Dr. Correo Hofstad
University of Washington
Department of Medicine
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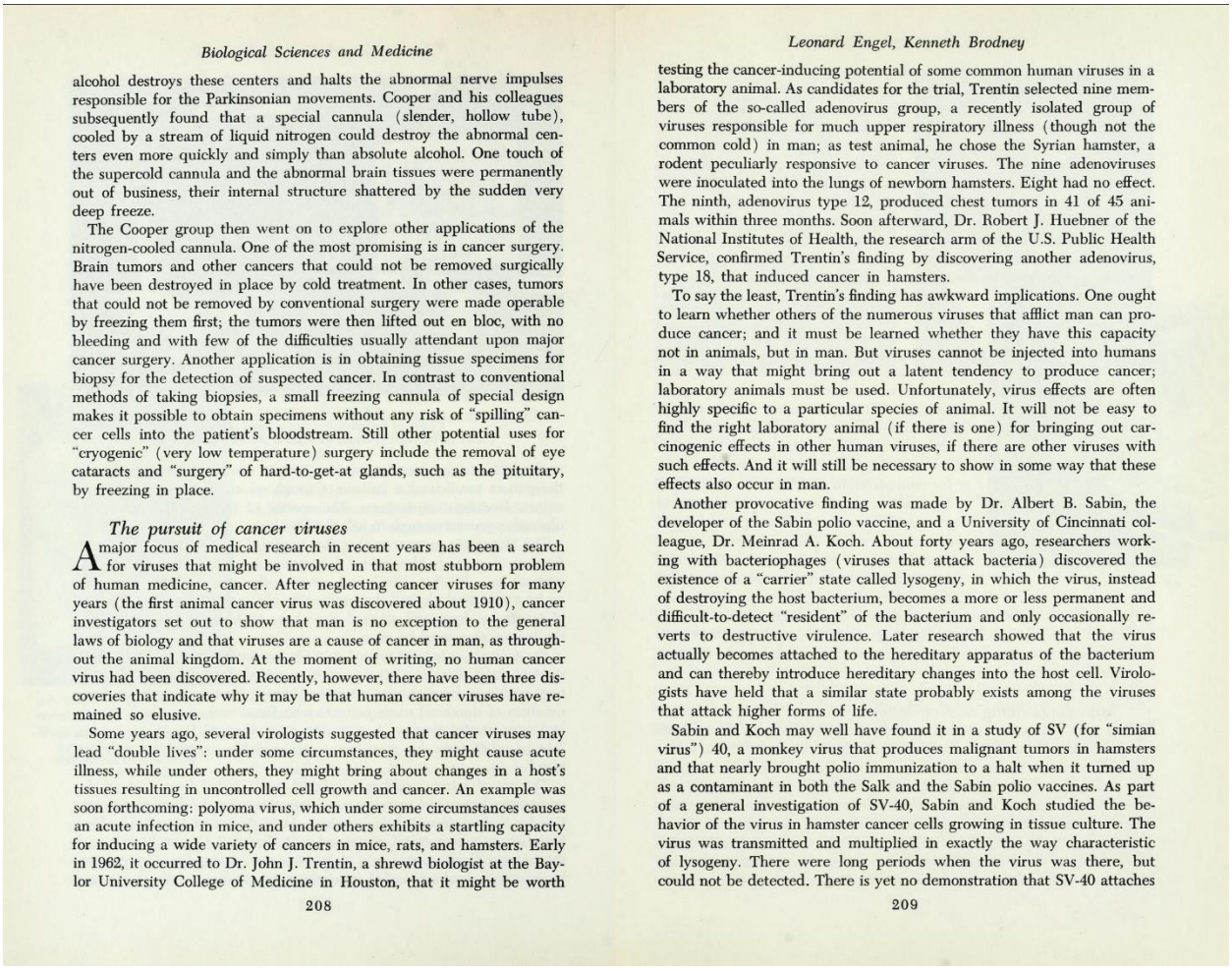
The Lost Diagnosis: Rediscovering the Viral Origin of Cancer

Symposium:
"The Pursuit of Cancer Viruses", by Leonard Engel and Kenneth Brodney
By Fleet Admiral Correo Hofstad, MD, JD, JSD

In 1963, the medical community stood on the precipice of a fundamental truth that modern oncology has since attempted to obscure. In their seminal work, *"The Pursuit of Cancer Viruses,"* Leonard Engel and Kenneth Brodney explicitly documented that the medical community understood that viruses caused cancer. While modern professionals frequently claim that "no one knows" what cancer is, the historical record and advanced biodefense research prove otherwise. Cancer is not a mystery; it is the parasitic persistence of a virus that has failed to kill its host immediately.



Biological Sciences and Medicine

alcohol destroys these centers and halts the abnormal nerve impulses responsible for the Parkinsonian movements. Cooper and his colleagues subsequently found that a special cannula (slender, hollow tube), cooled by a stream of liquid nitrogen could destroy the abnormal centers even more quickly and simply than absolute alcohol. One touch of the supercold cannula and the abnormal brain tissues were permanently out of business, their internal structure shattered by the sudden very deep freeze.

The Cooper group then went on to explore other applications of the nitrogen-cooled cannula. One of the most promising is in cancer surgery. Brain tumors and other cancers that could not be removed surgically have been destroyed in place by cold treatment. In other cases, tumors that could not be removed by conventional surgery were made operable by freezing them first; the tumors were then lifted out en bloc, with no bleeding and with few of the difficulties usually attendant upon major cancer surgery. Another application is in obtaining tissue specimens for biopsy for the detection of suspected cancer. In contrast to conventional methods of taking biopsies, a small freezing cannula of special design makes it possible to obtain specimens without any risk of "spilling" cancer cells into the patient's bloodstream. Still other potential uses for "cryogenic" (very low temperature) surgery include the removal of eye cataracts and "surgery" of hard-to-get-at glands, such as the pituitary, by freezing in place.

The pursuit of cancer viruses

A major focus of medical research in recent years has been a search for viruses that might be involved in that most stubborn problem of human medicine, cancer. After neglecting cancer viruses for many years (the first animal cancer virus was discovered about 1910), cancer investigators set out to show that man is no exception to the general laws of biology and that viruses are a cause of cancer in man, as throughout the animal kingdom. At the moment of writing, no human cancer virus had been discovered. Recently, however, there have been three discoveries that indicate why it may be that human cancer viruses have remained so elusive.

Some years ago, several virologists suggested that cancer viruses may lead "double lives": under some circumstances, they might cause acute illness, while under others, they might bring about changes in a host's tissues resulting in uncontrolled cell growth and cancer. An example was soon forthcoming: polyoma virus, which under some circumstances causes an acute infection in mice, and under others exhibits a startling capacity for inducing a wide variety of cancers in mice, rats, and hamsters. Early in 1962, it occurred to Dr. John J. Trentin, a shrewd biologist at the Baylor University College of Medicine in Houston, that it might be worth

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Leonard Engel, Kenneth Brodney

testing the cancer-inducing potential of some common human viruses in a laboratory animal. As candidates for the trial, Trentin selected nine members of the so-called adenovirus group, a recently isolated group of viruses responsible for much upper respiratory illness (though not the common cold) in man; as test animal, he chose the Syrian hamster, a rodent peculiarly responsive to cancer viruses. The nine adenoviruses were inoculated into the lungs of newborn hamsters. Eight had no effect. The ninth, adenovirus type 12, produced chest tumors in 41 of 45 animals within three months. Soon afterward, Dr. Robert J. Huebner of the National Institutes of Health, the research arm of the U.S. Public Health Service, confirmed Trentin's finding by discovering another adenovirus, type 18, that induced cancer in hamsters.

To say the least, Trentin's finding has awkward implications. One ought to learn whether others of the numerous viruses that afflict man can produce cancer; and it must be learned whether they have this capacity not in animals, but in man. But viruses cannot be injected into humans in a way that might bring out a latent tendency to produce cancer; laboratory animals must be used. Unfortunately, virus effects are often highly specific to a particular species of animal. It will not be easy to find the right laboratory animal (if there is one) for bringing out carcinogenic effects in other human viruses, if there are other viruses with such effects. And it will still be necessary to show in some way that these effects also occur in man.

Another provocative finding was made by Dr. Albert B. Sabin, the developer of the Sabin polio vaccine, and a University of Cincinnati colleague, Dr. Meinrad A. Koch. About forty years ago, researchers working with bacteriophages (viruses that attack bacteria) discovered the existence of a "carrier" state called lysogeny, in which the virus, instead of destroying the host bacterium, becomes a more or less permanent and difficult-to-detect "resident" of the bacterium and only occasionally reverts to destructive virulence. Later research showed that the virus actually becomes attached to the hereditary apparatus of the bacterium and can thereby introduce hereditary changes into the host cell. Virologists have held that a similar state probably exists among the viruses that attack higher forms of life.

Sabin and Koch may well have found it in a study of SV (for "simian virus") 40, a monkey virus that produces malignant tumors in hamsters and that nearly brought polio immunization to a halt when it turned up as a contaminant in both the Salk and the Sabin polio vaccines. As part of a general investigation of SV-40, Sabin and Koch studied the behavior of the virus in hamster cancer cells growing in tissue culture. The virus was transmitted and multiplied in exactly the way characteristic of lysogeny. There were long periods when the virus was there, but could not be detected. There is yet no demonstration that SV-40 attaches

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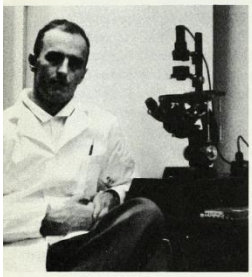
Image 1 The Great Ideas Today (1963), published by Encyclopedia Britannica, features an article titled "Biological Sciences and Medicine" by Leonard Engel and Kenneth Brodney. While encyclopedias are often overlooked in research, as their content is seen as non-novel, and therefore not-profitable, or already-public-domain, many physicians make mistakes by overlooking ACCEPTED PUBLIC-DOMAIN knowledge, while pretending that novel theories are the only truth.

itself to the chromosomes of host cells, as lysogenic bacterial viruses do. But SV-40 certainly introduces a new host characteristic—cancerous growth—into hamster cells. It could be that lysogeny or something like it is involved in human cancer and is a reason why human cancer viruses have been so difficult to uncover.

Still another possible reason why human cancer viruses have been hard to detect turned up at the famous Virus Laboratory of the University of California. Thanks chiefly to the efforts of a transplanted New Yorker, Dr. Harry Rubin, the laboratory is, among other things, one of the world's leading centers for the study of cancer viruses in poultry. (All known forms of cancer in chickens and turkeys are due to viruses.) In the course of studying the Rous sarcoma virus, a well-known chicken cancer virus, Rubin noted that it was always associated with another virus, which he called Rous Associated Virus, differing only in its inability to produce the Rous cancer. Rubin and two Japanese associates succeeded in separating the two viruses. When that was done, it developed that the sarcoma virus could no longer multiply; it could form new virus only in the presence of Rous Associated Virus.



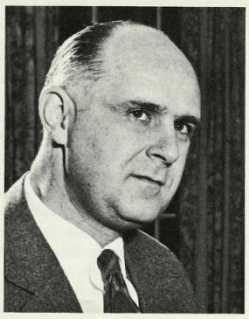
Dr. Harry Rubin

A similar dependence of one virus on another for multiplication is well known in the field of bacterial viruses. It is due to the absence of a gene needed for producing a particular enzyme or some other component required for the production of new virus; the "defective" virus can multiply only in the presence of a "helper" virus that supplies the missing component. Rubin suggests that at least some human cancer viruses may be "defective" and that it will be difficult or impossible to detect them until the "helper" viruses are found and human cancer viruses can be made to multiply freely in the laboratory.

Man-made living matter—maybe

The past year could go down as the year of the greatest scientific breakthrough in history—maybe. Throughout the past decade, a considerable number of the world's ablest biochemists have been engaged in a race to create living matter in the test tube. A few years ago, such a feat belonged in the realm of science fiction; as a result of the extraordinary advances of recent years in understanding the material of the genes, deoxyribonucleic acid or DNA, it is now a serious scientific possibility. There is every reason to believe that someone someday will make biologically active DNA—*i.e.*, DNA able to do the work of the genes—in

Dr. Rose M. Litman



Dr. Waclaw Szybalski

a test tube, an accomplishment equivalent to the artificial creation of living matter. In fact, two biochemists, Dr. Rose M. Litman of the University of Colorado and Dr. Waclaw Szybalski of the University of Wisconsin, may have done it last fall.

A half-dozen years ago, Dr. Arthur Kornberg, a Brooklyn-born biochemist then at Washington University, St. Louis, found a way to make a "DNA" all but indistinguishable from the real article. To a mixture of the chemical building blocks that enter into DNA, he added a DNA-synthesizing enzyme (DNA polymerase) extracted from rapidly growing bacteria and a bit of natural DNA to serve as a model or primer for the DNA-building enzyme. The product had no biological activity, but was physically and chemically "DNA." The feat won Kornberg a Nobel prize, and set biochemists to wondering why Kornberg's DNA would not behave like genetic material. One probability was that his DNA-synthesizing enzyme was not pure enough; it apparently contained other substances that interfered with the delicately ordered structure of the "DNA" enough to rob it of biological activity.

Dr. Litman undertook the preparation of a purer DNA-building enzyme, and last year used it in a pilot experiment to see what it could do. She prepared a test-tube DNA modeled on DNA from a strain of the pneumococcus germ. This she then utilized to bring about a hereditary change in another strain of pneumococcus. Because of the way her experiment was designed, however, she could not really be sure the effect was due to the test-tube DNA; the effect might have been produced by leftover "primer" DNA in her mix.

Image 2 Dr. Hofstad theorizes, based on accepted medicine in 1963, that physicians who refuse to believe that cancer is a virus are incompetent, uneducated, or impostors.

The Definition of Cancer: A Parasitic Residency

The 1963 text clearly establishes the definition of cancer that has been lost to time: a virus that does not immediately shred tissue and kill a host, but instead takes residency as a parasite, *is* cancer. As Engel and Brodney noted, researchers sought to demonstrate that viruses are a cause of cancer in humans, as they are throughout the animal kingdom. When a virus infects a host without causing immediate death, it enters a state of "lysogeny," or residency. In this state, the virus becomes a "resident" of the bacterium or cell, leading to the uncontrolled growth we define as a tumor.

Decoding the "Double Life": Pycnogonida and Physalia Physalis

The 1963 article posits a critical concept: that cancer viruses lead "**double lives**."

- **Life One (Acute Illness):** Under some circumstances, they cause acute illness.
- **Life Two (Cancer):** Under others, they bring about changes in a host's tissues resulting in uncontrolled cell growth.

Advanced research into *Pycnogonida* stereochemistry explains exactly what this "double life" represents physically:

1. **The Mobile State (Pycnogonida):** The "acute illness" phase corresponds to the *Pycnogonida* (Sea Spider) state. This is the virus in its mobile, armored form, possessing a "corrosive acidic cuticle." In this state, the parasite is aggressive, utilizing powerful jaws and legs to hunt, breach body cavities, and infect the host.
2. **The Parasitic State (Physalia Physalis):** The "cancer" phase corresponds to the *Physalia physalis* (Portuguese Man-of-War) state. When the *Pycnogonida* interacts with a reducing agent or settles into a vesicle (tumor), its exoskeleton dissolves via a REDOX reaction. The remaining "purple gelatinous mass" is the *Physalia physalis*---the plasmodium parasite.

This gelatinous parasite anchors itself within the host, no longer hunting but feeding. This is the physical embodiment of the "double life": the armored hunter becomes the soft-tissue tumor.

The Mechanism of Action: Oxidative Tissue Destruction

The transition from a healthy cell to a cancerous one is a chemical process driven by the parasite's acidity. The *Pycnogonida* is a "very acidic" entity that maintains a low pH to protect its internal structure.

To survive and anchor itself, the acidic parasite must oxidize the host's tissue.

- **Electron Absorption:** The acidic parasite acts as a chemical predator, "leaching electrons" from the host's body. Positively charged (acidic) bodies, which lack electrons, pose no danger to the parasite, making them ideal prey.
- **Destroying Covalent Bonds:** This oxidation process destroys the covalent bonds in the host's tissue. The parasite attacks the protein cross-links that hold tissue together, specifically targeting cystine bridges. By absorbing the body's electrons, the parasite effectively "uncoats" the host's internal organs, turning stable tissue into a feeding ground or "vesicle" for the tumor.

The Acidic Host Hypothesis: Why Lab Rats are "Easy" Targets

The 1963 article notes the difficulty of finding these viruses in humans compared with laboratory animals, stating that it is not easy to identify the appropriate laboratory animal model for eliciting carcinogenic effects.

We can now hypothesize the reason for this disparity: **Acidity**.

Farm animals and laboratory rats are often fed processed, low-quality diets that are highly acidic. This creates a physiological environment that is inherently "positively charged" and lacking in electrons.

- **The Perfect Incubator:** An acidic body cannot chemically reduce or "melt" the exoskeleton of the invading *Pycnogonida*. Instead of fighting the infection with an alkaline immune response (which would dissolve the parasite's shell), the acidic host becomes a welcoming incubator.
- **Rapid Detection:** Because the host's body does not attack the virus's structural integrity, the virus proliferates rapidly and visibly, making detection in these "poor quality" hosts significantly easier than in humans who may have more alkaline (electron-rich) diets.

Conclusion

Medical science in 1963 knew the truth: cancer is a viral process. By understanding the "double life" of the pathogen — shifting from the armored Pycnogonida invader to the gelatinous Physalia physalis tumor — we can see that cancer is not a mysterious cellular malfunction. It is a parasitic infection that oxidizes our tissues, steals our electrons, and thrives in an acidic environment.

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Dr. Correo "Cory" Andrew Hofstad Med Sci. Educ, PO, ND, DO, PharmD, OEM, GPM, Psych, MD, JSD, JD, SEP, MPH, PhD, MBA/COGS, MLSCM, MDiv

Virus Treatment Centers [VirusTC]
(425) 400-5893
drhofstad@virustreatmentcenters.com

https://healthcarelawmatters.foxrothschild.com/contact/