Viruses-Challenging Human Intelligence

Author′s Details

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Abstract:

In search of new methods of eradicating all sorts of viruses Synthetic modifications in familiar Antiviral drugs-New Proposals

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Introduction

No one denies if any one says, human brain is the ultimate machine as far as living system is concerned. Right from ice age till now, humans achieved remarkable heights in all fields. No doubt all of us will die on one day or another, yet, we (humans) always try to lead a healthy life and in fact at least few might be thinking about **eternal life style** because such people always want to challenge this nature and want to disprove **supernatural power** i.e **God**. When comes to the term **death**, it may garland every species in different ways and it may be either **clinically dead** or **medically dead** state. Even in alive state, our body contains innumerable number of bacteria (*even viruses too) which will be either useful (*which do not damage our cells) or harmful (*which damage our cells causing any specific type of ailment or even death). The nature itself is an immense treasure of information which is stored in every cell (*right from prokaryote to eukaryote) and each cell has marvelous memory too. This memory is **eternal** and seldom or very rarely fails, if not, how bacteria generate exactly its own mirror image (replica),how a set of chromosomes (from male & female together) unite to generate a human (or even an animal) with all parts at exactly at specified positions! (*few abnormalities occur but very rare). We usually call this memory as **genetic based trait**. Though science has been grown to a level of creating artificial organs, but, yet for that too, we need the **basic platform** i.e **tissue**. So an extraordinary synchronization among various chemical worlds leading to the origin of each type of species itself a wonderful aspect. Though we (humans) achieved many things, yet, few aspects are really challenging us and among those, **viruses** take position-1. The strange aspect is though viruses do not have their own replicating ability, yet, viruses are capable of exploiting all sorts of organisms including humans. The nature is so smart that, it made few species to have **unique** features and that too, with unbelievable power. Almost all of us knew about this joke, “when we take medicine, we can get rid of cold in 7 days and if not, in one week”. Though it seems like a joke, yet, it has an inherent message that, viruses cannot be killed by medicines though controlled. One more astonishing aspect is that, though all living systems have the same five nucleic acids (*genetic material) and the same 20 amino acids (*building a numerous varieties of proteins), yet, all of us (*living things) are not exactly same and every species has certain upper hand over other species which in fact the reason behind our “constant war with viruses, bacteria or else”.

Part-1

There are many questions that point out our power or our ability to take an upper hand over viruses and the most important question is “is it possible to kill virus”? As on today, the answer is no!, but what about future? Few countries are planning to setup colonies even on Moon (*in fact, the planet Mars too) and spending huge amounts on those projects. My small question is “we still do not know 100% of our mother earth, we still do not know 100% of our own”, but, want to rule others (* planets, satellites). Don′t you think that we are mad!? We have sophisticated equipment that can give all information about viruses (*RNA, DNA, Protein structure etc.,), but, still, we do not know how to eradicate viruses! No doubt, few types of viruses were completely (99.999%) disappeared by powerful vaccines (ex: Polio), but however, many types of viruses still existing, evolving and challenging us. I am not against vaccines or their development or am not questioning human abilities, but, I would like to emphasize that “let us fight against viruses like a famous quotation **like charges repel**”. Here the word charge does not necessarily mean the term what we use in physics. My view is “why can-not we fight with viruses with **artificial** (synthetic) viruses”? Though it sounds like vaccines (*which we used to isolate from infected systems) yet, it is totally different. My view is to design an anti-code

to destroy virus but, purely by chemical means. The **synthetic virus** will act as mirror image of native (natural) virus and tries to attract it so as to destroy its **genetic memory** (*RNA or DNA based) i.e after interacting with synthetic virus, natural one fails to identify the its own genetic sequence, so cannot survive even in host cells. Now, question is "shall we be able to synthesize such artificial one" (?). The answer is yes, but, relatively a difficult task. (*even invention of every vaccine too, a greater task, at its initial stages). Actually, my interest is to create a universal artificial virus. This means, it can destroy **genetic memory** of any sort of virus (existing or yet to be evolved!). The following part illustrates the new methods (*purely my personal) which may control viruses, of course the methods need suitable R&D platforms to get approved in future.

Part2

Method-1

Synthesis of a protein based on protein coat of Virus:-

Every microbiologist/virologist/pharmacist/biochemist/biotechnologist knew about those four varieties of protein which all viruses possess, especially the one that protects its surface. So I thought of destroying that envelope by creating a new protein with or without a prosthetic part. Every protein is a complex which consists of a number of polypeptide chains (minimum = 2) that arrange themselves in 3D fashion. Each polypeptide chain contains all 20 varieties of those α-amino acids (Alanine to Valine) in variable number, at variable positions with particular sequence.

I always believe that all viruses are originated from other living systems only because in a virus, mostly it is the m-RNA that interferes with host m-RNA to synthesize a protein of its own. This eventually utilizes the energy content of host cells which often leads to fatalistic conditions (covid-19) So, it must be essential to destabilize this m-RNA of virus or its basic protein envelope. Usually, all proteins are **colloidal type** with definite charge (i.e net charge) and based on their **3D-conformation** often they act as enzymes too. So, if we destabilize this conformation, enzymatic activity will be lost. Or in other way, destabilization of m-RNA by some way also leads to **coagulation** of virus particles. It is very normal aspect that, greater the number of **hydrophobic** based α-AA moieties associated with a polypeptide chain, easier it be to destabilize it by using a suitable electrolyte or other PPC (having more number of **hydrophobic** based α-AA) of opposite charge. This means, a +ve PPC can be coagulated using a –ve PPC or a +ve or a –ve PPC can be coagulated using opposite type with the help of a prosthetic part which also has electrolytic property. For this, I am here with proposing a method which involves the following steps:

Step-1: Identification of correct m-RNA or/and correct protein sequence of virus (native)

Step-2: Dividing this long m-RNA or complex protein into a number of fragments of our interest

Step-3: Start synthesizing new m-RNA which is exactly the **reverse** order of original virus or start synthesizing a new protein whose PPC sequence is exactly **reverse** order of that of original virus.

Step-4: Unification of all individual fragments to get new m-RNA or new protein which I named it as **synthetic virus** (refer part-2).

Step-5: This new synthetic virus must be made as either **iv**-type or capsule type which needs to target virus affected regions. This **iv**-type or capsule type must have a suitable electrolyte (say NaCl/KCl/$CaCl_2$/ anhydrous $ZnCl_2$/anhydrous $CoCl_3$) with proper partition/barrier. It should be borne in mind that, this partition shall break up only at the target.

Illustration: Usually, the specific sequence of α-AA that is adopted by specific virus is purely (*my own view) an accidental only. Some viruses have very lengthy m-RNA chain (so, polypeptide chain too) which may exceed even 1000 or more also. The difficulty in tackling all viruses is this length only because, such a lengthy chain has so much information so, decoding is becoming a great task (*no doubt, present equipment can do it in less time) hence preparation of suitable vaccine too, has become so much hectic and complicated. But, it's now relatively easier to prepare peptide chains of desired length and also desired sequence, which itself is the basis of my proposal. I am here with quoting few α-AA (say 1 to 20 from starting and last 20 towards the end) related to Covid-19*

M-E-S-L-V-P-G-F-N-E-K-T-H-V-Q-L-S-L-P-V [α-AA, 1 to 20]------[**1**]

R-L-I-I-R-E-N-N-R-V-V-I-S-S-D-V-L-V-N-D [α-AA, Last 20]----[**2**]

att-aaa-ggt-tta-tac-ctt-ccc-agg-taa-caa-acc-aac-caa-ctt-tcg-atc-tct-tgt-aga-tct ----[3]

[Gene Sequence, First 60]-DNA

taa-aat-taa-ttt-tag-tag-tgc-tat-ccc-cat-gtg-att-tta-ata-gct-tct-tag-gag-aat-gac ------[4]

[Gene sequence, Last 60]-DNA

auu-aaa-ggu-uua-uac-cuu-ccc-agg-uaa-caa-acc-aac-caa-cuu-ucg-auc-ucu-ugu-aga-ucu -----[5] [m-RNA, First 1 to 20]

uaa-aau-uaa-uuu-uag-uag-ugc-uau-ccc-cau-gug-auu-uua-aua-gcu-ucu-uag-gag-aau- gac ---[6] [m-RNA, Last 20]. The following table [page-3] illustrates [codon & α-AA] relationship.

Table-1

Second letter

First letter	U	C	A	G	
U	UUU UUC Phenyl-alanine; UUA UUG Leucine	UCU UCC UCA UCG Serine	UAU UAC Tyrosine; UAA UAG Stop codon Stop codon	UGU UGC Cysteine; UGA Stop codon; UGG Tryptophan	U C A G
C	CUU CUC CUA CUG Leucine	CCU CCC CCA CCG Proline	CAU CAC Histidine; CAA CAG Glutamine	CGU CGC CGA CGG Arginine	U C A G
A	AUU AUC AUA Isoleucine; AUG Methionine; initiation codon	ACU ACC ACA ACG Threonine	AAU AAC Asparagine; AAA AAG Lysine	AGU AGC Serine; AGA AGG Arginine	U C A G
G	GUU GUC GUA GUG Valine	GCU GCC GCA GCG Alanine	GAU GAC Aspartic acid; GAA GAG Glutamic acid	GGU GGC GGA GGG Glycine	U C A G

Observe one letter codes for all those 20 amino acids:

List-1

[Ala[A], Arg [R], Asn [N], Asp [D], Cys [C], Gln [Q], Glu [E], Gly [G], His [H], Ile [I], Leu [L],Lys [K], Met [M], Phe [F], Pro [P], Ser [S], Thr [T], Trp [W], Tyr [Y], Val [V]]

Now, let us start synthesizing new m-RNA from the reverse direction i.e

m-RNA sequence : cag-**aua**-gag-**gau**-ucu-ucg-aua-uua-uua-gug-uac-ccc-uau-cgu-**gau**-**gau**-uuu-auu-**aua**-**aau** [first 1 to 20 codons in new or synthetic virus]. If at all there exists any stop codon during this reverse process, exchange of two nucleotides will give a new codon that continues the synthesis.

So, first 20 α-AA in this new synthetic polypeptide chain are :

Q-**I**-E-**D**-S-S-I-L-V-Y-R-D-D-P-**D**-**D**-P-I-**I**-**N** ----- **[7]**

[First 1 to 20 α-AA in polypeptide chain in new virus particle = Last 20 α-AA in parent or native virus]

[*note : Bold-Underlined parts show that there exist stop codon on reversing sequence in the native virus, so, exchange of two nucleotides has been done to modified it]

Similarly, the last 20 codons in new virus = first 20 codons in native virus [order reversed]

ucu-aga-ugu-ucu-cua-gcu-uuc-aac-caa-cca-aac-aau-gga-ccc-uuc-cau-auu-uug-aaa-uua

[Last 20 codons in m-RNA], consequently, the last 20 α-AA in new virus are given by:

S-R-C-S-L-A-P-N-Q-P-N-N-G-P-F-H-I-L-K-L -----**[8]**

In this sequence-[8], there exists no stop codon hence no modification has been done.

As most of viruses possess very long polypeptide chain(s) [either one or more than one but in 3D-folding manner], a set of R&D is needed to synthesize new synthetic virus by diving total part into several segments and finally applying recombination principle.

For example, covid-19 which first shown its origin in Wuhan, China has a total of 7156 α-AA moieties which starts with M (methionine) and ends with N (asparagine). A new R&D team having 100 sub-teams (each sub-team = 70 to 80 young scientists) should be framed from all parts of world and each sub-team needs to synthesize a new sub-virus (100 α-AA unit), later up on recombination of all sub-viruses to get this new synthetic virus.

For a trial basis, I would like to request R&D to take just first 20 (m-RNA codons & α-AA) of covid-19 and synthesize new sub-sub-virus part and test whether combination of two results in coagulation of original oligopeptide part or not (refer page-2,sequence-1). Similarly R&D needs to synthesize new sub-sub-virus part taking last 20 (m-RNA codons & α-AA) and test for coagulation process. (refer page-2, sequence-2). In order to facilitate coagulation, new virus protein part may be enriched with Zn^{+2}, Co^{+3}, Mo^{+3}, Mg^{+2}, Ca^{+2}, Cu^{+2} etc., Even using a small portion of new virus enriched with C^{14} too useful to check the rate of coagulation.

The interaction between two oligopeptide chains is shown below:

M-E-S-L-V-P-G-F-N-E-K-T-H-V-Q-L-S-L-P-V [Native Virus, 1-20]----[9]

Q-I-E-D-S-S-I-L-V-Y-R-D-D-P-D-D-P-I-I-N [Synthetic Virus, 1-20] ----[10]

When cofactor (ex: Zn^{+2}) is available, it would try to bind [9] & [10] by coordination thus [9] starts departing from its native chain thus starts coagulating. By using two or more types

of cofactors, it is possible to destabilize parent virus protein (even 1^0 structure may be).
About 914-α-AA sequence is given in table-2 [page-4]

Table-2 [Covid-19]

α-AA Sequence/Covid-19/Wuhan-SARS

```
 translation="MESLVPGFNEKTHVQLSLPVLQVRDVLVRGFGDSVEEVLSEARQ [44]
HLKDGTCGLVEVEKGVLPQLEQPYVFIKRSDARTAPHGHVMVELVAELEGIQYGRSGE [102]
TLGVLVPHVGEIPVAYRKVLLRKNGNKGAGGHSYGADLKSFDLGDELGTDPYEDFQEN [160]
WNTKHSSGVTRELMRELNGGAYTRYVDNNFCGPDGYPLECIKDLLARAGKASCTLSEQ [218]
LDFIDTKRGVYCCREHEHEIAWYTERSEKSYELQTPFEIKLAKKFDTFNGECPNFVFP [276]
LNSIIKTIQPRVEKKKLDGFMGRIRSVYPVASPNECNQMCLSTLMKCDHCGETSWQTG [334]
DFVKATCEFCGTENLTKEGATTCGYLPQNAVVKIYCPACHNSEVGPEHSLAEYHNESG [392]
LKTILRKGGRTIAFGGCVFSYVGCHNKCAYWVPRASANIGCNHTGVVGEGSEGLNDNL [450]
LEILQKEKVNINIVGDFKLNEEIAIILASFSASTSAFVETVKGLDYKAFKQIVESCGN [508]
FKVTKGKAKKGAWNIGEQKSILSPLYAFASEAARVVRSIFSRTLETAQNSVRVLQKAA [566]
ITILDGISQYSLRLIDAMMFTSDLATNNLVVMAYITGGVVQLTSQWLTNIFGTVYEKL [624]
KPVLDWLEEKFKEGVEFLRDGWEIVKFISTCACEIVGGQIVTCAKEIKESVQTFFKLV [682]
NKFLALCADSIIIGGAKLKALNLGETFVTHSKGLYRKCVKSREETGLLMPLKAPKEII [740]
FLEGETLPTEVLTEEVVLKTGDLQPLEQPTSEAVEAPLVGTPVCINGLMLLEIKDTEK [798]
YCALAPNMMVTNNTFTLKGGAPTKVTFGDDTVIEVQGYKSVNITFELDERIDKVLNEK [856]
CSAYTVELGTEVNEFACVVADAVIKTLQPVSELLTPLGIDLDEWSMATYYLFDESGEF [914]
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Method-2

I would like to consider one of the important chelating agents in coordination compounds to act against microbes. But, instead of direct injection (either through iv or capsule) into host body I want to use this agent along with a possible polypeptide chain. What I assume is that, this agent shall interfere with either protein part of virus or metal ions that promote RNA replication which virus does with the help of host cell(s). No doubt, this agent has certain limits according to medical studies. So, we need to use this agent in most suitable way so that it would not affect host cells. The reagent is **EDTA** [**E**thylene **D**iamine **T**etra **A**cetic acid]. Usually, EDTA is used in medicine just to remove toxic substances such as Pb^{+2}, Bi^{+3}, Cd^{+2}, As^{+3} etc., in case of poisoning. However, EDTA may induce other side effects if dosage exceeds certain level. Many transition metal ions (or even inner transition metal ions) readily complex with this reagent and such complex species normally secreted out through urine.

My view regarding EDTA:

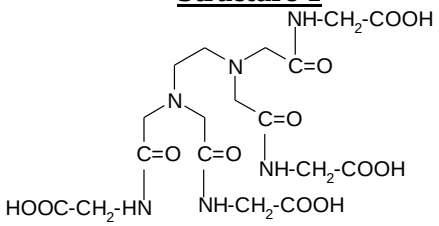
Step-1: EDTA to be used to bind with α-AA to form a new complex like system.
Step-2: This new complex system by name $^{(\alpha AA)}$EDTA interacts with protein envelope of the virus particle thus prevents it to open.
Step-3: $^{(\alpha\text{-}AA)}$EDTA-Virus complex gets coagulated and passes through blood stream, reaches kidney (or Liver) where it gets excreted (or degraded in case of Liver).

Formation of $^{(\alpha\text{-}AA)}$EDTA:

[1] EDTA has four COOH groups (molecular state).
[2] Each COOH group can bind with NH_2 part of α-AA (similar/dissimilar) where it results in the formation of four O=C-NH bonds per EDTA unit.
[3] The free COOH part of each such α-AA moiety starts forming peptide link with other α-AA moiety so that, a long "PPC-EDTA complex" unit is formed.
[4] The two N-atoms in EDTA molecule are capable of coordinating with metal ions like Zn^{+2} based on coordination number (C.N). As C.N = 2 (each N contributes one electron pair), to satisfy C.N = 4 or 6, two or three "PC-EDTA complex" units are needed to get a new type of "^{M}PC-EDTA complex" [M = metal ion, PC = polymeric chain]

Illustrated Example: $(HOOCCH_2)_2$-N-$(CH_2)_2$-N-$(CH_2COOH)_2$ is simplest structural formula of EDTA (molecular state). If this agent interacts with Glycine (say), then, one unit of EDTA consumes 4 units of Glycine forming one $^{(Gly)}$EDTA complex.

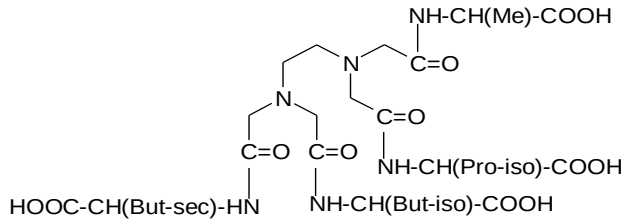
Structure-1



[one single ^{Gly}EDTA complex]

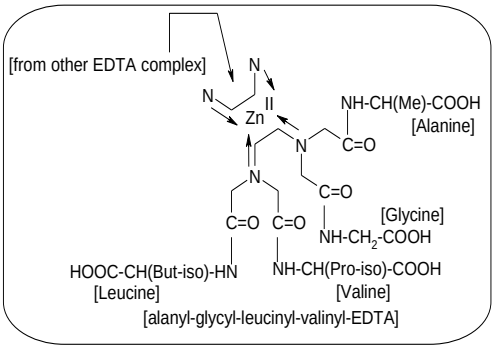
If M = Ag^{+}, then, this single ^{Gly}EDTA itself is enough to satisfy C.N = 2, but, if M = Zn^{+2} or Ni^{+2} or Cu^{+2} or Co^{+2} or Fe^{+2}, then two such ^{Gly}EDTA complex units are needed to satisfy C.N = 4. Owing to steric crowding, C.N = 6 rarely achieved. When EDTA is taken along with any four α-AA in 1:1 (by mole or number of units), EDTA likely to form $^{\alpha\text{-}AA\text{-}4}$EDTA complex where each α-AA likely to interact with one CH_2-COOH of EDTA unit. Like this we can prepare 5 sets of $^{\alpha\text{-}AA\text{-}4}$EDTA complexes using 5 sets of α-AA. Observe the following typical $^{\alpha\text{-}AA\text{-}4}$EDTA (alanyl-isoleucinyl-leucinyl-valinyl-EDTA complex)

Structure-2



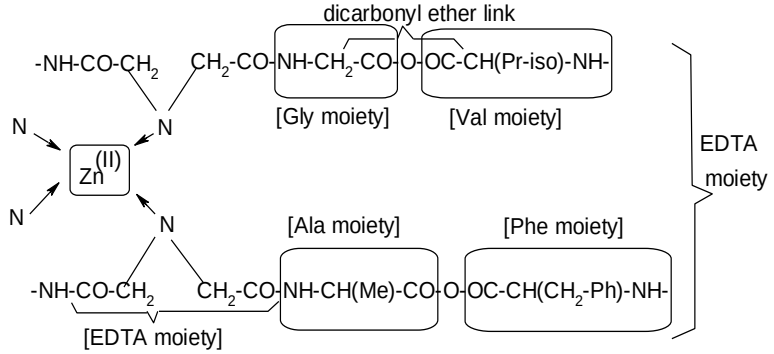
Now, unlike usual polypeptide, here, on mixing all such 5 types of $^{\alpha\text{-}AA\text{-}4}$EDTA complexes, we get a typical complex where two COOH parts of two different α-AA unite through special type of link such as O=C-O-C=O (carbonyl carboxy or dicarbonyl ether). Here, each $^{\alpha\text{-}AA\text{-}4}$EDTA is bound to a metal ion (say Zn^{+2}) and such a complex mixture serves as protecting environment for host cells or forms a layer around protein envelope of virus thus preventing it to interact with host RNA or it delays such a interaction, so that, viral activity will be greatly declined with progress of time. As I observed, most of viruses usually frame their long polypeptide chains with more number of α-AA which are hydrophobic in nature (ex: Alanine, Leucine, Isoleucine, Valine), so, in preparing such a complex $^{5\text{-}\alpha\text{-}AA\text{-}4}_{EDTA}$M, we may increase concentration of $^{\alpha\text{-}AA\text{-}4}$EDTA having hydrophobic α-AA.

Structure-3



[Note: Here, Zn^{II} = Zn^{+2} with four N (from two **$^{\alpha\text{-}AA\text{-}4}$**EDTA complex parts) => C.N = 4, Tetrahedral]

Structure-4



Structure-4 is a smallest part of $^{5\text{-}\alpha\text{-}AA\text{-}4}_{EDTA}$M linear polymeric chain. Usually, it runs parallel to each other which resembles β-pleated like structure of proteins. This long polymeric chain would encircles protein zone of virus particle or virus particles may get trapped between two chains and fails to interact with host RNA. As the polymeric chain mostly contains less hydrophilic parts, so, a virus usually gets destabilized (*as a rule, hydrophilic protein stabilizes hydrophobic type). I made all 20 α-AA into five groups/sets where each set contains 4 α-AA.

Table-3

Group-1	Alanine	Glycine	Isoleucine	Leucine	$^{\alpha\text{-}AA4}_{EDTA}$M-1

Group-2	Phenylalanine	Valine	Serine	Cysteine	$^{\alpha\text{-AA4}}{}_{EDTA}$M-2
Group-3	Aspartame	Glutamine	Arginine	Lysine	$^{\alpha\text{-AA4}}{}_{EDTA}$M-3
Group-4	Aspartic acid	Glutamic acid	Histidine	Tryptophan	$^{\alpha\text{-AA4}}{}_{EDTA}$M-4
Group-5	Methionine	Threonine	Tyrosine	Proline	$^{\alpha\text{-AA4}}{}_{EDTA}$M-5

Since each α-AA moiety in $^{\alpha\text{-AA-4}}{}_{EDTA}$M can interact with 16 types of α-AA from other 4 types of $^{\alpha\text{-AA-4}}{}_{EDTA}$M, based on $^{16}C_4$ principle,

[a] number of permutations = 16!/(16-4)! = (16 x 15 x 14 x 13 x 12!)/(12!) = 43680

[b] number of combinations = (43680)/(4!) = 1,820 [neglecting order and without repetition]

[c] number complex species possible = 64 x 5 = 320 (without repetition)

[d] as we can select any four α-AA from 20 to get $^{\alpha\text{-AA-4}}{}_{EDTA}$M, the number of such complexes thus possible to synthesize = (20 x 19 x 18 x 17 x 16!)/(20 – 4)! = 1,16,280

Method-3

Antibiotics –(α)-AA interaction

Antibiotics are meant for controlling the growth of microbes or even killing those microbes. Unlike viruses, in bacteria, there exists replication (auto/self) even in the absence of host cells. So antibiotics which effectively act on microbes remain dumb when applied on viruses. However, my view is why cannot we combine the two fundamental aspects namely antibiotics and α-AA? Since viruses need proteins (energy source), disturbing RNA (or protein envelope) of viruses using any typical complex synthesized by using both antibiotics and α-AA (or polypeptide chains) together may give a good result. Unlike vaccine development which is time consuming, my method-3 need not require a long time (like method-2) and the results are also can easily be ascertained within short span. My method-3 prefers broad spectrum antibiotics and preferably bactericidal. But, the antibiotic thus selected must have an ability to interact with α-AA (see method-2). Secondly a new antibiotic-amino acid complex thus produced shall not have much side effects, especially on DNA and major organs.

Unlike method-2, in method-3, it is to be decided first whether to use only one single type of antibiotic or a mixture (*preferably having certain resemblance).Few popular (or well-known) antibiotics which I preferred are listed below with their structural formulae:

[a] Aminoglycoside [ex: Tobramycin]with few free NH_2 groups

[b] Ampicillin, Amoxicillin with only one free NH_2 group

[c] Chloramphenicol with only one 2^0 amino group [-NH-]

[d] [R]-Dysidizirine with N in ring system and also a free ester part*

[e] Gentamycin [sub division of (a)] with three free NH_2 groups

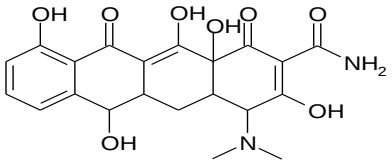
[f] Moxifloxacin with one 2^0 and one 3^0 amino part, but within ring

[g] Prontosil [related to Salvarsan] with two free NH_2 (aromatic)

[h] Streptomycin [resembling (a)] with four free NH_2 groups

[i] Tetracycline with one amide part

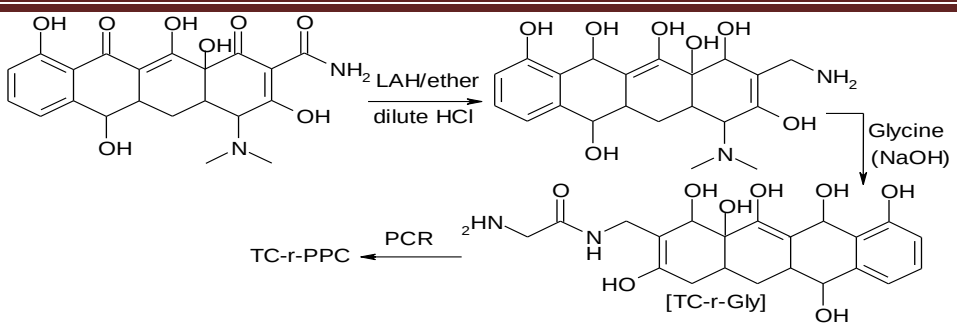
Type-[i] : **Tetracycline**



[i] In tetracycline, there is no free NH_2 part. It is amide [O=C-NH_2] which cannot form any peptide link with α-AA. So, this amide part must be converted into amino part (1^0). This is achieved by reduction with $LiAlH_4$/ether, H^+. But, however this reagent affects C=O part on ring-2 & ring-4 [reading from L to R]

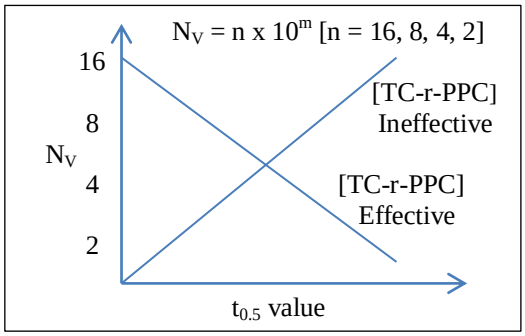
[ii] Once this is completed, we can allow this reduced form of tetracycline to interact with a given α-AA which forms peptide bond with amino part (side chain) attached to ring-4.This complex (new) can be further linked with another α-AA and so on.

[iii] These two steps can be done very easily, but however, formation of a long polypeptide chain may need a suitable m-RNA strand based on type of sequence needed.



Here, TC-r-Gly is a new condensation product from tetracycline (reduced form, TC-r) while PCR leads to a long TC-r-PPC (*polypeptide chain). It is in alkaline medium as sodium salt, so it can be administered as iv-type. Alternatively, TC-r-α-AA complex can be prepared from all known 20 α-AA and such a mixture can be used to treat virus stains. The result is to be analyzed by means of a graph which is plotted between $t_{0.5}$ (half-life period) of virus and number of units of virus that undergo growth or decay.

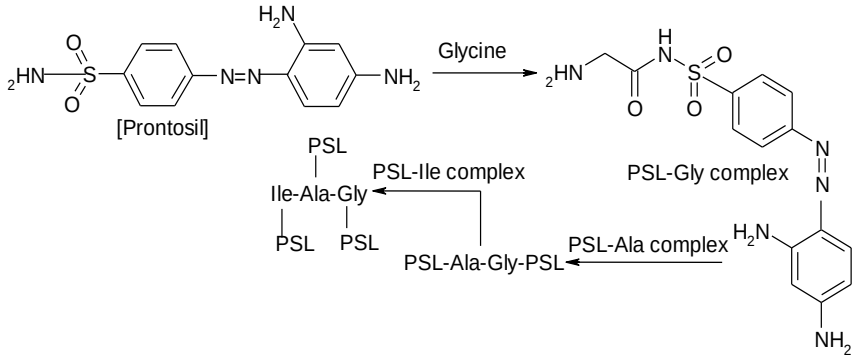
Graph-1



Type-[g] : **Prontosil :- [4-(2,4-diaminophenyl)azo-benzenesulphonamide]**

This antibiotic in fact, a modification of another popular drug by name Salvarsan where the chief part is As=As while in Prontosil, it is N=N (azo part). In prontosil, there exists two 1^0 amino parts attached to aromatic ring in meta position w.r.t each. These two NH_2 groups are weakly basic while NH_2 attached to O=S=O part at para-position is relatively more acidic due to strong polarity from S-atom, so, this group will alone form a peptide bond with α-AA.

Illustration:



This long chain PSL-α-AA complex part when injected into host cell, first it breaks down due to hydrolysis and thus releases N-(α-AA)-BSA [N-α-amino acid based benzene sulphonamide] and 2,4-diaminophenol under pH = 6-7. Thus the long chain N-(α-AA)-BSA acts as protective coat for host cell or envelopes viral particle thus preventing it to open its surfacial protein layer or even it may coagulate viral particle which later disintegrated by anti-bodies or liver cells.

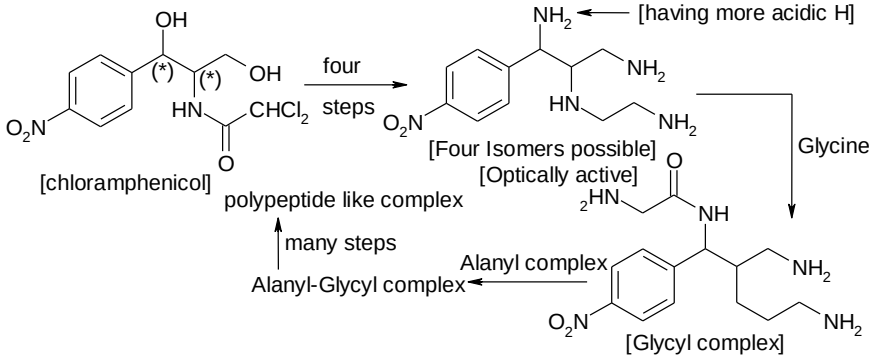
[c] **Chloramphenicol**:-

This antibiotic does not have any free NH_2, instead, there exists 2^0 amino part in contact with powerful electron withdrawing C=O, which in contact with $CHCl_2$ end. There are two OH groups out of which one is 1^0 and other one, 2^0 type. There are two stereogenic centres so, we can expect 2 pairs of enantiomers (two d and two ℓ) and out of these four, whether all show or few show or only one shows antibiotic property is important. This drug itself has one O=C-NH (peptide link) apart from NO_2 at 4^{th} position from 2^0 benzylic alcoholic part. We can generate NH_2 (terminal)

by using $CHCl_2$ part as follows: [G = the entire structural part in contact with $CHCl_2$]

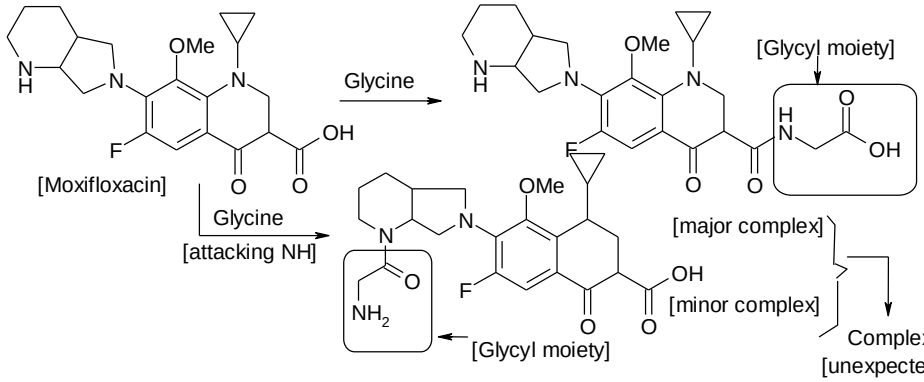
step-1 step-2 step-3
G-$CHCl_2$, $OH^{(-)}$ → G-CHO, $LiAlH_4$/ether, acid → G-CH_2OH, PCl_3 → G-CH_2Cl, alc.NH_3 → G-CH_2-NH_2 (1^0).
step-4

However, this series of reactions disturb other two OH also (step-3) and C=O part of the peptide too gets reduced to 2^0 alcoholic part (step-2). So, there needs a structural change before going to proceed to make other interactions with α-AA.



When injected into host body, there may be breaking of peptide links, however, all individual $CA^{(m)}$-α-AA-complex [(m) = modified, CA = chloramphenicol] units form cluster like system around virus particle thus preventing protein envelope to interact with host m-RNA. Even, the other two 1^0 NH_2 parts disturb protein structure of virus.

[f] **Moxifloxacin**:- It is five ring system out of which three are six membered, one five membered & one, three membered in which three are heterocyclic (N-based). It has one free COOH which itself facilitates peptide link with α-AA.



Here, both major and minor complexes $MFLX^{[\alpha\text{-}AA]}$either unite forming rather complex cyclic part or can interact with other $MFLX^{[\alpha\text{-}AA]}$ (from other α-AA) to give a long polymeric chain. Like stated above, this long polymeric chain interacts with viral protein and binds to it, thus preventing it not to interact with host m-RNA.

A Humble Request!

[i] All these concepts are purely my own views and not copied from elsewhere. As far as my conscious is concerned, I have no knowledge about similar concept whether already tested else (in R&D) or not. [ii] All rights are reserved*[iii] I hope, if any R&D comes forward to make a trial on one or more methods, I hope, we may open new doors to enter new horizons of biotechnology.