Review Article

The Third Constant

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Accepted on: 23-04-2013; **Finalized on:** 30-06-2013.

ABSTRACT

We know that virus do not have their own metabolism hence no need for food, this means a virus cannot be starved to death. Since the structure of a virus is mainly a DNA/RNA (The Life Particle) in a protein coat, this leads us to understand that just like a virus the DNA/RNA of any cell does not have a requirement for nourishment and thus cannot be starved to death. Microorganisms in their struggle for survival on being deprived of food either face regressive evolution or death due to starvation. Crush an insect under your feet; do all the cells get crushed? Owing to their small size probably none of them gets crushed but the system that feeds the individual cells gets destroyed and the cells are starved to death. Death of a human due to some disease results in failure of some organ and thus the system, resulting into cessation of supplies to the heart and the brain causing death, individual cells however are later starved to death. So we see that in most of the cases the cause of cell death is starving. In this paper we have provided evidences proving that 'The Life Particle' is indestructible, it does not get destroyed by starving, under extreme circumstances or under the effect of antibiotics, antimicrobials or antivirals. Thus we conclude that if life is added to any planet it will remain there forever. Further research is required to find ways to add life to other planets?

Keywords: The Life Particle, Life is indestructible, life once added to a planet will remain there forever, 'Jeev' of Rigveda, rediscovering the Vedas.

STRUCTURE OF A VIRUS

Viruses are small obligate intracellular parasites containing either a DNA or RNA genome surrounded by a protective virus coded protein. A virus does not have its own metabolic system, for propagation it depend upon host cells supplying complex metabolic and biosynthetic machinery of eukariyotic or prokariyotic cell.[1]

THEORY OF EVOLUTION OF VIRUS

There are three classical hypotheses on the origin of viruses: Viruses may have once been small cells that parasitized larger cells (*Theory of regressive evolution or* the *degeneracy hypothesis* or *reduction hypothesis*); some viruses may have evolved from bits of DNA or RNA that "escaped" from the genes of a larger organism (the *vagrancy hypothesis* or *escape hypothesis*); or viruses could have evolved from complex molecules of protein and nucleic acid at the same time as cells first appeared on earth (the *virus-first hypothesis*). [2]

Although none of the above theories fully explain the existence of virus and are still under controversy. Whichever may be the correct theory, but one thing is true:-

- Penultimate step in evolution of virus is formation of or achieving a complete, naked DNA/RNA which may later take up a protein coat.

If a naked DNA/RNA can develop into a virus, there are many other ways in which a complete naked DNA/RNA may be achieved. This means that there may be more than one ways in which virus may be formed.

DEATH OF A MICROORGANISM

Nuclear material of any cell or organism is composed of DNA or RNA, for convenience of understanding herein after I shall refer them jointly as 'LIFE PARTICLE'. By virtue of characteristic features of virus we know that DNA/RNA (THE LIFE PARTICLE) do not have any nutritional requirement. This means starving any organism or any cell will not have any effect on the life particle, only the cell body may be destroyed or face regressive evolution.

The 'LIFE PARTICLE' remains safe and intact in spite of regressive evolution as well as after death of the individual due to starving. Thus we see that what is achieved after regressive evolution is also achieved after death of a micro organism due to starving, i.e. a complete, intact and naked life particle. In the natural struggle for survival cause of death for most of the individuals is starvation. Thus we can presume that under normal circumstances if death occurs the life particle remains safe and intact.[3]

There is another way how we achieve a complete DNA/RNA i.e. through antibiotic, antimicrobial or antiviral action, provided in table 1.

Antibacterial Activity and Mechanism of Action of a Novel Anilinouracil-Fluoroquinolone Hybrid Compound

251D combines the in vitro target specificity of its AU and FQ components, inhibiting both topoisomerase/ gyrase and pol IIIC, and maintains the specificity for inhibition of DNA synthesis and bactericidal mechanism of antibacterial activity of both parents.[4]

Table 1: Mode of action of antibiotics, antibacterials and antiamoebics[3]

Chemical class	Examples	Biological source	Spectrum (effective against)	Mode of action
Beta-lactams (penicillins and cephalosporins)	Penicillin G, Cephalothin	Penicillium notatum and Cephalosporium species	Gram-positive bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Semisynthetic beta-lactams	Ampicillin, Amoxicillin		Gram-positive and Gram-negative bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Clavulanic Acid	Augmentin is clavulanic acid plus Amoxicillin	Streptomyces clavuligerus	Gram-positive and Gram-negative bacteria	Inhibitor of bacterial beta-lactamases
Monobactams	Aztreonam	Chromobacterium violaceum	Gram-positive and Gram-negative bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Carboxypenems	Imipenem	Streptomyces cattleya	Gram-positive and Gram-negative bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Aminoglycosides	Streptomycin	Streptomyces griseus	Gram-positive and Gram-negative bacteria	Inhibits translation (protein synthesis)
	Gentamicin	Micromonospora species	Gram-positive and Gram-negative bacteria esp. Pseudomonas	Inhibits translation (protein synthesis)
Glycopeptides	Vancomycin	Amycolatopsis orientalisNocardia orientalis (formerly designated)	Gram-positive bacteria, esp. Staphylococcus aureus	Inhibits steps in murein (peptidoglycan) biosynthesis and assembly
Lincomycins	Clindamycin	Streptomyces lincolnensis	Gram-positive and Gram-negative bacteria esp. anaerobic Bacteroides	Inhibits translation (protein synthesis)
Macrolides	Erythromycin, Azithromycin	Streptomyces erythreus	Gram-positive bacteria, Gram-negative bacteria not enterics, Neisseria, Legionella, Mycoplasma	Inhibit translation (protein synthesis)
Polypeptides	Polymyxin	Bacillus polymyxa	Gram-negative bacteria	Damages cytoplasmic membranes
	Bacitracin	Bacillus subtilis	Gram-positive bacteria	Inhibits steps in murein (peptidoglycan) biosynthesis and assembly
Polyenes	Amphotericin	Streptomyces nodosus	Fungi (Histoplasma)	Inactivate membranes containing sterols
	Nystatin	Streptomyces noursei	Fungi (Candida)	Inactivate membranes containing sterols
Rifamycins	Rifampicin	Streptomyces mediterranei	Gram-positive and Gram-negative bacteria, Mycobacterium tuberculosis	Inhibits transcription (bacterial RNA polymerase)
Tetracyclines	Tetracycline	Streptomyces species	Gram-positive and Gram-negative bacteria, Rickettsias	Inhibit translation (protein synthesis)
Semisynthetic tetracycline	Doxycycline		Gram-positive and Gram-negative bacteria, Rickettsias Ehrlichia, Borrelia	Inhibit translation (protein synthesis)
Chloramphenicol	Chloramphenicol	Streptomyces venezuelae	Gram-positive and Gram-negative bacteria	Inhibits translation (protein synthesis)
Quinolones	Nalidixic acid	synthetic	Mainly Gram-negative bacteria	Inhibits DNA replication
Fluoroquinolones	Ciprofloxacin	synthetic	Gram-negative and some Gram-positive bacteria (Bacillus anthracis)	Inhibits DNA replication
Growth factor analogs	Sulfanilamide, Gantrisin, Trimethoprim	synthetic	Gram-positive and Gram-negative bacteria	Inhibits folic acid metabolism (anti-folate)
	Isoniazid (INH)	synthetic	Mycobacterium tuberculosis	Inhibits mycolic acid synthesis; analog of pyridoxine (Vit B6)

The mode of antibacterial action of the novel agentZM240304 (D-arabino-2,3,4-tris(4-chlorobenzyl) pentane-l,5-diamine)

ZM240304 is an example of a new class of antibacterial agents that is active against experimental infections in animals. The compound was demonstrated to be a membrane-active compound that disrupted the outer membrane of Gram-negative organisms and allowed the leakage of periplasmic enzymes. Respiration was inhibited and cellular ATP levels were reduced, leading to cell death. The ability of the bacteria to take up small molecules such as amino acids and sugars was inhibited, probably by interference with inner membrane function.[5]

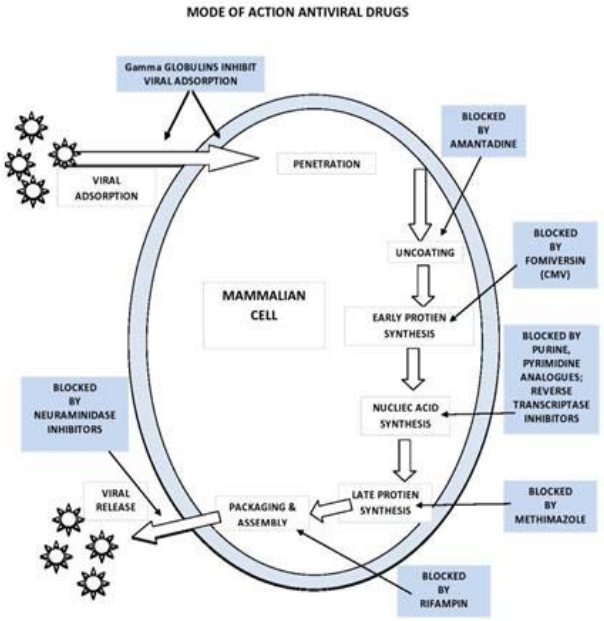
Black Pepper

An injury of membrane function is proposed as the mechanism of action.

MODE OF ACION OF ANTIVIRAL DRUGS[6]

The general idea behind the current strategy in antiviral therapy is to target the viral proteins or parts of proteins that can be disabled. These target proteins should be as unlike any proteins or part of proteins in humans as possible.

Various strategies have been adopted in preparing antiviral drugs which include action at various stages or in different ways:



Before cell entry

One anti-viral strategy is to interfere with the ability of a virus to infiltrate a target cell.

Entry inhibitor

A very early stage of viral infection is viral entry, when the virus attaches to and enters the host cell. A number of "entry-inhibiting" or "entry-blocking" drugs are being developed to fight HIV.

Uncoating inhibitors

Amantadine and rimantadine are uncoating inhibition agents.

During viral synthesis

These drugs target the processes that synthesize virus components after a virus invades a cell.

Reverse transcription

Nucleotide or nucleoside analogues are developed that look like the building blocks of RNA or DNA, but deactivate the enzymes that synthesize the RNA or DNA once the analogue is incorporated. This approach is more commonly associated with the inhibition of reverse transcriptase (RNA to DNA) than with "normal" transcriptase (DNA to RNA).

Integrase

Another target is integrase, which splices the synthesized DNA into the host cell genome.

Transcription

Antivirals are now being designed to block attachment of transcription factors to viral DNA.

Translation/Antisense

These are segments of DNA or RNA that are designed as complementary molecule to critical sections of viral genomes, and the binding of these antisense segments to these target sections blocks the operation of those genomes.

Protein processing and targeting

Interference with post translational modifications or with targeting of viral proteins in the cell is also possible.

Protease Inhibitors

Blocks the enzyme protease to inhibit viral synthesis.

Assembly

Rifampicin acts at the assembly phase.

Release Phase

Zanamivir and oseltamivir act at this stage by blocking the molecule neuraminidase found on the surface of virus.

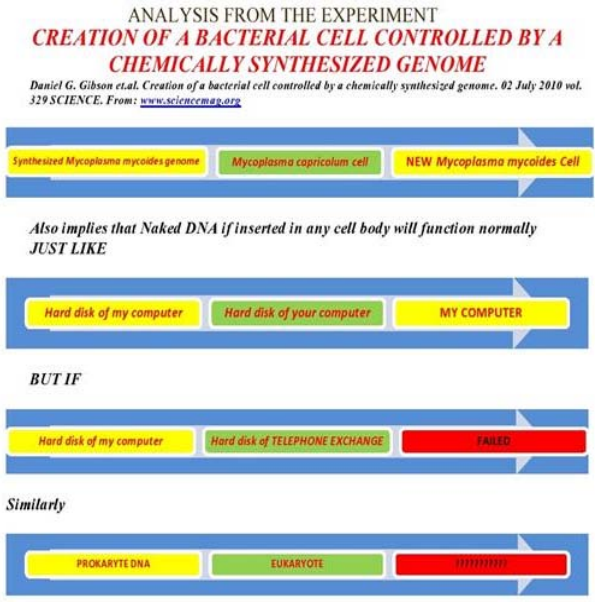
The Analysis

There is a endless and ever increasing list but **any antibiotic, antimicrobial or antivirus that acts against the EXISTING DNA/RNA and destroys it completely is yet to be discovered, designed, formulated or invented**, thus we see that while the micro organism is killed the LIFE PARTICLE is left untouched and unharmed, DNA/ RNA replication or its synthesis may be hampered, but there is no effect on the existing DNA/RNA. Hence what is achieved gradually during regressive evolution or at varying speeds in case of theory of cell origin or theory of parallel origin is also achieved through antimicrbial/antiviral action i.e. an intact & unharmed

DNA/RNA (i.e. the penultimate step in the evolution of virus).

We have never analyzed what happens to the DNA of the microorganism that is killed by antibiotic action within our body. We only presume that it has been eliminated. But what if it is not????

From J. Craig Venter's experiment 'Creation of a bacterial cell controlled by chemically synthesized genome'[7] we can also conclude that a DNA survives even without a body form and if inserted in any body form it will behave like the individual whose DNA it is. It is just like if I insert the hard disk of my computer into your computer, your computer will start behaving like my computer. This is the case when both the computers are compatible, but what if I insert the hard disk of my computer into the server of a telephone exchange? The system will either fail or start behaving erratically. Exactly this is the difference between a prokaryotic cell and a eukaryote. Does this mean that the DNA of microorganism killed by antibiotics may somehow be responsible for the erratic behavior of cancer cells?



The questions thus arise are:-

Q2. The outcome of regressive evolution and effect of antibiotics is the same i.e. an unharmed, intact DNA/RNA is achieved in both the cases then WHAT IS DEATH of the micro organism? (In both the cases the entire body form is lost sparing the LIFE PARTICLE. In one case we believe that it may take a protein coat and become a virus, in the other case we believe that the organism is killed and eliminated and we are cured) do we have evidence for the elimination of DNA/RNA from the body after antibiotic action?

Q3. Can the LIFE PARTICLE achieved through antibiotic action also take a protein coat and become a virus? Or, Can this LIFE PARTICLE **enter into healthy cells of the body and make them cancerous?**

EVIDENCE FROM BEHAVIOUR OF MICROORGANISMS

According to a paper published in Cell Research exogenous plant miRNAs in food can regulate the expression of target genes in mammals.[8] So we can suspect that if miRNA from food can affect the genes then why not DNA/RNA of organisms killed within the body. If we simply look into the characteristic features of a bacterial cell and a human cell and put them into a simple algebraic equation x × y = xy, where x = characteristic features of bacterial cell, y = characteristic features of human cell. We shall find that y will be equal to characteristic features of a cancer cell:

CHARACTERISTIC FEATURES

BACTERIAL CELL = X	HUMAN CELL = Y	CANCER CELL = XY
Continuous multiplication	----------	Loss of regulation of mitotic rate.
Increased requirement of nutrients due to quick multiplication.	----------	Cancer cells have increased glucose and amino acid uptake.
Immortality (with every division weather binary fission or copulation only daughter cells are formed there is no parent cell).	----------	Immortality
----------	Have the capacity to recruit a food supply.	Recruiting a food supply.

It seems that the DNA of the Prokaryote cell attaches to the DNA of the human cell, it may overwrite the instructions so as to induce the properties of the prokaryote into the human cell, it does not have the capacity to mask or hamper the functioning of the human cell hence the human cell continues to perform its functions normally.

This hypothesis needs to be proved and can be conveniently proved if we repeat 'J. Craig Venter's experiment 'Creation of a bacterial cell controlled by chemically synthesized genome' using a human cell instead of Mycoplasma capricollum to insert the copied and synthesized DNA.

EVIDENCES OF IMMORTALITY OF LIFE PARTICLE FROM LIFE OF MICROORGANISMS

Extremophyles

It is believed that extremophyles have survived from the early times of formation of the earth when the conditions were not suitable for other life forms. Thomas Brock in 1966 discovered that many organisms are able to grow in boiling hot sprigs of Yellow Stone National Park. Since then thermophyles have been discovered in varied geothermal features all over the world which include Iceland, Kamchatka Newzealand and many other locations which are otherwise impossible for most other life forms to survive. Some of them can survive in conditions with more than one extreme like high concentration of Sulphur, calcium carbonate, acidic water

or alkaline springs and may be able to face temperatures as high as 140°C.[9] This means that DNA/RNA of these microorganisms continuously multiplied to form new ones while the microorganisms faced life and death. The LIFE PARTICLE is not destroyed even under these circumstances.

Multiplication of microorganisms

Microorganisms multiplying by binary fission or sexual reproduction form another evidence as in these cases all the new cells produced are daughter cells, the existing life particle replicates to form another one taking a new body form every time.

DEATH OF A MULTICELLULAR ORGANISM (Cell death occurs due to starvation)

We know that the body of a multi cellular organism is a collection of cells specialized to perform specific functions. Death of the organism occurs due to failure of one or more organs or systems; all the cells of the body do not face apoptosis at once but are starved after the supply lines fail for example we consider death of a human due to cardiac failure. In this case the circulatory system fails leading to death of the patient, cessation of nutrient and oxygen supply to rest of the cells results into starving and death of the cells, in case of a kidney failure accumulation of toxins in the body may lead to multiple organ failure, but again all the cells of the body do not face apoptosis, they are starved after the cessation of the supply. We know that starving does not affect the life particle.

Q4. Can the destruction of the entire body form of the micro organism, be termed as DEATH of the organism? (Craig Venter has already proved that a DNA/RNA taken out from one organism or synthesized in the laboratory will continue to function in the same way if inserted in another body form).

Not by starving, neither by antibiotics, antimicrobial or antiviral action, nor under extreme circumstances can the LIFE PARTICLE be ever destroyed or is It a reusable chip loaded with 'Information of life'?

We can now conclude that life particle is indestructible and once planted on any planet life will remain there forever; the question however remains are how to plant it in such a way that it thrives.

SWAMI DAYANANDA SARASWATI's Interpretation of RIGVEDA

**According to the Rigveda there are three "ANADI's" Brahma, Prakriti and Jeev. Swami Dayananda Saraswati has defined 'JEEV' as 'Reason for life'. 'ANADI' is a Sanskrit word meaning 'that which has no beginning i.e it is there ever since life initiated'.*

If we look at this in context with the 'Law of Thermodynamics' we can say, Brahma(The abstract power) can be considered to be an equivalent to 'ENERGY', Prakrit(Nature) can be considered to be an equivalent to 'MASS' and THE LIFE PARTICLE may be considered to be an equivalent to 'Jeev'. Swami Dayananda Saraswati explained the 'Jeev' mentioned in the Rgveda as' reason for life'.

It may be considered as reason for life because we know that DNA/RNA is non living but when it enters into a cell body it causes life. This is also proved by the experiment of Daniel G. Gibson et.al[10], outside the body of Mycoplasma capriculum the synthesized DNA was non living but as soon as it was inserted in the body it started to function normally.

Hence'JEEV – THE LIFE PARTICLE' deserves to be the third constant of the law of thermodynamics.

**reference – RIGVEDA mandal 1, Sukta 164.*

SOURCE – 'Satyarth Prakash' by Swami Dayananda Saraswati,56th edition Nov. 2003,Eighth Samullas, Pg. no. 148. Published by AArsh Sahitya Trust, 455, Khari Bawli, Delhi-6. H.O:- 427, naya baans, Delhi-6.

THE CONCLUSIONS

1) DNA/RNA of a virus or any individual cell cannot be starved to death only the cell body gets destroyed or faces regressive evolution.

2) Cause of death for most of the cells is starvation.

3) Human activities may be responsible for evolution of virus.

4) No antibiotic, antimicrobial or antiviral drug can destroy a DNA/RNA.

5) Use of antibiotics may be responsible for carcinogenic transformations in normal human cells.

6) DNA/RNA is the 'THIRD (INDESTRUCTIBLE) CONSTANT' to be added to the law of thermodynamics along with energy and mass.

7) LIFE ONCE ADDED TO A PLANET WILL REMAIN THERE FOREVER.

8) The above findings have the potential for more research to further confirm the above conclusions experimentally.

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Source of Support: Nil, **Conflict of Interest:** None.