Cancer and infection

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Cancer may be induced through multiple factors. These include environmental factors, carcinogens, as a factor of ageing, genetic mutations, immune system disorders, poor diet and by some viruses. There is a long history found in the scientific literature implicating bacterial infection as cancer induction. For over 100 years scientists have cultured varying bacteria from tumours and in many cases have cured cancer by treating such infections. This area of science has been ignored and at times actively discouraged from full investigation. Bacteria known to induce cancer are neither routinely screened in patients nor routinely treated.

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Our current scientific position on cancer causation is that of induction through multiple factors. These include environmental factors, carcinogens, as a factor of ageing, genetic mutations, immune system disorders, poor diet and by some viruses. There is another history of cancer induction that has been clearly documented in the scientific literature, implicating bacterial infection as cancer induction.

These lesser known areas of cancer research (bacterial induction of cancer) receive minimal mention in most medical textbooks and information spread appears to have been actively discouraged. This suppression of information has occurred in three main ways and continues to occur:

1. Failure to fund such research.
2. Proclamations from people of importance in cancer medicine, trivialising and negating such research results.
3. Omissions from medical teachings of a complete history of research in fields differing from the dominant approaches.

Koch: the rise of bacteriology

The discoveries of Pasteur laid the foundation for the work of other scientists such as Robert Koch (1843–1910), eventually giving rise to the discipline of bacteriology. Koch isolated Bacillus anthracis and inoculated it into mice to cause anthrax, thus convincing the medical community that these tiny bodies, bacteria, actually could cause disease. Koch later also isolated and identified bacteria as causative agents for tuberculosis and cholera.

Koch’s Postulates

Koch is still remembered for his criteria (known as Koch’s Postulates) for judging whether a bacteria could cause a particular disease:

- The bacteria must be present in every case of the disease.
- The bacteria must be isolated from the host with the disease and grown in pure culture.
- The specific disease must be reproduced when a pure culture of the bacteria is inoculated into a healthy susceptible host.
- The bacteria must be recoverable from the experimentally infected host (Medterms 2006).

Current bacteriology has subsequently discovered some exceptions to this definition. For example the...
bacteria *Mycobacterium leprae* that causes leprosy cannot be grown in ‘pure culture’; and generally accepted ‘harmless’ bacteria may cause immense damage if an immuno-compromised patient becomes infected.

### 1884: Tumours contain parasites but are not caused by parasites

In 1884 the President of the Royal College of Surgeons, Dr Henry Butlin, made the following statement:

‘The theory of parasitism, applied to tumours, has during centuries been more or less popular with surgeons; for in no other way can some of the most complicated processes of malignant tumours be so well explained, as by assuming that the tumours or their elements are parasitic. But of late years the parasitic theory has been discredited by the discovery that the elements of even the most malignant tumours are derived more or less directly from the natural tissues of the body, and that they differ only in degree, and, perhaps, in certain properties which they have acquired from the natural elements.

It is quite clear therefore that the view formerly maintained that malignant tumours are actually parasites is incorrect. But the recent discoveries of micro-organisms and of the part they play in relation to certain diseases, have led me to consider whether the theory of parasitism may not again be applied to malignant tumours, with this difference that the tumours are no longer now conceived to be parasites, but to contain them (Butlin 1884).

This is the first reference that can be found on the concept of a bacterial cause of cancer. The term ‘parasite’ was common when referring to bacteria in earlier years. Certainly the search for the elusive ‘parasite’ did not cease then.

### 1889: Parasites found in cancers

This statement was followed in quick succession by work from several other scientists. Dr Thoma published a paper *Ueber-eigenartige parasitare Organismen in den Epithelzellen der Carcinome* (translated as over-peculiar parasitic organisms in the epithelial carcinoma) in 1889 in the journal *Fortschritte der Medicin* (Progress of Medicine).

In 1890 in the same journal, a paper entitled *Ein parasitarer protozoarer Organismus in Carcinomen* (A Parasitic Protozoan Organism in Carcinoma) was published by the scientist Nils Sjobring.

### 1885: Cancer vaccine from bacteria

In 1885 a French scientist Thomson Doyen not only isolated a bacterium (that he named *Micrococcus neoformans*) from tumours, but he also produced a vaccine from the bacteria. He claimed the vaccine produced cures in cancer patients (Doyen 1905).

### 1899: Histology shows parasites in active parts of tumour

The monograph *On the Aetiology and Histology of Cancer*, published in April 1899 by Dr HG Plimmer, outlined various staining and fixing methods to demonstrate cellular inclusions. Plimmer also stated that, over a six year period, he had examined tissue from 1278 cancers (excluding sarcomas) and had found parasitic bodies in 85% of these. Interestingly he did not find these organisms spread homogenously throughout the tumours. The organisms only appeared at the growing edges of the tumours where the cells were active, and not where there appeared to be degeneration or reversal of the tumours (Glover date unknown).

### 1911: Virally-induced cancer

In 1911 Peyton Rous published one of the earliest proofs of virally-induced cancer in *A Sarcoma of the Fowl Transmissible by an Agent Separable from the Tumour Cel*. Significantly it took until 1966 for him to be awarded a Nobel Prize for this discovery.

### 1925: Micrococcus cultured from breast cancer

Dr J Nuzum, in 1925, cultured a minute gram-positive micrococcus unidentified (but possibly a member of the streptococcus group) from a breast tumour. Inoculation with this bacteria into mice and dogs caused the growth of some pre-cancerous lesions and, in some cases, mammary carcinomas. Control mice inoculated with cultures of other strains of streptococcus and staphylococcus did not develop such lesions.

### 1925: Cancer induced by virus with an irritant

Also in 1925 *The Lancet* published a section entitled *New Research into the Origin of Cancer* including papers from Gye and Barnard. Dr Gye had come to the conclusion that cancer was a disease caused by a virus or group of viruses. Although he found that the virus alone was insufficient to induce cancer, in the presence of an
irritation such as coal-tar or paraffin oils the virus would multiply in the cell provoking the host cell to multiply.

Dr Barnard’s paper was on microscopy techniques for the examination of small filterable spheroids. Consistency in microscopy techniques was needed to allow other researchers to view these small organisms.

1930: Pleomorphic forms from cancerous tissue

In 1930 Dr TJ Glover, working at the Hygienic Laboratory in Washington, found an organism that was shown in subculture to be highly pleomorphic: thus its life cycle included coccoids, rods, mycelial stages and filter passing forms. These organisms were able to be stained in cancerous tissue appearing as intracellular forms.

He obtained such an organism from an adenocarcinoma of the human breast. He inoculated the organism into the breast tissue of full grown female guinea pigs and female albino rats. Tissue from the resulting lesions was cultured and the organisms obtained were subcultured several times before being passed through four successive groups of rats. After the fourth passage the rats developed peritoneal carcinomas with metastases to the upper abdomen and peritoneal endotheliomas with focal infiltration.

Glover found this organism in 85% of 3000 cases.

Further studies on pleomorphic forms

Glover’s work was reproduced by Dr JL Engle in Philadelphia and subsequently in a larger study by Dr George A Clark (1953). Clark found that he could consistently isolate a highly pleomorphic organism from blood or tissue biopsies from his cancer patients.

In this study the organisms were cultured from patient tissue and the filtrate injected into two guinea pigs. One was given 1 cc of the filtrate and the other 5 cc. The guinea pig receiving the larger dose died 45 hours later. A drop of blood was aspirated from the heart and the liver of this guinea pig. The blood was cultured and by the next morning the same motile bacillus could again be shown to be present.

In Canada, OC Gruner (1935) was also studying pleomorphic organisms and cancer. He isolated such an organism, which he named Cryptomyces pleomorpha, from a breast tumour. He found that:

- The organism could be detected in circulating blood by direct examination.
- It was detected amongst tumour cells in the original neoplasm.
- An organism of the same type was found in seven previous cases.
- It resembled a fungoid organism, but with additional distinctive features.
- The organism in living cultures mimicked the cell-elements of human blood.

1941: Pleomorphic forms from Hodgkin’s lymphoma

Dr Mazet, a French physician, in Extrait de Montpellier Medicalle (1941), wrote of finding a bacteria in a patient with Hodgkin’s disease. He then cultured an acid fast organism from 12 Hodgkin’s patients. He regarded the organism as highly pleomorphic with phases varying from small granules to fungal type elements, including coccoid forms, mycelia and rod forms. He inoculated blood from a Hodgkin’s patient into a mouse that, when sacrificed 15 days later, yielded the same organism from its brain tissue.

1948: Siphonospora from cancer tissue

In Germany in 1948 Von Brehmer published his work on an organism which he named Siphonospora polymorphs that he claimed caused cancer. He had published earlier (1934) on this organism, which he had cultured from human blood. He found that this organism parasitised epithelial cells as well as erythrocytes and leucocytes. Von Brehmer developed a therapy that involved the use of pooled cultures of Siphonospora isolated from several different types of neoplasm.

1952: Pleomorphic studies of micromyces

From more than 1000 samples of tumour tissue, blood and ascites fluids of cancerous patients, Franz Gerlach (1952) isolated a pleomorphic, filter passing organism that he called Micromyces biastogenes. He later renamed this organism Micromyces universalis innatus and regarded it as a micro fungus. Again this organism was filterable (able to pass through a fine filter). One of the stages in its life cycle resembled a Mycoplasma like organism.
Gerlach produced a ‘polyvalent’ vaccine by passing the organism through numerous passages of culture media. He claimed his vaccine stopped the growth of cancers enabling many patients to go into remission, without side effects (Gerlach 1961).

1955: Cancer ‘virus’ extracted from 1000 cancers

Dr John E Gregory published the last edition of his book Pathogenesis of Cancer in 1955. In it he described finding cell wall deficient forms which he referred to as a cancer virus, extracted from tissue samples of 1000 human cancers.

In total he examined 31 types of cancers and found the virus to be present in all but eight of the 1000 samples. The negative results were found in five Hodgkin’s blood cultures, although he obtained positive cultures from 10 lymph nodes of Hodgkin’s patients. He could not culture the virus from two lymphosarcoma patients’ blood cultures, but had positive results from five other patients with the same disease.

Gregory produced a culture from malignant melanoma which he injected into mice and baby chickens: 25% of the injected animals developed cancers. These included cancers of the ovary, adrenal gland, breast and stomach, spindle cell sarcoma, myosarcoma and leukaemia. His control group, which was larger than the research group by a factor of ten, developed no malignancies.

Gregory found that the virus isolated from the induced cancers was the same as the injectable form and could be re-cultured to again produce the same cancer. Because the types of cancer varied from the original melanoma, he concluded that the inoculations were not cancer cells from the host, but viral forms that induced a cancer.

Early drugs utilising bacterial effect on virus

Gregory experimented with various bacteria to find if they would affect this virus he had found, and had some success in his treatment of cancer patients using the bacteria Bacillus subtilis Tracy 1. He produced a filtrate of this bacteria, mixed it with a saturated magnesium sulphate solution, and gave this to patients as a daily injection.

He showed many remissions using this treatment, particularly in late stage patients for whom no other treatments were used.

1955: Dark field microscopy reveals pleomorphic forms in blood of cancer patients

In Paris Dr E Villequez (1955) used dark field microscopy, noting what appeared to be bacteria in the blood of cancer patients. When cultured the organisms were noted to be highly pleomorphic. He wrote that they had some characteristics that linked them to mycobacteria but that at other times they resembled spore forming bacteria.

1959: Scientist self inoculates with carcinoma isolate

Clara Fonti (1959) wrote on the parasitic theory of cancer and the transmissibility of cancer, citing 30 cases from her own practice. To demonstrate transmissibility, she inoculated herself in the chest wall with fluid from a metastasising mammary carcinoma. After a few days, an erythematous papillary eruption developed between her breasts, growing into a nut-sized lesion with numerous small ancillary papules. These papules were diagnosed as baso-cellular epithelioma. Fonti’s own blood was then transfused to a patient with multiple abdominal metastases, giving an amelioration of the patient’s condition.

1948–1990: Livingston-Wheeler et al, pleomorphic studies on neoplasms

Dr Virginia Livingston-Wheeler worked with many distinguished scientists throughout her long career, including Dr Roy Allen, an expert microscopist and histologist. In August 1948 Allen published The Microscopy of micro-organisms associated with neoplasms in which he stressed the pleomorphic appearance of the microbe isolated from the blood of cancer patients. Cantwell (2005) quotes Allen:

‘He described it as ranging in appearance from a rod-shaped or coccus shaped form. That the non-acid-fast coccal forms could appear as single, double, or as densely-packed round forms. That these coccal forms could vary in size from 1 micron to the smallest microscopic size the eye could detect with a microscope approx. 0.2 microns, and that the microbe could live both inside and outside the cells, and that the tiniest forms of the cancer microbe were filterable and virus sized.’

Livingston-Wheeler collaborated for many years with three well known women scientists who undoubtedly influenced her research and career:
• Eleanor Alexander-Jackson PhD, a Cornell University microbiologist. Her work with the tuberculosis mycobacterium gave her familiarity with the concept of pleomorphism, and she described some of these variants in her PhD thesis (published in the American Review of Tuberculosis).

• Irene Diller PhD, a cell cytologist at the Institute for Cancer Research in Philadelphia and editor of Growth, a biological journal.

• Florence Seibert, a well known refereed tuberculosis researcher, famous for her development of the TB skin test (Cantwell 2005).

The paper Cultural Properties and Pathogenicity of Certain Microorganisms obtained from various Proliferative and Neoplastic Disease was first published in 1950, a team effort by Virginia Wuerthele-Caspé (Livingston-Wheeler’s name from a previous marriage), Eleanor Alexander-Jackson, John Anderson, James Hillier and Roy Allen.

In this they described how they cultured pleomorphic organisms from human and animal neoplasms, and that these could not be cultured from normal controls. When inoculated into experimental animals, the cultured organisms induced characteristic pseudocaseous lesions.

1966: Studies on the Rous virus as a pleomorphic form of Mycoplasma

An important paper was published in 1966 by Dr Eleanor Alexander-Jackson who had been working for some time with the Rous virus. She had isolated many times and over many years a highly pleomorphic, gram variable Mycoplasma from the blood and tumours of Rous virus infected chickens and from other sources of the virus.

Dr Alexander-Jackson postulated that the Rous virus was ‘the virus size stage and virus like form of a single type of pleomorphic intermittently acid fast organism with a Mycoplasma transitional L phase, belonging under the order Actinomycetales’.

1969: Livingston-Wheeler cancer clinic and autologous vaccine

Livingston-Wheeler established her first cancer clinic in San Diego in 1969 and produced an autologous vaccine utilising her Progenitor cryptoceides organism for the treatment of cancer patients. Her later husband, Owen Webster Wheeler, developed a malignant lymphoma of the neck in 1972, and he chose to treat it only with the vaccine. The lymphoma was reportedly gone in six months (Livingston 1977).

1973: Link between bacterial endocarditis and colorectal carcinoma

The possible association between bacterial endocarditis and colorectal carcinoma was raised in 1973 by Dr Daniel Roses and Dr Arthur Localio, following their investigation into three patients presenting with bacterial endocarditis and carcinoma of the colon or rectum (Roses 1974). Each patient was treated with antibiotics for endocarditis followed by surgical removal of the carcinoma.

A causal link between the two conditions must be considered as speculative, but the authors suggested that in patients with no history of heart disease, the concurrent development of these diseases certainly warrants further research.

1970s–1980s: Histology of pleomorphic forms in cancers

Between the late 1970s and early 1980s Dr Alan Cantwell, a dermatologist who considered Dr Livingston-Wheeler somewhat his mentor, began to publish on the presence of pleomorphic organisms he had found in breast cancer, lymphoma, Hodgkin’s disease and pre-AIDS Kaposi’s sarcoma (Cantwell 1981, 1982, 1997).

1993: Mattman on cell wall deficient forms

Professor Lida Mattman’s knowledge of cell wall deficient bacteria and of the strange group of divergent organisms called Mycoplasmas has been invaluable to most researchers interested in this field. Although not the first to work with cell wall deficient bacteria, Professor Mattman’s work has added greatly to the body of information on this phenomenon.

Bacteria that become cell wall deficient have the ability to make enormous changes in their appearance. They have the following characteristics:
They may disintegrate totally if fixed on a slide by heating (the standard method of fixing).

They usually grow on soft agar.

They may grow within red blood cells.

They are often serophilic.

They often grow best in a hypertonic environment (Boca Raton 1997).

### Late 20th century: Mycoplasmas in Gulf War Syndrome patients

The end of the 20th century saw renewed interest in the investigation of Mycoplasmas. Mycoplasmas are of the class Mollicutes and are the smallest of the bacterial forms. Unlike other species of bacteria Mycoplasmas are unable to make cell wall components. They do not enter a cell wall deficient stage but they share many of the characteristics of the cell wall deficient (Boca Raton 1997). The work of Professor Garth Nicolson on the diagnosis and treatment of Gulf War Syndrome patients showed the pathogenicity of Mycoplasma infection. Infections of *Mycoplasma pneumoniae* were identified through antibody testing. Today the polymerase chain reaction (PCR) test is considered the gold standard for identification of such organisms.

### Mycoplasmas inducing chromosomal instability and malignancy

Researchers at the American Registry of Pathology at the Armed Forces Institute of Pathology, Washington, have shown that chronic infection or colonisation by some Mycoplasmas in cell lines induced chromosomal instability and malignant transformation.

Their hypothesis was that chronic infection could promote tumour growth of mammalian cells. They also showed that infection by several, but not all species of Mycoplasma would prevent murine myeloid cells from undergoing apoptosis, and that these Mycoplasma infected cells gradually underwent malignant transformation over a period of four to five weeks. The two Mycoplasma strains used in this study were *M. fermentans* and *M. penetrans* (Lo 1999).

### Affinity of Mycoplasmas for cancer cells

One the most fascinating characteristics of the Mycoplasmas is their affinity for cancer cells. All scientists working with cancer cell lines must continually check for Mycoplasma infection, and many papers are devoted to studies of how to eliminate Mycoplasmas from these cells (Uphoff 2002).

Why these particular species are the most likely contaminants of cancer cells does not yet appear to be answered. Testing for contamination is now recommended, by DNA fingerprinting or cytogenetic analysis, as the effect of Mycoplasmas in the cell lines may render research data highly unpredictable and questionable (Drexler 2002). Much research performed prior to DNA technology, (utilising cell lines) should be repeated with attention to possible infiltration of the cell lines by Mycoplasmas in order to verify the published outcomes of the studies.

### Stats on infection of cancer patients with Mycoplasmas

The numbers of cancer patients who are infected with these bacteria is unknown, but where reasonable studies have been carried out the results are frightening. A study from China by Su Huang (2001) published in the *World Journal Gastroenterology*, gave the results shown below.

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>Number Positive for Mycoplasma</th>
<th>Total Patient Number</th>
<th>Percent Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast Cancer</td>
<td>25</td>
<td>63</td>
<td>39.7%</td>
</tr>
<tr>
<td>Colon Cancer</td>
<td>32</td>
<td>58</td>
<td>55.1%</td>
</tr>
<tr>
<td>Adenomorous polyp</td>
<td>10</td>
<td>49</td>
<td>20.9%</td>
</tr>
<tr>
<td>Gastric Carcinoma</td>
<td>50</td>
<td>90</td>
<td>56.0%</td>
</tr>
<tr>
<td>Oesophageal Cancer</td>
<td>27</td>
<td>53</td>
<td>50.9%</td>
</tr>
<tr>
<td>Lung Cancer</td>
<td>31</td>
<td>59</td>
<td>52.6%</td>
</tr>
<tr>
<td>Glioma</td>
<td>38</td>
<td>91</td>
<td>41.0%</td>
</tr>
</tbody>
</table>

### Connection between *Helicobacter pylori* and gastric cancer

Another bacteria, *Helicobacter pylori*, has been much in the news lately. A Nobel Prize was won by researchers showing its role in the induction of stomach ulcers. This bacteria was classified as a human pathogen in 1994 by the International Agency for Research on Cancer (IARC). *H. pylori* has a positive association with gastric cancer.
According to Correa (2003) more than half the world’s population is infected with *H. pylori*.

**Early misclassification of Mycoplasmas and cell wall deficient forms**

Viruses were distinguished from bacteria, particularly prior to 1940, based on their ability to pass through specific filters. Bacteria were larger so they were trapped by the filter, whereas viruses passed through. The size of the filters used at that time however allowed bacteria such as Mycoplasmas to easily pass through so they would have been mistakenly classified as viruses.

Recent work by Wainwright (2002) showed that the presence of the culture medium affected the ability of bacteria to pass through a 0.2 micron filter. When bacteria were given overnight incubation in a culture medium on the membrane, they formed small cell wall deficient forms that were able to pass through the filter. The bacteria that Wainwright used were all common human pathogens. This finding has significant repercussions for the field of microbiology and may indicate that studies carried out over the earlier part of the 20th century should be re-examined.

**High incidence of infection in cancer**

A 1997 paper by Pisani et al estimated that in 1990, 15.6% of the worldwide incidence of cancer could be attributed to infection with the Hepatitis B or C viruses, *Helicobacter pylori*, schistosomes or liver flukes. In developing countries the prevention of these infections would lower the cancer rate by 21%. The papilloma viruses are attributed with causing 89% of cancers of the cervix.

**Salmonella infections linked to gall bladder cancer**

Strong epidemiological evidence supports a link between infections with *Salmonella typhi* and gallbladder cancer. Carriers of *S. typhi* have 8.47 times the risk of gallbladder carcinoma compared with those who have had typhoid and have successfully cleared the infection (Lazzano-Ponce 2001, Welton 1979, Caygill 1994).

**Chlamydophila pneumoniae in lung cancer**

Chronic infections of *Chlamydophila pneumoniae* are now being found to correlate with an increased risk of lung cancer (Koyi 2001, Kocazeybek 2003, Anttila 2003). An elevated IgA antibody titre to *C. pneumoniae* has been reported to be associated with a 50% to 100% increased cancer risk (Littman 2004).

**Escherichia coli and Streptococcus bovis in colon cancer**

Several bacteria have now been linked to chronic infections of the colon and an increased risk of colon cancer. These include *Escherichia coli* (McCoy and Mason suggested this in 1951) and in more recent studies *Streptococcus bovis*. Colon cancer incidence that may be associated with *S. bovis* has been estimated at 18% to 62% (Zarkin 1990).

**Infection in oral squamous cell carcinomas**

Over 90% of oral cancers are oral squamous cell carcinomas (OSCC). These have one of the lowest survival rates (based on 5-year survival statistics), with no noticeable improvements in the last few decades (Canto 2002).

In a recent study (2005) Mager et al used DNA identification of oral flora to test for 40 microbial species. *Capnocytophaga gingivalis*, *Prevotella melaninogenica* and *Streptococcus mitis* were elevated in the saliva of patients with OSCC. When testing the presence of these three species as diagnostic markers, the authors found that their presence could predict 80% of cancer cases and absence could predict 83% of controls.

The sensitivity of nested PCR as compared to a kit method PCR (DNA probe) is significantly greater and allows detection of one organism per mL of blood. At Australian Biologics in Sydney we carried out a small scale PCR study of breast cancer patients choosing only patients who had not had conventional treatments such chemotherapy or radiotherapy. This was to eliminate any question of possible bacterial presence being due to immune suppression as a result of treatment.

**PCR results**

Eleven women were tested with varying types of breast cancer for the presence of the *Mycoplasma* species, the *Chlamydia* species and for *Streptococci*. The results were as follows:

- *Mycoplasma* spp. DNA was detected in 5/11 breast cancer patients (45.4%)
- *Chlamydia* spp. was detected in 2/11 (18.1%)
- No *Streptococcus* DNA was amplified.
**Case Study 1**

Male, 59, past history of prostate cancer, currently diagnosed with follicular lymphoma with multiple nodes. Nested PCR was carried out on the following sample types with the following results.

Lymphoma tissue: *M. fermentans*+ *M. hominis*+
Blood: *M. fermentans*+ *M. hominis*+ 
Bone marrow: *M. fermentans*+

**Treatment:** Klacid® (clarithromycin) and Augmentin® (amoxycillin/clavulanate).

**Response:** PSA increased from 3% to 12%.

**Case Study 2**

Male 63, diagnosed with prostate cancer. Nested PCR was carried out on the following sample types with the following results.

Swab (urethral): *Mycoblastella fermentans*+
Urine: *Mycoblastella fermentans*+
Semen: *Mycoblastella hominis*+

**Treatment:** Klacid® and Augmentin®.

**Response:** PSA increased from 5.1 to 5.8.

Current guidelines for the interpretation of free/total PSA ratio are: less than 10% is suggestive of prostatic cancer; between 10% and 25% is equivocal; greater than 25% is suggestive of benign prostatic disease.

While these cases were treated using orthodox antibiotics, particular consideration should be given to using specific herbal antibiotics, antimicrobials and other supportive medicines for cases such as these. These could include herbs such as Astragalus membranaceus, Echinacea species, Allium sativum, Calendula officinalis, Hydrastis canadensis, Berberis species, Tabebuia avellanendae, Phytolacca decandra, Olea europea, Thuja occidentalis, Thymus vulgaris, Hypericum perforatum and specific organ remedies, along with antimicrobial foods such as garlic, ginger and shiitake, and appropriate supplements.

Patients presenting with a cancer diagnosis are neither routinely tested for pathogenic infections nor routinely treated for infections at any time during their cancer treatment. There are as yet no studies showing the outcome for patients if such infections are identified and eliminated when cancer is first diagnosed.

Ignoring the possibility of bacterial induction of cancer in screening, or as a requirement in treatment, means that no understanding is gained of the possible benefits of such treatment for cancer patients. The potential value of the vast array of effective herbal antibiotics and other specifics in the treatment of cancer cannot be denied. The future expansion of these and other treatment modalities hopefully will answer this question and lead to improvements in survival.

**References**


Clark GA. 1953. Successful Culturing of Glover's Cancer Organism and Development of Metastasizing Tumours in Animals Produced by Cultures from Human Malignancy. *Sixth International Congress of Microbiology* Rome Italy.


In 1985 Jennie Burke established Australian Biologies Testing Services. This laboratory provides tests for holistic practitioners and reflects a naturopathic philosophy. Jennie’s training is in medical technology, herbal medicine and nutrition. Jennie has lectured at meetings of the German and Austrian Societies of Oncology, the International Cancer and Nutrition Society, at two World Breast Cancer Conferences in Canada and at the Collegium Humanum in Switzerland. She has been awarded life long memberships in the Societies of Oncology of both Austria and Germany. Jennie has been convenor of the 3 World Congresses on Cancer held in Australia, bringing together scientists from thirteen countries.