

Second thoughts about viruses, vaccines, and the HIV/ AIDS hypothesis - part I

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Viruses

“In the sciences, people quickly come to regard as their own personal property that which they have learned and had passed on to them at the universities and academies. If however, someone else now comes along with new ideas that contradict the Credo (that has been recited for years and passed on in turn to others) and in fact even threaten to overturn it, then all passions are raised against this threat and no method is left untried to suppress it. People resist it in every way possible: pretending not to have heard about it; speaking disparagingly of it, as if it were not even worth the effort of looking into the matter. And so a new truth can have a long wait before finally being accepted.”-Goethe

Introduction

The first isolation of a virus was achieved in 1892 by Russian bacteria hunter Dimitri Iwanowski, who gathered fluid from diseased tobacco plants. He passed this liquid through a filter fine enough to retain bacteria; yet to Iwanowski's surprise, the bacteria-free filtrate easily made healthy plants sick. In 1898 a Dutch botanist, Martinus Willem Beijerinck, repeating the experiment, also recognized that there was an invisible cause and named the infectious agent “tobacco mosaic virus.” In the same year as Beijerinck's report, two German scientists purified a liquid containing filterable viruses that caused foot-and-mouth disease in cattle (viruses were at one time called “filterable viruses,” but eventually the term “filterable” came to apply only to viruses, and was dropped). Walter Reed followed in 1901 with a filtrate responsible for yellow fever, and soon dozens of other disease-causing viruses were found.

In 1935 another American, Wendell M. Stanley, went back to the beginning and created pure crystals of tobacco mosaic virus from a filtered liquid solution. He affirmed that these crystals could easily infect plants, and concluded that a virus was not a living organism, since it could be crystallized like salt and yet remain infectious. Subsequently, bacteriologists all over the world began filtering for viruses, and a new area of biology was born-virology.

Historically, medical science has vacillated on the question of whether a virus is alive. Originally it was described as nonliving, but is currently said to be an extremely complex molecule or an extremely simple microorganism, and is usually referred to as a parasite having a cycle of life. (The term “killed” is applied to certain viral vaccines, thus implying an official conviction that viruses live.) Commonly composed of either DNA or RNA cores with protein coverings, and having no inherent reproductive ability, viruses depend upon the host for replication. They must utilize the nucleic acids of living cells they infect to reproduce their proteins (i.e., trick the host into producing them), which are then assembled into new viruses like cars on an assembly line. Theoretically, this is their only means of surviving and infecting new cells or hosts.

Birth of virology-a miscarriage?

Underlying the birth of virology was the doctrine of monomorphism-that all microorganisms (herein called microforms) are fixed species, unchangeable; that each pathological type produces (usually) only one specific disease; that microforms never arise endogenously, i.e., have absolute origin within the host; and that blood and tissues are sterile under healthy conditions. This last point warrants immediate comment. Theoretically, under ideal health conditions the blood might be sterile, though it has the inherent potential to develop morbid microforms, as discussed in the main text of this book. Long and repeated observation of live blood in the phase-contrast, dark-field microscope, however, shows that the blood can contain various microforms in an otherwise asymptomatic host, or in a condition defined as normal or healthy in orthodox terms. The forms are easily visible before other physical symptoms arise. (Since long and repeated observation has correlated their presence with other disease symptoms and their disappearance with the return of health, they serve as indicators of impending outward signs of disease.)

Monomorphism was the cornerstone of developments in 20th-century medical research and treatments. Refusal by the mainstream to examine fairly, much less accept, the demonstrated facts of pleomorphism-that viruses and bacteria (and also yeast and fungi) are evolutions from the microzoma; that microforms can rapidly change their form (evolve and “devolve”) in vivo, one becoming another dependent upon conditions in the inner terrain (environment); that blood and tissues are not necessarily sterile; and that there are no specific diseases, but only specific disease conditions-was the foundation of a latter day “Galileo debate.” It is so called because those who wore the “robes” of scientific authority, reprising the religious fanatics who punished the noted astronomer for his truths, would not be swayed from folly when presented with its contrary proofs. These proofs began in earnest with Antoine Bechamp in the last century (who also endured the indignation of a fanatical clergy).

In the early third of the 20th century, the heated debate took place over filterable bacteria versus non-filterable. This was a major battle

concerning micromorphology (discussed briefly below). The orthodox view prevailed: bacterial forms were not small enough to pass, or did not have a smaller, earlier stage. What passed through “bacteria-proof filters was something else, i.e., viruses. Standard medical textbooks long made this fettering distinction between bacteria and viruses. Subsequently, however, the cellular nature of many filterable forms originally thought to be viruses, such as some mycoplasmas, rickettsias, and various other groups, has been established. In this writer’s opinion, with the victory of the monomorphic view, deeper understanding of infectious “disease” was lost, setting the stage for cancer, degenerative symptoms and AIDS.

What you see?

A typical bacterium is about 1 micron in size. Most filterable forms now called viruses range in size from .3 microns (300 millimicrons) to .01 micron (10 millimicrons)—partially in the colloidal range (.1 to .001 micron). Most of the larger viruses are a third to a quarter the size of the average bacterium. Size is critical because .3 microns is the resolution limit of modern-day light microscopes (except for the claimed resolution of Canadian microscopist Gaston Naessens’ Somatoscope, at .015 microns). Thus, as viruses were discovered (except for the very large ones, such as mumps), they required an electron microscope to be seen, especially given the fact that Royal Rife’s microscope technology and career were destroyed by vested interests. Unfortunately, electron microscopes and the process of chemical staining disorganize all specimens, whereas Rife’s technology allowed life to proceed and thus evolve under its lens. As viruses became visible to advancing technology, the ramification was that the technology revealed, to minds infected with monomorphism, protein structures deemed foreign to the body.

A new theory

Formulated by Bechamp in the 19th century, microzymian principle is the basis of a new theory about “viruses.” Briefly, this principle holds that in all living organisms are biologically indestructible anatomical elements, which he called microzymas. They are independently living organized ferments, capable of producing enzymes and capable of evolution into more complex microforms, such as bacteria. Bechamp’s thesis is that disease is a condition of one’s internal environment (terrain); that disease (and its symptoms) are “born of us and in us”; and that disease is not produced by an attack of microentities but calls forth their endogenous evolution. (The common biological basis for this is discussed below.)

My studies and research suggest that the complexes science calls viruses and retroviruses originate in the cell as microzymian principle suggests. However, they are created in response to an alarming situation (condition of disease) for the purpose of genetic repair. They are repair proteins evolved from anatomical elements (microzymas), not pathogenic organisms.

It is known that normal cell activity includes genetic repair. Both enzymes and proteins must be involved. What is the mechanism? Viruses are organized around DNA or RNA, not both. Thus, they are quite probably intended to repair genetic molecules or other structures, and show up with disease symptoms because the body needs them. Since viruses require a living cell/host for reproduction, how do we know that the scenario is not set in motion for a purpose by the cell (i.e., its microzymas), rather than being the result of invasion? Because disease (disturbance of balance in the organism) is so prevalent, especially that which has not yet become indicated by common symptoms, repair proteins may be frequently or constantly present. A toxified cell may easily suffer localized damage to the genome. Since

most observers are not even aware of microzymian principle, much less understand or even consider it, and since monomorphism stresses invasion, these protein complexes are regarded as foreign and disease is attributed to them.

Another note of interest is the size of viruses compared to the microzyma. Viruses are considered to be some of the smallest biological particles and are frequently of colloidal size: e.g., hepatitis A, 27 nanometers (.027 microns); hepatitis B (.042 microns); poliovirus (.03 microns); EBV (.042 microns); fflV (.080 to .12 microns), influenza (.08 to .12 microns); mumps (.15 to .30 microns); smallpox (.30 x .24 microns); and, according to Bechamp, the microzyma (.0005 microns). This coincides with what Gaston Naessens says about the size of his somatid, which ranges from “a few Angstroms to a tenth of a micron”.¹

In his book, *The Blood and Its Third Anatomical Element*, Bechamp states: “The microzyma is at the beginning and at the end of all organization. It is the fundamental anatomical element whereby the cellules, the tissues, the organs, the whole of an organism are constituted living. ... In a state of health the microzymas act harmoniously and our life is, in every meaning of the word, a regular fermentation. In the condition of disease, the microzymas do not act harmoniously, the fermentation is disturbed, the microzymas have either changed their function or are placed in an abnormal situation by some modification of the medium”.² The virus is either a self-ordered microzymian polymerization, or (less likely) a structure made by microzymas. It is enveloped in protein which is also composed of microzymas, and could well be thought of as an autonomous molecular tool box.

Along with Drs. Glen Dettman and Archie Kalokerinos, I wonder, “whether Bechamp’s writing anticipated, in some respects, the discovery of RNA and DNA?” Could the genetic structure be the construct, thus a tool, of the microzyma? They quote a personal communication (1974) from a Professor Bayev of the USSR Academy of Sciences, who discusses his work showing that molecular self-restoration from its parts of pure transfer RNA from brewer’s yeast is possible.³

In my own research I have found molecular restorations similar to that described by Bayev. In my experiment I used five-year-old coagulated capillary blood from a woman with cancer. With one drop of 0.9% of sodium chloride, the blood was restored to an appearance and level of activity characteristic of a freshly drawn sample. In other words, the anatomical microzymas of the dried blood were restored to activity. Even the white globules became active. One might eagerly ask for an explanation of the reversal of polymers made during clotting. It is unclear at this point how this reversal takes place, except to say that what can evolve apparently has the potential to devolve. It is observable, however. For example, I have seen, and recorded on video, rod microforms retrograding without any visible decomposition from 10 microns in length to the vicinity of .1 micron.

This research supports the very important postulate that the cell is not the smallest living biological unit, as promulgated by conventional medical science. In fact, a smaller biological unit is the imperishable microzyma, which is an organized, living being “of a special category without analogue,” said Bechamp, who found them ready to become active in chalk deposits at least 11 million years old.⁴

The pleomorphic cycle

I suggest a developmental cycle in vivo consisting of three macrostages: (1) a primitive stage comprising the repair protein complexes; (2) an intermediate, or bacterial, stage including filterable

forms such as the cell-wall deficient forms described by Lida Mattman, Ph.D. (in Cell Wall Deficient Forms, Stealth Pathogens); and (3) a culmination stage consisting of yeast and fungal phases, and then mold, the end phase. The usual course of development would be from microzyma to repair protein and then to bacterium, etc. However, under certain conditions, such as trauma for example, it is highly likely that the microzymas can skip the primitive stage and become bacteria directly. Although these transformations are as astounding as that of a larva to a butterfly, what is equally impressive under observation is the rapidity with which they can take place—in minutes, even seconds, and sometimes. By the same token, when provoked by conditions and the cycle proceeds to yeast, fungus and then mold, it may occur so rapidly that the bacterial stage, if it happens, has no time to be of any significance.

Thus, symptomatic microforms can originate within higher organisms without invasion, via a permutation of the endogenous microzymas when the situation calls for such change. The situation is an imbalance referred to by Bechamp as a “modification of the medium.” Endogenous evolution is evident under the microscope when bacterial, yeast, and fungal forms are seen coming out of red blood cells which initially appear normal.

Biological basis for the pleomorphic cycle

There is a common biological basis for the pleomorphic cycle and its increasing complexity of organization: More complex forms evolve inherently upon the death of an organism for the purpose of recycling its anatomical and chemical structures in the carbon cycle. The process of rapid evolution (which is reversible) is an essential life process which, beyond the repair stage, is necessary to return a dead organism to the earth. The second and third-stage microforms degenerate the body’s vital substances and tissues via putrefaction (bacteria) and fermentation (yeast and fungus). Fermentation results in acid waste products, which further break down tissue. Disease symptoms, then, especially the degenerative type, are not produced by viruses, but manifest as chemical decomposition, or attempted recycling via fermentation and acid toxins, but with “host” survival processes still operable. Obviously, certain other factors may play important roles in producing symptoms, such as heavy metal toxicity, or state of mind, for example. Some of the body’s survival methods also produce symptoms commonly called diseases. An example is eczema, an emergency expulsion of acid toxins via the skin.

The aforementioned causal (alarming) situation, or modification of the medium, is chronic acidification (pH imbalance) and oxygen deprivation in the blood and tissues due to acid-forming foods, adverse lifestyle, emotional stress, and environmental stress. This is not oversimplification. Acidification/hypoxia biochemically signals a dead host to the microzymas, while creating collapsed areas (dead zones) of the colloidal system in the intercellular fluid, and it is the primary physiological disease condition out of which the symptoms commonly called specific diseases arise.

Thus, we distinguish between this disease condition and its consequent symptoms, which include both the morbidly evolved microzymas and the physiological signs commonly, thought of as specific diseases. As they develop, microforms (bacteria, yeast, fungus and mold) are actually scavenging forms of the microzyma, developed when disease in the cell life requires tissue to be broken up. These upper development forms are the ones easily visible in the blood before physical symptoms arise. They disappear (devolve) when the recycling task is complete, once again becoming microzymas of the earth and/or air.

Virus or toxin?

Regarding the early period of virus isolation, a question is whether the unseen entities isolated in filtered fluids were accompanied by the waste products (mycotoxins) of fermentation by yeast and fungus of cellular elements, such as DNA. If virus filtrates are injected into a host to prove virulence, it is almost certain that easily filterable molecular toxins will be introduced as well. Could Dr. Stanley’s “pure crystals of tobacco mosaic virus” have been crystallized toxins? If so, they would certainly be highly symptomatic, as are exotoxins at the intermediate stage of the cycle, for example. However, it is not proof of anything that you can create illness by poison injection, except proof of that tautological fact.

In my research utilizing dark-field and phase-contrast microscopy, it is common to see crystallizations in the blood. It is normal for the body to use calcium or other mineral salts, and fats as well, to chelate the waste products from the morbid fermentation of body proteins, fats and sugars. Such crystal deposits are found in cancer tissue as well. A malignant tumor removed from the breast of one of my research clients was found to have numerous calcium deposits attached to it. It is an attempt to render inactive the substances that make our inner streams filthy, poison our cells, and coagulate colloidal systems in blood and intercellular fluid.

The term “virus” is the Latin word for poison, and gives us insight into the immediate cause of disease symptoms—poisons: mycotoxins, endotoxins, exotoxins, and toxins from environmental sources (many of which are primary or secondary mycotoxins). Orthodox medicine is well aware that it is bacterial toxins more than the bacteria themselves (they feed in us), that cause the symptoms referred to as infectious disease. Little if any emphasis is placed on this fine but important distinction. Always, the germ is emphasized. There is little to no awareness (or acknowledgment), either, of the same role played by toxins of the culminate microforms of the pleomorphic cycle. Their action and the body’s response to them are frequently ascribed to viruses, which do not produce toxins but are said to wreak havoc by a number of other means. However, if they participate in symptomogenesis in a host it is because they are stimulated to evolve into more complex, toxigenic forms. Somewhat less likely is the possibility that they cause damage as a result of erroneous construction or function, for one reason or another—missing mineral nutrients leading to enzyme deficiencies, for example.

Misconception breeds contempt

In addition to chemical toxicity, however, what is the impact of the fear (emotional toxicity) that the word “virus” brings to mind and heart? It has been said that fear is the most deadly of disease conditions. If a “disease” kills one person, the fear of it may kill twenty. General prejudice concerning the danger of viruses is fundamental biological error based on Louis Pasteur’s germ theory, and is itself a perpetrator of auto-suggested illness. For example, in Africa doctors attribute some AIDS sickness to “voodoo death” syndrome, the term for illnesses induced psychologically. According to one nurse, “We had people who were symptomatically AIDS patients. They were dying of AIDS, but when they were tested and found out they were negative they suddenly rebounded and are now perfectly healthy”.⁵ Ironically, if the germ theory were founded on facts it would be correct to fear viruses, except there would be few, if any, humans living to discuss the issues. These so called pathogenic entities are to researchers, medical practitioners and the press what criminals are to detectives—the focus and justification of their existence.

The encyclopaedia britannica has this to say about bacteria, which relates also to viruses

The common idea of bacteria in the minds of most people is that of a hidden and sinister scourge lying in wait for mankind. This popular conception is born of the fact that attention was first focused upon bacteria through the discovery, some 70 years ago, of the relationship of bacteria to disease in man, and that in its infancy the study of bacteriology was a branch of medical science. Relatively few people assign to bacteria the important position in the world of living things that they rightly occupy, for it is only a few of the bacteria known today that have developed in such a way that they can live in the human body, and for every one of this kind, there are scores of others which are perfectly harmless and far from being regarded as the enemies of mankind, must be numbered among his best friends.

It is in fact no exaggeration to say that upon the activities of bacteria the very existence of man depends; indeed, without bacteria there could be no other living thing in the world; for every animal and plant owes its existence to the fertility of the soil, and this in turn depends upon the activity of the micro-organisms which inhabit the soil in almost inconceivable numbers. It is one of the main objects of this article to show how true is this statement; there will be found in it only passing reference to the organisms which produce disease in man and animals- for information on these see Pathology and Immunity.

The general message of the foregoing article applies even more aptly to viruses in the sense that much fear has been bred and cultivated around them, although they never produce disease symptoms, whereas some bacteria do. The writer of the above understands bacteria, with the exceptions that symptogenic bacteria found in man and animals do not produce disease (only secondary symptoms), that their precursors are endogenous to higher organisms, and they have not "developed in such a way that they can live in the human body." If anything, the reverse is true. According to one theory of microbiology, microforms have colonized over eons to become higher organisms. In one sense, then, the human body has developed as a specialized environment for them.

An important dimension of the bacterial dependence of higher life forms is the floral population in the human digestive tract. Literally, these "foreign species" keep us alive.

Most bacteria have the same underlying function, whether found in soil, sewage, in the human digestive tract, or elsewhere in nature: they are an essential part of the life processes of higher organisms. They will not or cannot attack healthy cells or tissues, but certain ones will recycle sick or dead tissue in much the same way insect pests are drawn to weaker plants. As Bechamp said, "Nothing is the prey of death; all things are the prey of life."

Following in the wake of misconceptions arising from the fundamental biological error known as the germ theory of disease, defining the filtrates of diseased tissue as a newly discovered infectious microform was the birth of a major corollary error in bioscience.

Viral behaviour reconsidered

Listed below are ways viruses are said to disrupt or destroy host cells according to orthodox medical science and the germ theory. Following each in italics is a different interpretation following from microzymian principle:

1. Viral proteins insert into the host cell's plasma membrane and directly damage its integrity to promote cell fusion (HIV, measles, and herpes viruses).

Proteins are attempting to repair membrane damage, or enter cells to make other repairs. There is the question as to whether viruses on cell walls are coming or going. In both cases it would be a matter of whether or not a cell has been disturbed by excess fermentation and acidity. But in the former case the cell would be dysfunctional before attachment occurs, thus requiring the repair complex. Another possibility, perhaps remote, is that dysfunctional receptors on cells are in need of repair, or they are covered by these complexes to inactivate malfunctioning cells. Positive electrical charges in a compromised terrain, primarily on acid molecules from fermentations, discharge cell membranes and act as mortar to stick cells together.

2. Viruses inhibit host cell DNA, RNA, or protein synthesis. For example, poliovirus inactivates cap-binding protein, which is essential for protein synthesis directed by capped host cell mRNAs, while allowing protein synthesis from uncapped poliovirus mRNAs.

Protein inactivation is probably being done by fermentation or by acidic toxins from fermentation, while "poliovirus" is produced in the cell to reverse the damage.

3. Viruses replicate efficiently and lyse host cells, e.g., liver cells by yellow fever, and neurons by *polio virus*.

Highly unlikely. The lysing is more likely caused by acid mycotoxicosis, or by free radicals (ROTS) released in response to mycotoxic stress, or from other sources (ionizing radiation, for example). Repair particles are residual after cell wall disruption.

4. Slow-virus infections (eg. sub acute sclerosing panencephalitis, caused by the measles virus) culminate in severe progressive diseases after a long latency period.

How is this demonstrated? Perhaps "latency" is a period of successful or attempted repair that eventually falters. Symptomology naturally appears in the weakest parts of the body. Excess acidity is always a systemic problem that localizes, just as cancer is a systemic condition that localizes, even though its symptogenic influence may later spread.

5. Viral antigen proteins on the surface of the host cells are recognized by the immune system, and the host lymphocytes attack the virus-infected cells (e.g., liver cells infected with hepatitis B).

Liver cells are damaged beyond repair by mycotoxicosis, and the immune system, our elaborate janitorial service, is cleaning up the garbage. Perhaps the repair protein antigen is expressed to signal immune response (because the cell is beyond repair), which is one explanation for why there are antibodies to these proteins.

6. Viruses damage cells involved in host antimicrobial defense, leading to secondary infections.

The function of immune cells is damaged by fungal infestation and/or overwork by toxic overload, preventing proper cleanup and elimination of disharmonious, symptogenic elements.

7. Viral killing of one cell type causes the death of other cells that depend on them, e.g., degeneration of muscle cells enervated by the attack of poliovirus on motor neurons.

Once again, a misinterpretation and lack of understanding that it is not viral microforms that damage neurons. Toxins from bacteria, yeast, fungus and mold-as well as the fermentation of glucose, proteins, hormones and fats-produce, or influence the body to produce, disease symptoms. Not recognizing the "virus," for what it is, observers attribute disease to it.

8. Host cell responses to viruses include metabolic derangements and transformations resulting in neoplastic changes.

Metabolic derangement has occurred prior to the appearance of repair proteins, due to toxic overload in the cell. It is more likely that the proteins attempt to prevent cell transformation, and that cancerous development is cell conversion from primarily oxidative to wholly fermentative metabolism, mediated by fungus and mold.

Listed below are further orthodox views regarding virus replication, etc., with alternative interpretations in italics

9. According to orthodox theory, viruses enter a host cell and replicate at the host's expense. Replication is accomplished using enzymes which are distinct for each virus family. For example, RNA polymerase is used by negative-stranded RNA viruses to generate positive-stranded mRNA, whereas reverse transcriptase is used by retroviruses to generate DNA from their RNA template and to integrate that DNA into the host genome.

It is normal for repair proteins to generate enzymes to do their work

10. One reason suggested for viral tropism (the tendency to infect some cells but not others) is the presence or absence of host cell receptors that allow the virus to attach. It is said, for example, that HIV binds to the protein (CD4) involved with antigen presentation on helper T-lymphocytes, that Epstein-Barr virus binds to the complement receptor (CD2) on macrophages, that rabies virus binds to the acetylcholine receptor on neurons, and that rhinoviruses bind to the adhesion protein (ICAM-1) on mucosal cells.

Theoretically, once attached, the entire virion, or a portion containing the genome and essential polymerases, penetrates into the cell cytoplasm in one of three ways: (1) Translocation of the entire virus across the plasma membrane; (2) receptor-mediated endocytosis of the virus and fusion with endosomal membranes; or (3) fusion of the viral envelope with the cell membrane. Theory suggests that within the cell the virus uncoats, separating its genome from its structural components and losing its infectivity before replication. In either the nucleus or cytoplasm, newly synthesized viral genomes and capsid proteins are assembled into progeny virions, which may then bud through the plasma membrane. Unencapsulated viruses may be released also, directly through the membrane.

It is interesting, however, that viruses can somehow choose the "infection" to be abortive, latent or persistent, meaning respectively: (1) viral infections with incomplete replication cycles; (2) persisting in a cryptic state, like herpes zoster within a dorsal root ganglion, which suddenly becomes active to produce shingles; (3) continuously synthesized virions, with or without altered cell function (eg. hepatitis B). These three ideas, especially latency, have arisen as feeble excuses for the untenable virus theory.

9. In order for viruses to reproduce, they must complete the following four steps:
- Adsorption and penetration of a cell. The viral particle binds to the host cell membrane. This is usually a specific interaction in which a viral encoded protein on the capsid or a glycoprotein embedded in the virion envelope binds to a host cell membrane receptor and is then internalized. This internalization occurs by endocytosis or by fusion of the virion envelope with the host cell membrane.

b. This is the mechanism whereby the viral particle enters the cell for the purposes of carrying out repairs to the damaged DNA or RNA.

- Uncoating of the virus, so that the nucleic acid can be released from the capsid into the nucleus or cytoplasm.

Repair work may require uncoating. An uncoated "virus" in the cytoplasm may have come from the nucleus and not yet have a coat, as in the case of hepatitis B according to med science. A coat is then created to protect the nucleic acid, to make a communicative or responsive protein complex, or to allow exiting the cell for remote function or for neutralization and recycling by the immune system.

- Synthesis and assembly of viral products as well as inhibition of the host cell's own DNA, RNA and protein synthesis.

Protein complexes produced in response to an alarming situation-fermentative and mycotoxic stress-are capable of self-ordered replication. As suggested by Bechamp, the microzyma is specific for each organ, therefore specific repair proteins will be needed for specific cells that make up specific organs that are being disturbed. There is the question of why the great numbers in some cases. One possibility is simply overreaction; for example, fever can be extreme.

- And finally, release of virions from the host cell either by budding or lysis.

- Complexes leave the cell for remote function or to be neutralized;
- Repairs have failed, and complexes are released prior to or during the breakdown of the cell by acid toxins or the immune system.

Further considerations

Virologists refer to certain microforms as passenger viruses, which are present in asymptomatic situations, riding on their host's genetic molecule like a passenger. To the conventional mind searching for new diseases or for a viral cause of unexplained ones, they are most interesting, because the status of virologists in the scientific community depends upon the pathogenic potential of the viruses they study. Due to their location, passenger viruses are thought to have much disease potential, thus their true function goes unnoticed. These colloidal passengers are the silent majority of animal and human intranuclear proteins essential for genetic repair.

Kalokerinos and Dettman quote Dr. Fred Klenner regarding the changeability of viruses: "I am of the opinion that virus units have the potential of going from one type to another by altering their protein coat. We see chicken pox at Thanksgiving, mumps at Christmas, red measles in the spring, and polio and Coxsackie in the summer".⁶ Seasonal appearance of different forms may be mediated by variations of imbalance in the biological terrain or nutritive medium due to the fermentation of dietary excesses such as sugar and animal proteins that accompany holidays and seasons, calling for different repair proteins. For example, outbreaks of polio have been associated with sugar consumption in summer. Various psycho-emotional stresses correspond to these seasons as well.

Supporting the general idea of dietary culpability is a statement published by the great English physician, Sir Robert McCarrison in 1936: "Obsessed with the invisible microbe, virus, protozoa as all important excitants of disease, subservient to laboratory methods of diagnosis, hidebound by our system of nomenclature, we often forget the most fundamental of all rules for the physician, that the right kind of food (nutrition) is the most important single factor in the promotion of health and the wrong kind of food the most important single factor in the promotion of disease"⁷

Six years before Bechamp identified the microzyma as a ferment and, with his devoted associate, Professor Estor, began a 13-year odyssey of research into its nature, Florence Nightingale published a statement about the germ theory. In *Notes on Nursing*, 1st ed., 1860, she said of infection:

“Diseases are not individuals arranged in classes, like cats and dogs, but conditions growing out of one another.

Is it not living in a continual mistake to look upon diseases, as we do now, as separate entities, which *must* exist, like cats and dogs, instead of looking upon them as conditions, like a dirty and a clean condition, and just as much under our own control; or rather, as the reactions of kindly Nature against the conditions in which we have placed ourselves?

I was brought up ... distinctly to believe that smallpox, for instance, was a thing of which there was once a first specimen in the world, which went on propagating itself in a perpetual chain of descent, just as much as that there was a first dog, (or a first pair of dogs), and that smallpox would not begin itself any more than a new dog would begin without their having been a parent dog.

Since then I have seen with my eyes and smelt with my nose smallpox growing up in first specimens, either in close rooms or in overcrowded wards, where it could not by any possibility have been “caught,” but must have begun. Nay, more, I have seen diseases begin, grow up, and pass into one another. ... I have seen; for instance, with a little overcrowding, continued fever grow up; and with a little more, typhoid fever; and with a little more, typhus, and all in the same ward or hut.

Would it not be far better, truer, and more practical, if we looked upon disease in this light? For diseases, as all experience shows are adjectives, not noun-substantives.

That is, symptoms (called diseases) are *describers* of a situation.”

I find legitimate Bechamp’s conclusion that what are called germs of the air are fundamentally microzymas of beings which are being consumed by the recycling process, i.e., some kind of vegetative digestion-putrefaction or fermentation. In short, there are no pre-existing disease-germ species. The principles of microbial medicine constitute a fundamental biological error. As Bechamp said, “The microbial doctrine is the greatest scientific silliness of this age.” This is not to say that there is no transmission, only that invasion is not necessary for symptomogenesis, nor is it the primary mechanism for illness. It is to say that for transmission to take place, susceptibility in the form of a compromised terrain must pre-exist in the receiver, who is then likely to be ill anyway. With the exception of the immune component in the mucosal barrier, primary host “resistance” is a function of terrain condition rather than immunity *per se*.

Phantom viruses hepatitis

Hepatitis can be a painful symptom that has yielded profitable virus-hunting opportunities in recent years. Although there are several categories of this disorder, three main varieties of what is called “acute viral hepatitis” exist: Type A (formerly “infectious hepatitis”), Type B (formerly “serum hepatitis”), and hepatitis C (formerly “non-A, non-B”). The corresponding viruses are HAV, HBV, and the non-A, non-B “group,” now called C. Type A is said to be caused by an RNA virus, spread primarily by fecal contamination of water and food, with blood and secretions also possibly being infectious (but it is due to the toxins associated with unsanitary conditions). Hepatitis B, discovered in the ‘60s, is said to be caused by a DNA virus which replicates in

the hepatocyte nucleus and receives its surface coat in the cytoplasm. It is said to be transmitted by transfused blood or blood products, or via common use of needles by intravenous drug users (but it is due primarily to over-acidification from the drugs, especially heroin). The exchange of body fluids into the blood, whether by unsterilized needles, abusive sexual activity, etc., can also play a role over time because of repeated immune stress caused by foreign proteins. Third World babies with poor nutrition and unsanitary conditions around the time of birth are also susceptible.

The third type of hepatitis, discovered in the ‘70s, is found among drug users and alcoholics, and accounts for 80 to 90% of hepatitis caused by blood transfusion. It is thus akin to B type and was at first thought by scientists to be hepatitis B until thorough testing of subjects revealed no virus B-nor A, for that matter. It was thus called “non-A, non-B” hepatitis and thought to be at least two viruses and perhaps more.

In 1987 scientists believed they found a single virus causing the third type, what is known today as the hepatitis C virus. However, what they identified was an antibody they associated with a virus. Now, just as with HIV, they could test patients for antibodies against an elusive or invisible virus. With this new observation, however, new paradoxes confronted the viral hypothesis. Huge numbers of people testing positive for the phantom C virus never developed any symptoms. Hepatitis is truly the result of over-acidification or toxification of the largest filter in the human body by such substances as lactic acid, acetic aldehyde and ethanol-not the disease of a pathological virus. It is interesting to note also that all these hepatitis viruses have incubation periods of 2 to 25 weeks, violating Farr’s Law (see below), yet are not classified as slow viruses. Also, the point at which a “natural invasion” takes place, as opposed to a highly artificial injective one, and thus, how true incubation periods are determined, is another interesting question.

Hantavirus

A recent example of unwarranted panic in American biomedicine was the eminent *hantavirus* of 1994. Presumably it had jumped species, from mouse to man (the American Navaho Indians). However, after supposedly killing a number of people, this phantom virus apparently made peace with the Indians and retired to its mouse reservoir. The virus failed to materialize.⁸ A front-page article in the *San Francisco Chronicle* reported that CDC “epidemiologists across the nation are carefully monitoring the deer mouse population and the level of virus within it.” But all that was left to discover of the former “Navaho flu” by the CDC epidemiologists (shown in their space suits) were healthy mice in the mountains.⁹ The Navaho flu is nothing new to the native Americans and is most likely tied to sanitation, nutrition and lifestyle.

Ebola

In May 1995 the CDC announced the new, threatening *Ebola virus*. The deadly killer virus was expected to leave its hidden reservoir in the rain forests of Africa to claim Europe and the United States. An article in *Time* magazine was peppered with men in space suits and colored electron micrographs of the virus (even though electron microscopes cannot take color pictures). A CDC virologist suggested the virus could leave the rain forest if “we get a virus that is both deadly to man and transmitted in the air.” We are thus asked to fear the image of viruses somehow being launched into the air, perhaps by ejection from a host, and then floating on killer breezes to other lands. A more imaginable scenario was suggested by a European epidemiologist who heads the United Nations AIDS program. Echoing the CDC’s

alarm, he stated, “It’s theoretically feasible that an infected person from Kuwait could go to Kinshasa, get on a plane to New York, fall ill, and present transmission risk there.” But within a month the virus had disappeared in Africa, and not a single Ebola case was reported in the United States or Europe.¹⁰

The World Health Organization announced originally on December 19, 1995 that the *Ebola virus* epidemic that killed 245 people in West Africa was over. (This announcement came again in 2014) All tests on any remaining suspected cases were negative. A somewhat unsettling revelation was that every Ebola outbreak in Africa “is associated to have spread through public hospitals”.¹¹ As it turned out, it was associated with re-used hypodermic needles in these hospitals. Just like *hantavirus*, Ebola vanished, never to be heard from again. Most interesting is that this epidemic, as epidemics will, stopped without vaccines or other drugs. But consider the impact such stories have made upon our minds and on the way we view and understand germs. What’s next in virodrama, the Andromeda Strain?

There is one insidious possibility that must be mentioned in passing. Some mysterious outbreaks of the past have been shown years later to have been man-made. In some cases, government agency has used the public to test releases of organisms and weak biochemical toxins in order to verify, through medical reports, expectations of biowarfare activity. These incidents and the whole story of such behavior is well documented in the book, *A Higher Form of Killing* by Robert Harris and Jeremy Paxman. In this scenario, the cause of such an incident would be constructed officially, or left as a mystery, in order to draw attention away from the truth.

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Conflicts of interest

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References

1. Gaston Naessens. L’Orthobiologie Somatidienne. 1991.
2. Bechamp, Montague R Leveson. Pierre Jacques Antoine. *The Blood and Its Third Anatomical Element*. John Ouseley Ltd., London. 1912. p.205–229.
3. Kalokerinos A, Dettman G. Second Thoughts About Disease: A Controversy and Bechamp Revisited. *Biological Research Institute, Warburton, Victoria, Australia*. 1997;4(1): 9.
4. Hume E Douglas. *Bechamp or Pasteur?* The CW Daniel Co. Ltd., Ashingdon, Rochford, Essex, England. 1923. p.109.
5. Farber C. ut of Africa, Part I. *Spin Magazine*. 1993. p.61–63,86–87.
6. Kalokerinos and Dettman, op. cit., p. 12.
7. Ibid.
8. Denetclaw TH, Denetclaw WFJ. Is “Southwest US mystery disease” caused by hantavirus? *Lancet*. 1994;343:53–54.
9. Russel S. On the Trail of Hantavirus. *San Francisco Chronicle, USA*. 1995.
10. Russel S. Signs that *Ebola virus* Is Fading Away. *San Francisco Chronicle, USA*. 1995
11. Kaiser R. Africa State Hospitals Make Viruses, Not Patients, Feel at Home. *Washington Post*. 1995.