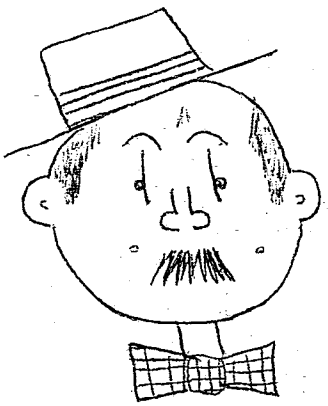
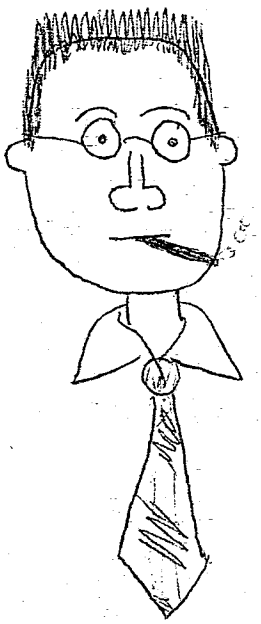
Adaptation of Living Organisms To Their Environment

William Arendshorst Early 1970s





after each of man,s visits to the moon, the returning astronauts and their gear were examined and the men had to go into isolation for a couple of weeks to determine whether any contaminating organisms were returned to earth. None have been found, in fact the periods of isolation have been shortened in the last moon explorations. All of this may have seemed a little too cautious to lay-people but to the professional bacteriologist, it was an unknown and had to be treated as one.

All available evidence on the origin of life points to an evolutionary progression from simple to complex life forms. This is true on Earth and it is progably true throughout the universe. It is also probably true that if any extraterrestrial life form were to infect man it would consist of contact with organisms similar to or identical to earth bacteria or viruses. Three percent of the bacteria living on earth can cause some deleterious effect on man.

I didn't think any more about all of this until I heard of a most secret government sponsored project. The study was to determine what organisms were living in outer space and what possibliities there were that anything from space could be returned to earth. To do this, scoop satellites were shot into space, kept in orbit around earth 3-4 days and returned to earth. The plan was to study 17 of these, changing them as experience dictated. Nothing unusual happened to the first 6 but Scoop VII failed to cpmplete its orbiting and was brought back to earth in a remote area of Arizona. A team that went out to recover Scoop VII found everyone in the small town dead except an old man who drank Sterno and a month old infant who had been nursing (I think). There were some gory details, and there was evidence, found later, that the something that killed in the town had spread to other areas.

The Scoop satellite was recovered and brought to a super super all-out tax-built undergrouhg 5-level laboratory, called Wildfire. Here very

involved studies were carried out by leading professional people in the fields appropriate to the study--Bacteriologists, Mathematicians Technicians and physicians. It was decided that the satellite was knocked out of orbit by a meteorite or a piece of debris from other man-made satellites. Living organisms were isolated from thisrecovered capsule. Consideration was directed to possible sources of these invading bacteria:

(1) They could orginate in another planet or galaxy

(2) They could have left earth for outer space as spores or other dormant forms, eons ago, slowly descended and evolved through environmental stress into unusual forms or

(3) They could have originated from space vehicles after having been changed by enviromental stress factors such as weightlessness oxygen lack, radiation and temperature extremes into unusual forms. The organism had unusual characteristics all right. It killed laboratory animals in seconds, it was air=borne, it grew poorly in oxygen, it grew only in a narrow pH range and it grew poorly in darkness. Under the electron microscope it was a hexagon interlocking with other hexagons but showing variations in the interlocking. It was composed of C, H, O and N but no amino acids were present. The organisms killed by coagulating blood beginning in the lungs but if coagulation were not possible, it injured cerebral blood vessels resulting in massive cerebral hemorrhage. There appeared to be no limitations to its growth and most fantastic of all it could convert matter to energy and energy to matter.

Research on Wildfire went on at a rapid intensive pace but before long there was a leak from one room. To prevent a spread of the contamination, the area was sealed off automatically. In short order other areas were contaminated as rubber and plastic seals developed leaks. It had been pre-arranged that if such an emergency state ever occurred an atomic bomb would automatically arm itself and Wildfire would be

destroyed. However when this was planned, it was untinkable that an organism could interconvert matter and energy. An atomic bomb would make available unlimited energy and this organism would then grow without limitation. But the crisis passed, the bomb was deactivated, the organism mutated to a benign form and when last identified was heading out to outer space. The sources of growth media (food) and environmental stress (too much oxygen) made life as a pathogen on earth too difficult for the visiting microbe.

This is all a little too may out to be real but many science fiction tales such as this have come close to practicality. I would like to use this story to introduce the ways in which organisms can adjust to their surroundings.

When we think along these lines we eventually ask the question, How did it all begin? One of the more satisfactory explanations held that the developement of obviously living organisms was preceeded by a period of chemical pre-biotic evolution, occupying perhaps the first 2 billion of the earth's 4-5 billion years. During this period bodies of water on the surface of the earth accumulated an increasing variety of organic compounds, formed with the aid of such agencies as ultraviolet light, lightning, volcanic heat and inorganic surface catalysts. These substances could accumulate because the primative earth lacked microbial cells and molecular oxygen which in our age would have prevented such accumulation. The thin soups ~~resulting from this organic accumulation are believed to have developed systems that slowly catalyzed their own formation from simpler substrates~~ of simple substrates were catalyzed to more complex structures.

When this went on long enough, there was eventually enough concentration of C, H, O and N that with proper catalyzation, the simplest unit of life--the cell--appeared. It is a unit wherein catalysts bring about replication of the genetic material and synthesis of the surrounding intracellular material. We can't imagine what these first cells looked like. They might have looked much like the cells we see today but

when we consider the tremendous number of possible environmental pressures that have gone on through the ages it is hard to believe that there haven't been changes.

Anton Van Leeuwenhook in the 17th Century first saw and described some one-celled organisms. He called the round ones cocci, the rod-like ones bacilli and the helical ones spirilla. Many bacteria have a cell wall that surrounds the fragile plasma membrane of the cell and protects it from its environment. The rigid wall is characteristic of bacteria, our animal cells only have the membrane. The cell wall is composed of a cellulose-like material and peptides. This outer coat is important because it, and certain substances attached to it, are largely responsible for the virulence of the bacteria. The symptoms of many bacteria can be produced by the injection of their cell walls into animals, and, the same mechansim can be used to immunize animals. Penicillin is a good example of ~~the~~ a mechanism whereby drugs kill bacteria. It interferes with cell-wall material synthesis. The polysaccharide-peptide linkage is blocked from forming disrupted and the cell wall doesn't form. This has to take place during bacterial growth. Obviously organisms that don't have cell walls aren't bothered by this type of drug. Penicillin doesn't affect us because our cells doen't have cell walls.

Bacteria cause disease because of their ability to invade tissue, their ability to produce toxin, or a combination of both. Bacteria that cause disease (pathogens) are conveniently divided into those capable of surviving and even multiplying within the phagocytic cell and those that are promptly destroyed by the phagocyte. When in proper balbnce and the bacteria isn't destroyed, a state of intracelllular parasitism persists and we have a chronic disease such as tuberculosis. Species that are quickly destroyed when phagocytized are diplococcus pneumonia and such a bacteria damages the host only as long as it remains outside the phagocyte (of course). Often the bug that isn't phagocytized and remains an extracellular parasite does so because it has a capsule that has adequate resistance.

In a bacterial cellthe central body or nucleus contains DNA (desoxyribonucleic acid) and the area about the nucleus contains RNA (ribonucleic acid). The nucleus determines the charateristics of future cells in reproduction and the RNA guides the type of protein produced in the cell. DNA in a cell would be duplicated a billion times in perpetuation of a certain species. In this repetetion, abnormalities would be certain to occur and they would be pepeated resulting in a cell with different characteristics. Small fragments of hereditary material called episomes are able to alternate between existing free in the cytoplasm of host cells and existing as integral parts of the host cell's chromosomes. One bacterial episome, called R-factor, carries genes for immunity to one or several drugs. The spread of this factor can create strains of disease bacteria able to resist the best efforts of modern medicine--drugs. Research in this field is constantly going on. The R-factor can duplicate itself just as chromosomal DNA can. An important difference is that R-factor can duplicate itself quickly in ~~bacterial cytoplasm~~ the protoplasm outside the nucleus of the cell and then be transferred to other bacte ria of its own or even unrelated species. This is the basis of the growing problem of ~~infectious~~ drug resistance. Because of this, large numbers of all kinds of bacteria become resistant at a rate much faster than would be possible were resistance the result of a mutation in a chromosomal gene of a single bacterium.

Another form of genetic change==genetic transformation is when genetic material (DNA) as from a virus, penetrates the boundry of a bacterial cell and gets incorporated into the cell's genetic apparatus. Again it is a means by which bacterial cells acquire new genes and thus new traits, with a frequency many orders of magnitude higher than if such changes occurred only through normal random mutation. The invasion of cells by extraneous genetic material takes place in the bacterial and viral world, and perhaps in normal animal cells. An example is the phenomenon wherein a mixture of heat-killed pneumococci of a virulent strain and ~~along with~~ live non-pathogenic pneumococci ~~of strain~~

are injected into mice. The mice die and living virulent bacteria ~~were~~ are isolated at autopsy. The normal harmless saprophytes had been converted into virulent bacteria. A segment of DNA of the living bacteria encoded the property of virulence from the dead pathogens. But viral DNA cannot penetrate all bacteria. ~~T~~The cell wall must have a certain composition or the cell wall must be modified by an activator substance.

I have mentioned some of the changes that can take place with any organism whether it be a regular Darwinian change, or a change forced on the organism by his environment. I would like to mention now changes that have been observed in some of the organisms that are always about us.

Streptococci have a capsule and a specific antigen that impedes phagocytosis. Rapid human passage during an epidemic appears to increase the antigen content of the cell. The streptococcus has changed sufficiently through the years so that 55 strains can now be identified. Some strains could not be ~~typed~~ identified until new techniques were devised recently and there are undoubtedly more waiting to be identified as differentiated ~~more~~ new members are constantly being formed.

Another example of a bacterial adjustment is that of the pneumococcal organism which can grow into an S (capsulated) or an R (unencapsulated) form. Either form will predominant in a culture depending on whether the culture medium is conducive to S or R-form growth but in the absence of an animal, the unencapsulated R-form is killed off by the body cells--phagocytes whereas the capsulated form is protected and grows.

The association between staphyloccus aureus and man is an ancient one. It is an endemic dwellar of our skin and mucosal surfaces and in association with sebaceous gland areas. Therefore ~~many~~ most of us are carriers. Both the host and parasite have evolved a complex array of capabilities for adaptation and defense. It has a true capsule and this enhances its virulence. The organism possesses enzymes--lipase and esterase whhch can dissolve the fatty accumulation

on the skin surface (sebaceous gland sites). It adapts easily in a changing environment. Pathagenic staphylococci can also secrete enzymes--coagulase, fibrinolysin, hyaluronidase which help it invade, spread and establish colonies in animal tissue. It secretes an a-toxin which kills skin cells, breaks down RBC's, causes muscle spasm, and causes clumping of blood platelets. It primarily acts on cell membranes. Another exotoxin is leucociden-- a killer of WBC's. Both a-toxin and leucociden protect S. aureus against the defence system of the WBC's. It can secrete an enterotoxin which is a contaminant in food-- notably starchy foods. Don't eat old warm custard pies.

A component of it's cell surface stops the spread of phagocytes. It is more refractory to intracellular killing in WBC's and macrophages than are other pus-producing cocci. Its genetic compostion can be changed to enable it to adapt more universally. Bacteriophage changes strains of staphylocci when the DNA of the bacteriophage becmes associated with the chromosomes of the bacterial cell, during cell division the new properties are then transmitted to daughter cells. The capacity to elaborate penicillinase and hence resistance to penicillin and other antibiotics may thus be transduced by bacteriophage. But The determinants of penicillinases production and the resistance to antibiotics can be irreversably eliminated by treatment with acriflavone or ultrabiolet light. Strains of staphyloccus resistant to one group of antibiotics can be mixed with strains resistant to another group of antibiotics to give a staph specimen resistant to all these. Thus strains of staphylococcus aureus with multiple resistance are created. another type of resistance is created When the antibiotic has not been of high enough dosage or not used long enough. Then the bacterial cell may mutate and the mutated form then may be resistant to the antibiotic.

appear which exhibit changes in metabolic characteristics ~~including~~ they have become resistante to the antibiotic in question. Substances that form stable complexes withDNA could abolish the frequency of such mutations. Atabrine and phenothiazine do this. Thus atabrine and an antibiotic stopped a urinary infection when previous treatment with ~~an~~ the antibiotic alone failed.

Staphylococcus infections do well in a host who has had trauma, is malnutritioned or whose metabolism is abnormal. The overpopulated areas of the world affer this bug great opportunity.

Staph. aureus does another most interesting thing. When it is grownin the presence of a cell-wall inhibiting drug such as penicillin or methicillin, it may grow as L-forms which are small protoplasmic bodies having reduced or negligible amounts of cell wall substance. The L-form may persist as such or may go back to the parent bacterial form on removal of the inhibiting drug. These L-forms may be reaponsible for recurrent staphylococcal disease. They are resistnat to all antibiotics that act on cell walls but they are susceptable to antibiotics that act on bacterial protoplasm.

, And now that Frendh disease--Syphylis! It is believed that Treponema pallidum has become less virulent through the centuries. This is probably an adaptive response on the part of the organism and man. The organism lives better in a cooler environment and this has led to the ~~thre~~ theory that the disease Yawa in tropical countries is a variant of Syphilis--present in cooler areas..(When you cool it, it gets hotter). The organism and chanacteristics of the 2 diseases are very similar. The organism has become quite resistant to penicillin so that tremendous dosages (15-20 mega units)are now required to obliterate the infection compared to the million or so units that did the job 20 years ago. The old diagnostic tests dependant on antibodies in the patient*s serum are no longer adequate, probably because of man*s adaptation to a less virulent bug. Now the tests depend on the presence of the treponema itself, the florescent

treponemal antibody test which is specific in 95% of the cases.

Mycoplasmas are similar to L-forms of bacteria and may have evolved from them. However mycoplasmas do not convert to any bacterial form as an unstable L-form does. These forms have been isolated from people with urogenital disease, with atypical pneumonia, with arthritis and with pharyngitis and are now thought to cause these diseases by many researchers. They contain DNA and RNA, they grow on lifeless media, they don't change the metabolism of the host cell and don't depend on the host cell for energy. They become established in the host animal when there is a favorable environment for growth. At least 3 species of mycoplasma damage chromosomes of cultured human cells and they are in cancerous tissue. Mycoplasma is absorbed in the RBC and stimulate the host to produce auto antibodies. The body makes antibodies against its own normal cell. These may alter the blood cell's surface and facilitate the invasion of blood cells by viruses. In cancer, the mycoppasma may play a role in allowing viruses to penetrate the cell and in changing the characteristics of the cell.

Probably 60% of all episodes of illness are attributable to viral infections alone. We never read in our history books that in the 16-17 century smallpox and measles viruses killed more Indians than any war with the white man. Eight major groups with 300 different immunological types can cause human infections. The childhood exanthams, Yellow Fever, Smallpox, Influenze, Poliomyelitis are examples Viruses are smaller than bacteria, lack enzymes which function in energy metabolism and are dependent on the host cell for its multiplication. The simplist virus particle has an outer coat of protein and an inner core of nucleic acid. Viral protein is antigenic different from that of thehost cells and causes an immune reaction. Viruses show no evidence of life when they are outside of susceptible host cells but when they gain entrance they cause the host cell to produce new material and to develope properties different from the normal cell. Cells infected with the virus die, change to a different cell or harbor the virus

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without transformation. In an infection leading to cell death, the virus blocks the normal metabolism of the host cell, the cellular archetecture collapses, then the cell dies, and the virus moves on. They can be considered as vehicles for the transmission of infective nucleic acid from one susceptible cell to another. But they may live in harmless equilibrium in one host and bring disease to another host. Black Africans harbor the Yellow Fever virus without getting the disease but white man gets the disease. Through the ages the former developed an immunity.

Spontaneous mutation does occur in animal viruses such as virulent street rabies changing to avirulent fixed rabies, or reduction in virulence of myxoma virus during the course of its life in Australia where it was introduced to control the rabbit population . Mutations have been produced experimentally by attacking the extracellular particle with chemical agents such as nitrous oxide or hydroxylamine. The acquisition of wider host range and enhanced virulence are readily explained by mutations. Injluenze virus exhibites more variation than most and may be more mutable. The poliovirus is more stereo= typed. Perhpas the conditions under which influenza virus survives is greatly different from those to which the poliovirus is subjected The selection of human avirulent viral mutants is of great interest in the field of research in prophylactic immunizing agents. Get them to mutate to an avirulent strain by adapting the virus to a new host, grow that strain and use it in developing immunity to the virulent strain. Often if virulence is developed in a new host through mutation, it will have lost its virulence for the old host. Example is the propagation of measles in tissue culture or in chick embros resulting in a sharp loss in virulence for other hosts. On the contrary other virus may gain virulence by host passage. These are simple examples of how viruses can change when environments are not ideal and this is really a change in the balance of nature. It would be interesting if we could understand how these submicroscopic forms were affected by what we do

to the earth, the streams the lakes and our atmosphere.

Smallpox has changed through the years. The vaccinia we use came from the variola virus by transfer to the cow and adaptation by passage in that host, presumably by selective growth of mutants, or by continuous passage in human skin through variolation, the virus became gradually attenuated for man until it produced the mild disease now characteristic of vaccinia in man.

Poliovirus owed its success as a self-sustaining parasite to the ability of wild strains of the agent to maintain themselves in the human gastro=intestinal tract where they were passed from child to child. It could not be naturally attenuated so Salk did it with formalin.

In general no organism has become more virulent with time unless we consider cancer to be definetly caused by an organism. Virulent strains have spontaneously become weaker because of environmental stress or man has weakened them artificially.

This discussion was not undertaken originally as a means of updating my knowledge of bacteriology although it has but it was an attempt to acquaint you with some information about today*s concepts of micro=organisms. We dont* have Andromeda Strains as far as we know but there is a lot going on that we are just beginning to undefstand The key to cancer is somewhere in immunology and virology. It is always surprising that there is as much regularity in everything living that there is. In the thousands of bacteria in a bottle, or the thousands of cells in a plant, or the multitude of cells in any organ of our body or in the sea of faces watching an olympic event or listening to a tricky dick, One is astounded at the exactness of duplication of each species.