial infection. These data were supported by a study (33) from the United States.

In 1993 (34), PCR and immunocytochemistry were used to show C. pneumoniae in coronary artery atheromas in approximately one half of study patients. Electron microscopy showed that typical pear-shaped C. pneumoniae elementary bodies were present in 6 of 21 atheromatous plaques, providing further evidence that C. pneumoniae may be involved in atherosclerosis and coronary artery disease.

Cancer

The most recent type of cancer to be linked to an infectious agent (Table 1) is Kaposi sarcoma, the most common neoplasm in patients with the acquired immunodeficiency syndrome (AIDS). An infectious origin for Kaposi sarcoma has been suggested by epidemiologic studies showing that patients with AIDS who are bisexual or homosexual are 20 times more likely to develop Kaposi sarcoma than are heterosexual patients with AIDS (43).

Kaposi sarcoma occurs more frequently than would be expected in homosexual men who do not have human immunodeficiency virus (HIV) infection. In these persons, it behaves in the same manner as classic Kaposi sarcoma (43), supporting the theory that Kaposi sarcoma may be caused by sexual transmission of an infectious agent.

In 1994, Chang and colleagues (63) used a technique that had been described a year earlier (64) that enables investigators to find and amplify unique DNA sequences in tissue. This technique can find

foreign (nonhuman) DNA and differentially amplify it, leaving the host DNA behind.

This application is a brilliant use of PCR technology. Chang and colleagues found unique DNA sequences in Kaposi sarcoma lesions in 25 of 25 patients with AIDS. These sequences were shown to be nonhuman and similar, but not identical, to genes of the Epstein-Barr virus and herpesvirus saimiri (whose natural host is the squirrel monkey). These DNA sequences identified a totally new herpesvirus-the eighth to be discovered.

Was this new virus an etiologic agent or did it just preferentially colonize Kaposi sarcoma lesions in immunosuppressed patients? To answer this question, the novel herpesvirus DNA was looked for (43) in Kaposi sarcoma lesions from patients with AIDS, patients with classic Kaposi sarcoma, and patients with Kaposi sarcoma who were homosexual but HIV negative.

Also studied were patient tissues not involved with Kaposi sarcoma lesions and control tissues from healthy persons. A strong association was seen between Kaposi sarcoma lesions and the newly described herpesvirus. Viral sequences from the three types of Kaposi sarcoma were more than 98% identical (43), suggesting that all were caused by the same agent. It appears that a new human herpesvirus is the cause of Kaposi sarcoma.

Other investigators (44) looked for this new herpesvirus DNA in lymphomas from patients with and without AIDS. Of 193 patients with lymphoma who were studied, the so-called Kaposi sarcoma-associated herpesvirus was found in only 8 patients, all of whom had a body cavity-based lymphoma. These 8 samples also contained the Epstein-Barr virus genome.

The Kaposi sarcoma-associated herpesvirus sequences were 40 to 80 times more abundant in lymphoma cells than in Kaposi sarcoma cells. This same virus has since been shown in patients with Castleman disease (45) and in the lymphomatous pleural effusion (65) of an HIV-negative elderly man without Epstein-Barr virus infection.

Conclusions

A quiet scientific revolution has been taking place, showing that infectious agents can be the causes of, precipitating factors for, or risk factors for various diseases that were not previously thought to be caused by transmissible agents, thereby expanding our understanding of human-microorganism interactions.

Are all diseases infectious? Of course not. But who would have guessed 20 years ago that ulcers could be eradicated with antibiotics, that spongiform encephalopathies were caused by transmissible agents composed only of tiny bits of protein, or that an unknown herpesvirus was the cause of Kaposi sarcoma?

We have every reason to believe that science will show still more infectious causes for degenerative, inflammatory, and even hereditary diseases. Although this prospect is sobering, the opportunity for disease prevention through immunization is exciting to contemplate. Developments in this area are

eagerly anticipated ...

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Back to top

Why Are Germs from Hospitals Now the Fourth Leading Cause of Death Among Americans?

FEATURE ARTICLE from The Chicago Tribune, 7-21-01, By Michael J. Berens, Tribune staff reporter

Infection epidemic carves deadly path

CHICAGO — A hidden epidemic of life-threatening infections is contaminating America's hospitals, needlessly killing tens of thousands of patients each year.

These infections often are characterized by the health-care industry as random and inevitable byproducts of lifesaving care. But a Tribune investigation found that in 2000, nearly three-quarters of the deadly infections–or about 75,000–were preventable, the result of unsanitary facilities, germ-laden instruments, unwashed hands and other lapses ...

Deaths linked to hospital germs represent the fourth leading cause of mortality among Americans, behind heart disease, cancer and strokes, according to the federal Centers for Disease Control and Prevention. These infections kill more people each year than car accidents, fires and drowning combined ...

"The number of people needlessly killed by hospital infections is unbelievable, but the public doesn't know anything about it," said Dr. Barry Farr, a leading infection-control expert and president of the Society for Healthcare Epidemiology of America ...

In a Detroit hospital, as doctors and nurses moved about the pediatric intensive care unit without washing hands, infections killed four babies in the same row of bassinets, according to court records and interviews. But it took three months for administrators to close the nursery for cleaning.

Staphylococcus germs thriving inside a West Palm Beach, Fla., hospital invaded more than 100 cardiac patients, killing 13, according to court records. The survivors underwent painful and debilitating surgery, as rotting bone was cut from their bodies ...

Even a term adopted by the CDC–nosocomial infection–obscures the true source of the germs. Nosocomial, derived from Latin, means hospital-acquired. CDC records show that the term was used to shield hospitals from the “embarrassment” of germ-related deaths and injuries.

To document the rising rate of infection-related deaths, the Tribune analyzed records fragmented among 75 federal and state agencies, as well as internal hospital files, patient databases and court cases around the nation. The result is the first comprehensive analysis of preventable patient deaths linked to infections within 5,810 hospitals nationally.

The Tribune’s analysis, which adopted methods commonly used by epidemiologists, found an estimated 103,000 deaths linked to hospital infections in 2000. The CDC, which bases its numbers on extrapolations from 315 hospitals, estimated there were 90,000 that year …

Government and hospital industry reports analyzed by the Tribune reveal that:

Serious violations of infection-control standards have been found in the vast majority of hospitals nationally. Since 1995, more than 75 percent of all hospitals have been cited for significant cleanliness and sanitation violations.

In thousands of cases observed by federal or state inspectors, surgeons performed operations without washing hands or wearing masks. Investigators discovered fly-infested operating rooms where dust floated in the air during open-heart surgeries in Connecticut. A surgical assistant used his teeth to tear adhesive surgical tape that was placed across an open chest wound during a non-emergency procedure in Florida.

Hospital cleaning and janitorial staffs are overwhelmed and inadequately trained, resulting in unsanitary rooms or wards where germs have grown and multiplied for weeks, sometimes years, on bed rails, telephones, bathroom fixtures–most anywhere …

Since 1969, when U.S. Surgeon General William Stewart confidently told Congress that the nation could “close the book on infectious diseases,” hospital infection rates have quietly pushed higher each year, registering a 36 percent increase in the last 20 years, according to CDC records.

Today, about 2.1 million patients each year, or 6 percent, will contract a hospital-acquired infection among 35 million admissions annually, CDC records show …

Nurses, in particular, say staffing cutbacks have made the most basic requirements of their jobs difficult to fulfill …

The national study of 799 hospitals found that patients were more likely to contract urinary tract infections and hospital-acquired pneumonia if nurse staffing was inadequate. The study projected that the widening nursing shortage could create even more problems, such as higher mortality rates.

“When you have less time to save lives, do you take the 30 seconds to wash your hands?” said registered nurse Trande Phillips, who works in San Francisco.

“When you’re speeding up you have to cut corners. We don’t always wash our hands. I’m not saying it’s right, but you’ve got to deal with reality.”

… doctors at Bridgeport Hospital voted on April 21, 1997, against testing all patients for infection because it was not “cost effective,” according to minutes of a meeting by the hospital’s infection-control committee obtained by the Tribune …

A hidden camera was installed outside Operating Room2, and the tapes revealed that up to half of doctors, primarily surgical residents from Yale University, did not wash their hands before entering the operating room, according to hospital records.

Operating rooms should be secured and sterile during surgeries, but nurses and doctors routinely stepped inside Room2, even while open-heart surgery was under way, to make personal calls on a phone mounted on the wall.

Doctors also are supposed to change from street clothes into clean scrub outfits in a changing room at the hospital, but many doctors wore the scrubs home and back into the hospital the next day–and then directly into the operating room …

In the 1840s, a Hungarian-born physician, Ignaz Philipp Semmelweis, stood in a Vienna auditorium before his medical peers and proffered a controversial theory: Washing hands saved lives.

When treated by doctors with unwashed hands, pregnant women often developed fatal infections following hospital births, but mothers rarely contracted infections if doctors thoroughly scrubbed their hands with soap and water, his groundbreaking study found.

European doctors … [after firing Semmelweis, later adopted] the soap-and-water regimen — the Semmelweis technique. Infection rates plummeted …

U.S. doctors debated the procedure for an additional two decades.

By the end of the century, however, America developed a hospital system second to none, in part through an obsession with cleanliness. Prevention became a life-or-death necessity because almost any infection could kill.

But by the 1950s, the widespread use of penicillin and other antibiotics allowed doctors to overcome once-lethal infections, and over the decades, prevention gradually became less of a priority. New generations of doctors have grown accustomed to responding to symptoms–wait until the patient is sick, prescribe a drug.

Within the average U.S. hospital today, about half of doctors and nurses do not wash hands between patients, a dozen recent health-care studies show.

The direct observations of federal and state inspectors in recent years underscore the carelessness that threatens patient health. In Baltimore, inspection records show, a doctor placed his stethoscope on the chest of a sweaty patient in the grip of pneumonia, then walked to another room and placed the

unwashed, moist device on the chest of a patient. The patient developed pneumonia.

In Loyola University Medical Center in Maywood, a resident physician dropped a surgical glove on a dirty floor, picked it up, put it on his hand and changed the bloody dressing on the open wound of a burn patient ...

Nurses and other health-care workers complain that it's virtually impossible to wash hands between every patient contact, which could number 150 times or more a day in a busy hospital ...

Consequently, most hospitals have begun to use a waterless disinfectant that kills germs and instantly dries on hands. Nurses can squeeze the solution on their hands from wall dispensers and continue to the next patient as their hands are cleaned. Studies show the waterless system kills germs as effectively as soap and water. However, many nurses fail to adopt even this simple measure, hospital inspection reports show ...

The sanitary condition of a hospital also depends on the diligence of its housekeeping staff, but in many facilities those staffs are poorly trained and overburdened.

Since 1995, federal inspectors have cited 31 Chicago hospitals for failure to properly sanitize rooms between patients, mirroring problems found in half of hospitals nationally ..."

Back to top

Why Are Drug-Resistant Germs From Hospitals Now Spreading Into the General Population?

FEATURE ARTICLE from The Chicago Tribune, 7-23-01, By Michael J. Berens, Tribune staff reporter

"Drug-resistant germs adapt, thrive beyond hospital walls

Lapses in infection control and overuse of antibiotics [in hospitals] are spawning drug-resistant germs that are spreading from hospitals into the community at unprecedented rates.

These new super germs — stronger, more elusive and deadlier — have multiplied for decades inside thousands of hospitals and now are hitching rides into outside communities on the clothes and skin of patients, workers and visitors.

Until the last few years, most germs quickly died after exposure to the harsher environmental conditions outside hospitals. But, increasingly, microorganisms survive for days, even months. And they have developed the ability to breed most anywhere.

"It was only a matter of time before hospital germs became strong enough to live in the community," said Dr. Donald Graham, department chief of infectious diseases at the Springfield Clinic and professor at Southern Illinois University School of Medicine. "We're seeing them pop up everywhere."

In Illinois, the Tribune identified 4,712 cases during 2000 in which individuals contracted hospital-

born germs without setting foot in a hospital or other medical center–a 1,000 percent increase in the last decade, an analysis of state patient records and public health reports show ...

The progression of drug-resistant germs from hospital to community has taken decades to occur, spurred by a long-standing practice to rapidly treat patients with antibiotics but not invest in the more time-consuming efforts to locate the sources of germs, federal studies show.

As a result, many hospitals have become reservoirs of microorganisms that continue to adapt to germ-fighting drugs.

The flow of germs outside the hospital also is aided by cost-saving strategies to discharge patients quickly. In the 1970s, the average stay was about seven days for most patients. Today, stays at most hospitals average three days, according to the American Hospital Association.

Most infections are not detectable during the first three days after exposure, so doctors commonly flood patients with antibiotics, even when they're not sure an infection is present. Health-care researchers cite this practice as one of the chief culprits behind the rise in drug resistance.

During longer stays, hospitals had more time to identify infections and provide treatment. Patients were more likely to leave the hospital without a lingering infection. Briefer hospital stays mean that more patients are at home when infections first show symptoms.

Recent federal studies conclude that up to 16 percent of patients' family members carry germs spread by the patient. In most cases, the germs remain inactive but continue to spread to other people or places, creating a chain of migration that is largely untraceable. Tens of thousands of people now are infected each year as the germs find hosts outside the hospital ...

In 1997, two pediatricians at the University of Chicago Hospitals stumbled onto a discovery that cast a national spotlight on hospital germs spreading into communities.

Their curiosity was aroused when they encountered a Chicago boy hospitalized at the U. of C. for a pneumonia that was resistant to methicillin, the most used and effective antibiotic against the infection. The germ is known as methicillin-resistant staphylococcus aureus ...

Drs. Betsy Herold and Robert Daum dug deeper. The boy had not been sick, nor had he recently visited a hospital or any medical center. Likewise, none of the boy's family members had been sick or visited medical centers ...

Worried they were witnessing a new and dangerous trend, the doctors pored over patient medical files dating to 1993. They discovered 35 previously unknown cases where children appeared to contract MRSA (methicillin-resistant staphylococcus aureus) outside the hospital.

Herold and Daum published their findings in the Journal of the American Medical Association in early 1998, the first documented proof in this country that MRSA (methicillin-resistant staphylococcus aureus) had spread into communities.

The Chicago discovery was part of a national groundswell of recognition …

In July 1997, a 7-year-old girl from Minnesota who complained of fever and a pain in her right groin died from MRSA (methicillin-resistant staphylococcus aureus).

In January 1998, a 16-month-old girl from North Dakota arrived at a local hospital in shock, with a temperature of 105 degrees. She died within two hours of admission. MRSA (methicillin-resistant staphylococcus aureus) was found in her lungs.

In January 1999, a 13-year-old girl from Minnesota was taken to an emergency room after complaining of fever and spitting up blood. MRSA (methicillin-resistant staphylococcus aureus) was found in her blood. She died seven days later.

In February 1999, a feverish 12-month-old boy from North Dakota was taken to the emergency room after repeatedly vomiting. MRSA (methicillin-resistant staphylococcus aureus), which was found in the lungs, resulted in pneumonia. The boy died a day later.

These and many other discoveries stoked renewed interest in infectious diseases as researchers delved into the molecular construction and behavior of germs.

Germs that once required moisture now survive on dry fabrics. Germs dependent on a living host can go dormant on inanimate objects for weeks before bursting to life upon contact with human skin. These germs are capable of reproducing in minutes, share their enhanced abilities with other germs upon contact and thrive on surfaces even after smothered with disinfectants …

Staph's ability to develop resistance … Beginning in the 1940s, penicillin was the first line of defense against staph, killing nearly every germ. By 1982, penicillin was effective in less than 10 percent of cases …

As penicillin's effectiveness waned, doctors turned to methicillin, a more powerful antibiotic. In 1974, the replacement drug killed 98 percent of staph germs. By the mid-1990s, it could kill just half of them, and the percentage of staph germs resistant to methicillin is rising.

The problem facing the medical community is that germs, which multiply into millions of one-cell organisms every few minutes, can undergo spontaneous mutations that result in resistance. Every time an antibiotic is used, it presents another opportunity for the germs' genetic material, their DNA, to mutate and be passed on to the next generations of germs.

"We humans can take generations to adapt to stress," said the U. of C.'s Daum. "Bugs can take minutes."

One measure of the hospital industry's decline in controlling germs and infections is found in hospital inspection reports compiled by state public health agencies and the U.S. Department of Health and Human Services.

Mirroring the national trend, nearly half of Illinois' 305 hospitals have been cited for potentially life-

threatening breakdowns of infection-control standards since 1995. Violations range from failure to disinfect rooms, including intensive care units, to unsanitary habits of health-care workers, such as wearing contaminated gloves or clothes or failing to wash hands.

Though carelessness is a big part of the problem, so too is the harsh calculus of hospital administrators who don't want to pay the cost of searching for the reservoirs of germs, said Dr. Victor Yu, a professor of medicine at the University of Pittsburgh who specializes in hospital-acquired germ research ...

"Too many administrators don't want to necessarily find germs inside their facility because repairs to equipment or extensive cleaning can mean shutting down a department or floor. Even a few hours is a significant loss of revenue," Yu said.

Yu and a growing body of infection-control experts are critical of the CDC-endorsed policy known as selective surveillance, in which hospitals don't screen all patients for infections, but target only the sickest or most vulnerable ones. Hospital officials argue that testing every patient is too costly.

Though selective testing identifies many hospital-acquired infections, it allows a significant number of germs and infections to go undetected, leaving colonies in the hospital that eventually can spread into communities, many of the nation's leading hospital epidemiologists say.

Hospitals, with their warm, constant temperature and immune-compromised patients, are ideal incubators for germs and prime hosts for outbreaks. Germs can find dozens of spots to multiply and wait for a person to infect ...

Heart patient Michael Lebedecker, 61, became concerned after health-care workers examined his surgical chest incision without wearing gloves, said his wife, Janet. She said she witnessed a surgical resident use his teeth to tear surgical tape that was placed across a chest bandage.

"My husband was worried about infections, but doctors said not to worry because there were no problems," she said.

Lebedecker, who underwent bypass surgery in 1999, contracted an antibiotic-resistant germ in the hospital and died five weeks after the operation. Hospital records show that he was infected by MRSA (methicillin-resistant staphylococcus aureus).

Lorraine Lydon, 57, survived a similar infection following cardiac bypass surgery, but she now lives a life tethered to oxygen tanks and dozens of expensive medicines. She said she saw instances where health-care workers failed to wash hands between patients, but she did not think the lapses were potentially dangerous at the time.

MRSA (methicillin-resistant staphylococcus aureus) was detected in her sternum within a week after the September 2000 surgery. Doctors told her they never determined the source of the germ.

Doctors have tried so many times to remove infection-ridden bones from Lydon that further surgeries

could prove fatal to her weakened body.

“I’ve already had 18 surgeries, but the germ is still inside me. It will never go away,” she said. “Besides, they’ve already removed virtually every bone in my chest. There’s nothing else to take.”

Investigative records from Florida’s Agency for Health Care Administration show 23 complaints have been filed by patients or employees relating to infections or unsanitary conditions at Palm Beach Gardens from 1997 through 2001 …

At lunchtime, for example, nearly a dozen nurses and other health-care employees streamed outside, carrying cafeteria trays of food to picnic tables on small grassy strips shaded by trees.

As the employees sat at picnic tables, ducks and other fowl darted about their legs in search of fallen crumbs. The birds frequently brushed feathers against the scrub uniforms of the nurses. Birds are considered major carriers of germs, particularly salmonella, which can cause lethal blood poisoning. Birds also can be carriers for staphylococcus germs.

Many employees wore protective slipcovers over their shoes as they trooped into the grass littered with bird feces. They did not remove the contaminated slipcovers before re-entering the hospital…

In one case study, coordinated for the CDC by the Rollins School of Public Health of Emory University in Atlanta, 4,303 samples of staph germs taken from intensive care units were studied. Nearly 36 percent were resistant to the most common and effective antibiotics.

For Buffalo Grove resident Debra Shore, the race to find new antibiotics could become an issue of life or death.

Last year, a staph germ resistant to methicillin infected her right foot, which had suffered complications related to her diabetes. She already had lost three toes to amputation, and the infection caused swelling and intense pain in her foot …

Shore received a new antibiotic that won FDA approval in 2000. The new drug, Zyvox, is marketed by Pharmacia Corp. as an alternative to vancomycin. Medical studies show Zyvox is as effective as vancomycin …

In July, infected by staph again, Shore began a new round of antibiotics to try to save her right foot …

“I fear there will be a day when there are no more drugs to help me,” Shore said. “My doctor said this germ only was found in hospitals years ago. Now it’s everywhere.

“If this germ gets any stronger, I may not be able to survive the next round.”

Back to top

Scientists shocked to find antibiotics alleviate symptoms of schizophrenia

Chance discovery of link between acne drug and psychosis may unlock secrets of mental illness

Thursday 1 March 2012

A cheap antibiotic normally prescribed to teenagers for acne is to be tested as a treatment to alleviate the symptoms of psychosis in patients with schizophrenia, in a trial that could advance scientific understanding of the causes of mental illness.

The National Institute for Health Research is funding a £1.9m trial of minocycline, which will begin recruiting patients in the UK next month. The research follows case reports from Japan in which the drug was prescribed to patients with schizophrenia who had infections and led to dramatic improvements in their psychotic symptoms.

The chance observation caused researchers to test the drug in patients with schizophrenia around the world. Trials in Israel, Pakistan and Brazil have shown significant improvement in patients treated with the drug.

Scientists believe that schizophrenia and other mental illnesses including depression and Alzheimer's disease may result from inflammatory processes in the brain. Minocycline has anti-inflammatory and neuroprotective effects which they believe could account for the positive findings.

Details of the trial were presented to the independent Schizophrenia Commission by Bill Deakin, professor of psychiatry at the University of Manchester, who is the lead investigator. The 12-member commission, set up by the mental health charity Rethink, is looking into the treatment and care of people with schizophrenia, and is due to report in the summer.

The first account of minocycline's effects appeared in 2007 when a 23-year-old Japanese man was admitted to hospital suffering from persecutory delusions and paranoid ideas. He had no previous psychiatric history but became agitated and suffered auditory hallucinations, anxiety and insomnia.

Blood tests and brain scans showed no abnormality and he was started on the powerful anti-psychotic drug halperidol. The treatment had no effect and he was still suffering from psychotic symptoms a week later when he developed severe pneumonia.

He was prescribed minocycline to treat the pneumonia and within two weeks the infection was cleared and the psychosis resolved. Minocycline was stopped and his psychiatric symptoms worsened. Treatment with the drug was resumed and within three days he was better again. Halperidol was reduced but he remained on minocycline. Two years after his psychotic episode, he was still well.

The UK trial aims to recruit 175 patients recently diagnosed with schizophrenia, half of whom will be randomly allocated to take minocycline with their standard anti-psychotic treatment while the remainder take a placebo.

Brain scans will be carried out at the start and end of the 12 month trial to compare loss of grey matter – an effect of schizophrenia – in the two groups. Tests will also measure inflammatory markers in the blood.

Back to top

Does Bacterial Biofilm in the Gut Trigger Lupus?

Nov. 27, 2015

Adding to the growing body of scientific evidence pointing to bacteria as a causative agent of rheumatic diseases, a ground-breaking study, led by researchers at Temple University School of Medicine (TUSM), in Philadelphia, was published in the journal, *Immunity*, on June 16th, 2015.

By using mice that are genetically-prone to lupus, the Temple researchers were able to provoke the onset of the disease by manipulating intestinal microbes that thrive and are protected in a sticky substance, called biofilm, and conclude:

"These data provide a mechanism by which the microbiome and biofilm-producing enteric infections may contribute to the progression of SLE..."

In a study review, published in Science Medicine Newsline on July 6th, 2015, a study author, Dr. Gallucci, Associate Chair, Microbiology and Immunology, as well as an Associate Professor in Microbiology and Immunology at TUSM, remarked:

"This work stresses the importance of considering infections as a possible trigger for lupus," Dr. Gallucci said. "Very little was known about how biofilms interact with the immune system because most of the research has been looking at how biofilms protect bacteria, how they make bacteria resistant to antimicrobials such as antibiotics, but almost nothing was known about what biofilms do to the immune response," she said.

(from http://www.roadback.org/)

Back to top

Can Arthritis Be Triggered By an Infection From a Pet Parrot?

Alma had suffered with arthritic pain from the age of sixteen and was later diagnosed with seropositive rheumatoid arthritis on the basis of her clinical presentation that included an elevated rheumatoid factor.

Her doctors prescribed numerous, common anti-rheumatic drugs throughout the years, but without complete relief. It was when her mother happened to read an article in *Prevention Magazine* about Dr. Brown's antibiotic approach to treating rheumatic diseases and Henry Scammell's book, *The New Arthritis Breakthrough*, that Alma's hopes were raised.

After calling RBF and talking by phone with Henry, she found an experienced AP (Antibiotic Protocols) doctor who was able to identify and diagnose her with an infectious disease, called

Psittacosis.

In Alma's case, the infection was thought to have been passed by the family's pet parrots. After suffering half of her young life with debilitating arthritis caused by this infection and possible heart-related issues, Alma was able to find swift remission with the prescribed antibiotic, minocycline.

Psittacosis, also known as Ornithosis, Parrot Fever or Pigeon Fancier's Lung, is caused by the organism *Chlamydia psittaci*. It can be transmitted to humans from birds, such as, chickens, turkeys, pigeons, cockatiels, parakeets, and ducks, as well as parrots.

In humans, the infection can cause fever and chills, nausea, vomiting and diarrhea, muscle and joint aches, headache, fatigue, weakness, cough and shortness of breath. In some instances, the infection can also affect the heart, liver, brain, or spinal cord and a possible causal link with psoriasis has been considered.

Some researchers have also associated this infection with the HLA B27 haplotype, considered to be a genetic predisposition to reactive arthritis. Treatment typically includes antibiotics, such as tetracycline or doxycycline, erythromycin or azithromycin, and others.

(from http://www.roadback.org/)

Back to top
