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THE RUSSELL BODY: THE FORGOTTEN CLUE TO THE BACTERIAL CAUSE OF CANCER

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Abstract

In 1890 the pathologist William Russell reported spherical forms in histopathologic sections from cancer which he interpreted as "the parasite of cancer." These forms were subsequently discredited as microbial forms but have became known to every pathologist as "Russell bodies." Identified in a wide variety of disease states, these forms are now widely considered to be immunoglobulins. This paper reviews the case for Russell's original belief that these forms are microbial in nature and in origin. It is theorized that Russell bodies are derived from bacterial-sized intracellular organisms that have been reported in cancer, proliferative, and inflammatory diseases by various authors over the past century. It is also suggested that some larger-sized Russell bodies could represent large L-forms (so-called "large bodies") that develop from the small coccal-sized intracellular and extracellular microbes described in cancer. Obviously Russell's idea of a cancer parasite is heretical. However, newer findings of the universal presence of cell wall-deficient bacteria in the blood of all human beings should lead to a reconsideration of the idea that such bacteria might be implicated in the pathogenesis of cancer. Furthermore, Russell bodies might represent cell wall-deficient growth forms of these universal bacteria in histopathologic sections and support Russell's nineteenth century view of an infectious agent in cancer.

Introduction

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, biowarfare, and bioterrorism

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.

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!

William Russell (1852-1940) and "the parasite of cancer"

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'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).

In 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 search of the PubMed database for "bacteria cancer" elicits 49,345 citations. 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. His book, The

Cancer Microbe, is available through Internet sources. A number of his full-length papers on the microbiology of cancer have been published by the Journal of Independent Medical Research (www.JOIMR.org/)

List of Figures.

Figure1: William Russell 1852-1940, as pictured in The British Medical Journal, August 24, 1940.

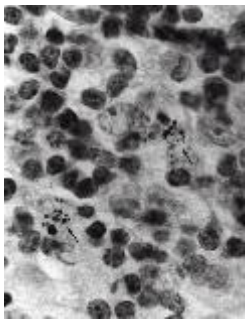

Figure 2: Russell bodies in a lymph node of Hodgkin's disease. Gram's stain, magnified 1000 times, (in oil). (click here to enlarge image).

Figure 3: Solitary "giant" Russell body in a lymph node of Hodgkin's disease (cancer), Gram's stain, magnified 1000 times (click here to enlarge image).

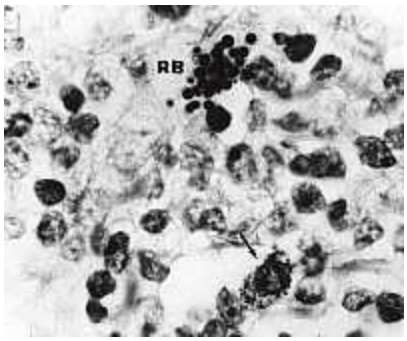

Figure 4: Larger Russell bodies (RB) in a lymph node showing non-cancerous "reactive lymph node hyperplasia" from a fatal case of AIDS. The arrow points to nearby bacterial-sized intracellular coccoid smaller forms from which the Russell bodies are derived. Fite (acid-fast) stain, magnified 1000 times (click here to enlarge image).


Figure 5: Extremely large "super-giant-sized" solitary Russell body in the skin of "cutaneous lupus erythematosus", a so-called "collagen disease." The perfectly round shape, except for one area, suggests this large body is developing inside a cell that is readly to burst. Kinyoun's (acid-fast) stain, magnification x 1000 (click here to enlarge image).

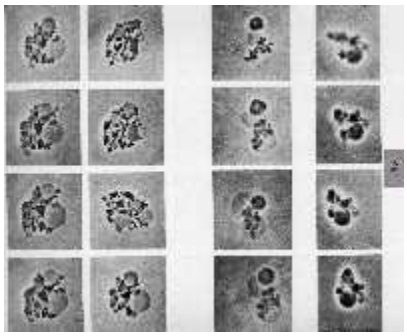
Figure 6: Pleomorphic growth forms (L-forms) of tuberculosis mycobacteria photographed with an electron microscope. Note the darker staining tiny coccal forms (similar in size to ordinary staphylococci) and the larger clear balloon-sized "ghost" forms similar in size and shape to Russell bodies found in tissue. These forms are all characteristic of "cell wall-deficient bacteria" and totally unlike the well-known "typical" acid-fast rod forms of Mycobacterium tuberculosis. Reproduced from L-forms of Mycobacteria and Chronic Nephritis (1970), by Dr. C. Xalabarder P., page 51 (click here to enlarge image).

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