Wave Mechanics of HIV/AIDS and the Prediction of Lambda (λ)

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Abstract

The aim of this study is to investigate the dynamic mechanical characteristics of HIV/AIDS in the human blood circulating system. In this work, we developed a constitutive carrier wave equation which we used as a solution to a second-order differential equation. It seems from the results, that the physical dynamic components of the HIV responsible for their destructive tendency are $b\lambda$, $n'\lambda$, $\varepsilon'\lambda$ and $k'\lambda$ with λ as a raising multiplier, whose physical range of interest is $0 \le \lambda \le 19332$. It is established in this study that when the HIV enters the human blood circulating system, it takes about 73 days before its absolute effects would begin to manifest. This study revealed that AIDS actually results when these destructive dynamic components of the HIV gradually become equal to their corresponding active dynamic components in the human blood circulating system. This is when $19234 \le \lambda \le 19332$ and the time it takes the HIV infection to degenerate to AIDS is about 8years (96months). The constitutive carrier wave equation that describes the biological system of man finally goes to zero - a phenomenon called death, when the multiplier approaches the critical value of 19332 and the time it takes to attain this value is about 12 years (144 months).

Key words: latent vibration, carrier wave, 'host wave', 'parasitic wave', raising multiplier, intrinsic parameters.

1.0   Introduction

Some waves in nature behave parasitically when they interfere with another one. Such waves as the name implies has the ability of transforming the initial characteristics and behaviour of the interfered wave to its own form and quality after a given period of time. Under this circumstance, all the active constituents of the interfered wave would have been completely eroded and the resulting wave which is now parasitically monochromatic, will eventually attenuate to zero, since the 'parasitic wave' does not have its own independent parameters for sustaining a continuous existence.

The interference of one wave say 'parasitic wave' y_1 on another one say 'host wave' y_2 could cause the 'host wave' to decay to zero if they are out of phase. The decay process of y_2 can be gradual, over-damped or critically damped depending on the rate in which the amplitude of the host wave is brought to zero. However, the general understanding is that the combination of y_1 and y_2 would first yield a third stage called the resultant wave say y, before the process of decay sets in. In this work, we refer to the resultant wave as the carrier wave and we think this is a better representation.

The role of Human-Immunodeficiency Virus (HIV) in the normal blood circulating system of Man (host) has in general been poorly understood. However, its role in clinical disease has attracted increasing interest. Human immunodeficiency virus (HIV) infection / acquired immunodeficiency syndrome (AIDS) is a disease of the human immune system caused by HIV (Sepkowitz 2001).

During the initial infection a person may experience a brief period of influenza-like illness. This is typically followed by a prolonged period without symptoms. As the illness progresses it interferes more and more with the immune system, making people much more likely to get infections, including opportunistic infections, which do not usually affect people with immune systems (Madel & Dolan 2010). In the absence of specific treatment, around half of the people infected with HIV develop AIDS within ten years and average survival time after infection with HIV is estimated to be 9 to 11 years (UNAIDS & WHO 2007).

According to the literature of clinical diseases, the HIV feeds on and in the process kills the active cells that make up the human immune system. This is a very correct statement but not a unique understanding. There is also a cause (vibration) that gives the HIV its own intrinsic characteristics, activity and existence. It is not the Human system that gives the HIV its life and existence, since the HIV itself is a living organism and with its own peculiar characteristics even before it entered the system of Man.

It is the vibration of the unknown force that causes life and existence. Therefore, for any active biological matter to exist it must possess vibration. The human heart stands as a transducer of this vibration. Fortunately the blood stands as a means of conveying this vibration to all units of the human system.

The cyclic heart contraction generates pulsatile blood flow and latent vibration. The latent vibration is sinusoidal

and central in character, that is, it flows along the middle of the vascular blood vessels. It orients the active particles of the blood and sets them into oscillating motion with a unified frequency as it passes. Elasticity of the vascular blood vessels supports pulsatile blood flow, connectivity network of the blood circulating system, and not the latent vibration.

Man and the Human-Immunodeficiency Virus (HIV) are both active matter, as a result, they must have independent peculiar vibrations in order to exist. It is the vibration of the HIV that interferes with the vibration of Man (host) in the circulating blood system after infection. The interference is destructive and it slows down or makes the biological system of man to malfunction since the intrinsic parameters of the host have been altered.

The activity of the HIV is everywhere the same within the human blood circulating system, mutation if at all does not affect its activity. That the HIV kills slowly with time shows that the wave functions of the HIV and that of the host were initially incoherent. As a result, the amplitude, angular frequency, wave number and the phase angle of the host (Man) which are the basic parameters of vibration were initially greater than those of the HIV.

The human aorta is the main truck of a series of vessels which convey the oxygenated blood from the heart to the tissues (http://education.yahoo.com) . It is described in several portions, viz, the ascending aorta the arch of the aorta, and the descending aorta. The ascending aorta is about 7cm (0.07m) in length and it has a radius of 1.5cm (0.015m). Arch of the Aorta is about 1.8cm (0.018m) in length and its radius is 1cm (0.01m) while the descending aorta has a length of 1.14cm (0.0114m) and a radius of 1.11cm (0.0111m). The human artery is an extension of the aorta and there are various forms with approximate radius of about 0.4cm (0.004m) (Mette *et al*. 2000)

The smallest vessels, the capillaries, have a diameter of about $5x10^{-6}$ m and $10x10^{-6}$ m, so that the red blood cells whose diameter is about $8x10^{-6}$m can pass through it [6]. There are about 250 capillaries/mm³of body tissues and average length of a capillary is about 600 microns ($600x10^{-6}$m).

However, we are going to utilize only the ascending parameters of the aorta in our calculation and assume a uniform geometry and structure for all the vascular blood vessels. This assumption is reasonable since the latent vibration takes its first unique course through the ascending aorta. The human veins are not taken into consideration in our work, because it only conveys denatured blood (deficient in oxygen and food nutrients) to the human heart for reactivation. Also because of the limited length of the human capillaries the exchange process of active blood in this region of space does not take time as results our computation will not include the capillaries as well.

Human blood is a liquid tissue composed of roughly 55% fluid plasma and 45% cells. The three main types of cells in blood are red blood cells, white blood cells and platelets. 92% of blood plasma is composed of water and the other 8% is composed of proteins, metabolites and ions (Cutnel *et al*. 1998). The density of blood plasma is approximately 1025 kg/m³ and the density of blood cells circulating in the blood is approximately 1125 kg/m³. Blood plasma and its contents are known as whole blood (Alexander *et al*. 1998). The average density of whole blood for a human is about 1050 kg/m³. Blood viscosity is a measure of the resistance of blood to flow, which is being deformed by either shear or extensional strain (Glenn 2008). The dynamic viscosity (μ) of the human

blood at 37^0C is usually between $0.003 kgm^{-1}s^{-1}$ and $0.004 kgm^{-1}s^{-1}$, while the arterial blood perfusion rate (

w_b), which is the delivery of arterial blood to a capillary bed in the biological tissue is $0.5 kgm^{-3}s^{-1}$ (Tzu-Ching Shih 2012). The viscosity of blood thus depends on the viscosity of the plasma, in combination with the particles. However, plasma can be considered as a Newtonian fluid, but blood cannot due to the particles which add non-idealities to the fluid.

If a wave is to travel through a medium such as water, air, steel, or a stretched string, it must cause the particles of that medium to oscillate as it passes (David *et al*. 2001). For that to happen, the medium must possess both mass (so that there can be kinetic energy) and elasticity (so that there can be potential energy). Thus, the medium's mass and elasticity property determines how fast the wave can travel in the medium.

The principle of superposition of wave states that if any medium is disturbed simultaneously by a number of disturbances, then the instantaneous displacement will be given by the vector sum of the disturbance which would have been produced by the individual waves separately. Superposition helps in the handling of complicated wave motions. It is applicable to electromagnetic waves and elastic waves in a deformed medium provided Hooke's law is obeyed.

The initial characteristics of a given wave with a definite origin or source can best be determined by the use of a sine wave function. However, for the deductive determination of the initial behaviour of a wave whose origin is not certain, the cosine wave function can best be effectively utilized.

Generally, it is the human blood that responds to the latent vibration from the heart with a specified wave form. The blood then propagates away from the region of the disturbance and in the process circulates oxygen and

food nutrients to nourish the biological cells of the human system. Any alteration to this process results to starvation, a gradual weakening of the fundamental cells and a subsequent breakdown in the entire human biological system if uncontrolled.

1.1   Research Methodology

1. The wave function of a HIV / AIDS free individual is measured (the blood specimen being used as the medium) and the following observations were recorded: (i) the amplitude, a (ii) the phase angle, ε (iii) the angular frequency, n (iv) the wave number, k. These parameters make up the characteristics of the host wave function.

 Note that a, ε and n are assumed to be constant in the human system except for some fluctuating factors, e.g. illness, which of course can only alter them slightly and temporarily.

2. The blood specimen HIV/AIDS infected candidate whose immune count rate is already exactly zero is collected and measured. The following observations were recorded: (i) the amplitude, b (ii) the phase angle, ε' (iii) the angular frequency, n' (iv) the wave number k'. These parameters make up the characteristics of the HIV wave function.

3. Since the immune system of the HIV/AIDS individual is exactly zero, the measured wave function in the asymptotic limit shall depend entirely on the vibration of the HIV only as every other active components of the blood system would have been exterminated.

4. The measured characteristic wave function of the two candidates cannot be the same. Whatever that makes the difference are the attributes of the HIV.

5. The measured wave function of the HIV infected candidate is independent of intrinsic variables such as the number, size, mass and of course the mutation property (if at all) of the invading HIV.

6. The measured characteristic wave function of the HIV infected candidate is the same everywhere within the host. That means, irrespective of the occupation of the HIV in the host system be it in the liver, bone marrow, or in the brain, the activity of the HIV is the same everywhere and hence the wave function must be the same.

7. The wave function of the HIV cannot be directly measured since it does not have definite independent existence outside the host system. As a result, the wave function of the HIV can only be deductively measured within the host system.

8. If the HIV exists it must have a peculiar vibration of its own which must be independent of the vibration of the host. And once the attributes of this vibration are known, then, it can be selectively destroyed from the body of the host by using anti-vibrating components.

9. That the HIV kills slowly with time shows that the wave function of the HIV and that of the man are incoherent. As a result, the amplitude, angular frequency, wave number and the phase angle of vibration of the human system (host) were initially greater than those of the vibration of the HIV (parasite).

Generally, it is the human blood that responds to the latent vibration from the heart with a specified wave form. The blood then propagates away from the region of the disturbance and in the process circulates oxygen and food nutrients to nourish the biological cells of the human system. Any alteration to this process results to starvation, a gradual weakening of the fundamental cells and a subsequent breakdown in the entire human system if uncontrolled.

The aim of this work is to describe the biomechanical behavior of HIV in the human blood vessels and to report the methodology developed in our labouratory to characterize the dynamics of the 'host wave' and those of 'HIV wave' in the constitutive carrier wave equation in the human blood circulating system. Understanding wave propagation in arterial walls, local hemodynamics, and wall shear stress gradient is important in understanding the mechanisms of cardiovascular function. Arterial walls are anisotropic and heterogeneous, composed of layers with different bio-mechanical characteristics which make the understanding of the mechanical influences that arteries contribute to blood flow very difficult (Valenta 1993).

This paper is outlined as follows. Section 1, illustrates the basic concept of the work under study.   The mathematical theory is presented in section 2. We present the results obtained in section 3. While in section 4, we present the analytical discussion of the results obtained. The conclusion of this work is shown in section 5, and this is immediately followed by an appendix and a list of references.

2.0   Mathematical theory of superposition of waves

The initial characteristics of a given wave with a definite origin or source can best be determined by the use of a sine wave function. However, for the deductive determination of the initial behaviour of a wave whose origin is not certain, the cosine wave function can best be effectively utilized. Now, let us consider two incoherent waves that are defined by the non - stationary displacement vectors

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$$y_1 = a\beta \cos(\vec{k}\beta.\vec{r} - n\beta t - \varepsilon\beta)$$

(2.1)

$$y_2 = b\lambda \cos(\vec{k}'\lambda.\vec{r} - n'\lambda t - \varepsilon'\lambda)$$

(2.2)

where all the symbols retain their usual meanings. In this study, (2.1) is regarded as the 'host wave' whose propagation depends on the inbuilt raising multiplier $\beta(= 0 \dots, 1)$. While (2.2) represents a 'parasitic wave' with an inbuilt raising multiplier $\lambda(= 0, 1, 2, \cdots, \lambda_{max})$. The inbuilt multipliers are both dimensionless and as the name implies, they are capable of gradually lowering and raising the basic intrinsic parameters of both waves respectively with time. Let us superpose (2.2) on (2.1), with the hope to realize a common wave function.

$$y = y_1 + y_2 = a\beta \cos(\vec{k}\beta\vec{r} - n\beta t - \varepsilon\beta) + b\lambda \cos(\vec{k}'\lambda\vec{r} - n'\lambda t - \varepsilon'\lambda)$$

(2.3)

Suppose, we assume that for a very small parameter ζ, the below equation holds,

$$n'\lambda = \zeta + n\beta$$

(2.4)

$$y \qquad = a\beta \cos(\vec{k}\beta.\vec{r} - n\beta t - \varepsilon\beta) \qquad + b\lambda \cos(\vec{k}'\lambda.\vec{r} - n\beta t - \zeta t - \varepsilon'\lambda)$$

(2.5)

Again in (2.5), we let

$$\varepsilon'_1 = \zeta t + \varepsilon'\lambda$$

(2.6)

$$y \qquad = a\beta \cos(\vec{k}\beta.\vec{r} - n\beta t - \varepsilon\beta) \qquad + b\lambda \cos(k'\lambda.\vec{r} - n\beta t - \varepsilon'_1)$$

(2.7)

For the purpose of proper grouping we again make the following assumption:

$$\vec{k}\beta.\vec{r} = \vec{k}'\lambda.\vec{r} = \xi$$

(2.8)

$$(\vec{k}\beta - \vec{k}'\lambda).\vec{r} = \xi$$

(2.9)

$$y \qquad = a\beta \cos((\xi - n\beta t) - \varepsilon\beta) \qquad + b\lambda \cos((\xi - n\beta t) - \varepsilon'_1)$$

(2.10)

We can now apply the cosine rule for addition of angles to reevaluate each term in (2.10), that is,

$$\cos(A \pm B) = \cos A \cos B \mp \sin A \sin B$$

(2.11)

$$y = a\beta\{\cos(\xi - n\beta t)\cos\beta\varepsilon + \sin(\xi - n\beta t)\sin\beta\varepsilon\} +$$
$$b\lambda\{\cos(\xi - n\beta t)\cos\varepsilon'_1 + \sin(\xi - n\beta t)\sin\varepsilon'_1\}$$

(2.12)

$$y = a\beta \cos(\xi - n\beta t)\cos\beta\varepsilon + a\beta \sin(\xi - n\beta t)\sin\beta\varepsilon +$$
$$b\lambda \cos(\xi - n\beta t)\cos\varepsilon'_1 + b\lambda \sin(\xi - n\beta t)\sin\varepsilon'_1$$

(2.13)

$$y = \cos(\xi - n\beta t)\{a\beta \cos\beta\varepsilon + b\lambda \cos\varepsilon'_1\} + \qquad\qquad \sin(\xi - n\beta t)\{a\beta \sin\beta\varepsilon + b\beta \sin\varepsilon'_1\}$$

(2.14)

For technicality, let us make the following substitutions so that we can further simplify (2.14).

$$A\cos E = a\beta \cos\beta\varepsilon + b\lambda \cos\varepsilon'_1$$

(2.15)

$$A\sin E = a\beta \sin\beta\varepsilon + b\lambda \sin\varepsilon'_1$$

(2.16)

$$y = A\{\cos(\xi - n\beta t)\cos E + \sin(\xi - n\beta t)\sin E\}$$

(2.17)

$$y = A\cos\{\xi - n\beta t - E\}$$

(2.18)

$$y = A \cos \left\{ (\vec{k}\beta - \vec{k}'\lambda).\vec{r} - n\beta t - E \right\}$$

(2.19)

The simultaneous nature of (2.15) and (2.16) would enable us to square though them and add the resulting equations term by term, that is

$$A^2 \cos^2 E = a^2 \beta^2 \cos^2 \beta\varepsilon + b^2 \lambda^2 \cos^2 \varepsilon_1' + 2ab\beta\lambda \cos \beta\varepsilon \cos \varepsilon_1'$$

(2.20)

$$A^2 \sin^2 E = a^2 \beta^2 \sin^2 \beta\varepsilon + b^2 \lambda^2 \sin^2 \varepsilon_1' + 2ab\beta\lambda \sin \beta\varepsilon \sin \varepsilon_1'$$

(2.21)

$$A^2 = a^2 \beta^2 + b^2 \lambda^2 + 2ab\beta\lambda \left\{ \cos \beta\varepsilon \cos \varepsilon_1' + \sin \beta\varepsilon \sin \varepsilon_1' \right\}$$

(2.22)

$$A^2 = a^2 \beta^2 + b^2 \lambda^2 + 2ab\beta\lambda \cos \left(\beta\varepsilon - \varepsilon_1' \right)$$

(2.23)

$$A^2 = a^2 \beta^2 + b^2 \lambda^2 + 2ab\beta\lambda \cos \left(\beta\varepsilon - (n'\lambda - n\beta)t - \varepsilon'\lambda \right)$$

(2.24)

$$A = \sqrt{a^2 \beta^2 + b^2 \lambda^2 + 2ab\beta\lambda \cos \left((\beta\varepsilon - \varepsilon'\lambda) + (n\beta - n'\lambda)t \right)}$$

(2.25)

$$y = \sqrt{a^2 \beta^2 + b^2 \lambda^2 + 2a\beta b\lambda \cos \left((\beta\varepsilon - \varepsilon'\lambda) + (n\beta - n'\lambda)t \right)} \times \cos \left((\vec{k}\beta - \vec{k}'\lambda).\vec{r} - n\beta t - E \right)$$

(2.26)

Upon dividing (2.16) by (2.15), we get that

$$\tan E = \frac{a\beta \sin \beta\varepsilon + b\lambda \sin \varepsilon_1'}{a\beta \cos \beta\varepsilon + b\lambda \cos \varepsilon_1'}$$

(2.27)

$$E = \tan^{-1} \left(\frac{a\beta \sin \beta\varepsilon + b\lambda \sin (\varepsilon'\lambda - (n\beta - n'\lambda)t)}{a\beta \cos \beta\varepsilon + b\lambda \cos (\varepsilon'\lambda - (n\beta - n'\lambda)t)} \right)$$

(2.28)

Hence (2.26) is the resultant wave equation which describes the superposition of the 'parasitic wave' on the 'host wave'. Equation (2.26) represents a resultant wave equation in which the effects of the constitutive waves are additive in nature. However, suppose the effects of the constitutive waves are subtractive and with the view that the basic parameters of the 'host wave' are constant with time, i.e, $\beta = 1$ and leave its variation for future study, then without loss of dimensionality we can recast (2.26) and (2.28) as

$$y = \sqrt{\left(a^2 - b^2\lambda^2 \right) - 2\left(a - b\lambda \right)^2 \cos \left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda) \right)} \times \qquad \cos \left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - E \right)$$

(2.29)

where we have redefined,

$$A = \sqrt{\left(a^2 - b^2\lambda^2 \right) - 2\left(a - b\lambda \right)^2 \cos \left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda) \right)}$$

(2.30)

$$E = \tan^{-1} \left(\frac{a \sin \varepsilon - b\lambda \sin ((n - n'\lambda)t - \varepsilon'\lambda)}{a \cos \varepsilon - b\lambda \cos ((n - n'\lambda)t - \varepsilon'\lambda)} \right)$$

(2.31)

Equation (2.29) is the constitutive carrier wave equation necessary for our study. It describes the activity and performance of most physically active systems. As the equation stands, it is a 'corrupt wave', in which it is only the variation in the intrinsic parameters of the 'parasitic wave' that determines the life span of the active biological system which it describes. Henceforth, we have agreed in this study, that the initial parameters of the 'host wave' are assumed to be constant and also they are initially greater than those of the 'parasitic wave'. Now by definition: the modulation angular frequency is given by $(n - n'\lambda)$, the modulation propagation constant is

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$(\vec{k} - \vec{k}'\lambda)$, the phase difference δ between the two interfering waves is $(\varepsilon - \varepsilon'\lambda)$, the interference term is given by ; $2(a - b\lambda)^2 \cos((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda))$, while waves out of phase interfere destructively according to $(a - b\lambda)^2$ and waves in-phase interfere constructively according to $(a + b\lambda)^2$. In the regions where the amplitude of the carrier wave is greater than either of the amplitude of the individual wave, we have constructive interference that means the path difference is $(\varepsilon + \varepsilon'\lambda)$, otherwise, it is destructive in which case the path difference is $(\varepsilon - \varepsilon'\lambda)$. If $n \approx n'$, then the average angular frequency say $(n + n')/2$ will be much more greater than the modulation angular frequency say $(n - n')/2$ and once this is achieved, then we will have a slowly varying carrier wave with a rapidly oscillating phase.

2.1   Equation of motion of the carrier wave in the blood vessels of the host

The carrier wave given by (2.29) can only have a maximum value if the spatial oscillating phase is equal to 1. Hence

$$y_m = \sqrt{\left(a^2 - b^2\lambda^2\right) - 2(a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)}$$

(2.32)

$$\frac{d\,y_m}{dt} = (n - n'\lambda)(a - b\lambda)^2 \sin\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right) \times \left((a^2 - b^2\lambda^2) - 2(a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)\right)^{-\frac{1}{2}}$$

(2.33)

$$\frac{d^2 y_m}{dt^2} = (n - n'\lambda)^2(a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right) \times \left((a^2 - b^2\lambda^2) - 2(a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)\right)^{-\frac{1}{2}}$$

$$- (n - n'\lambda)^2(a - b\lambda)^4 \sin^2\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right) \times \left((a^2 - b^2\lambda^2) - 2(a - b\lambda)^2 \cos\left((n - n'\lambda)t - (\varepsilon - \varepsilon'\lambda)\right)\right)^{-\frac{3}{2}}$$

(2.34)

The equation of motion obeyed by the constitutive carrier wave as it propagates along the human blood vessels is affected by two factors. Firstly, the resistance posed by the elasticity of the walls of the blood vessels, secondly, the elastic property of the blood medium where the carrier wave is propagating. The medium's mass and elasticity property determines how fast the wave can travel in the medium. Hence the equation of motion would be partly Newtonian due to the fluidize nature of the blood medium and non-Newtonian due to the particle constituent part of the blood which creates non idealities. We can therefore write the equation of motion as

$$F = -\mu\left(\frac{dy^2}{dt}\right) - \sigma\,y^2 \qquad (2.35)$$

$$\rho V \frac{d^2 y}{dt^2} + 2\mu\,y\left(\frac{dy}{dt}\right) + \sigma\,y^2 = 0$$

(2.36)

Where ρ is the density of the human blood (kgm^{-3}), V is the volume of the blood vessel which is considered to be cylindrical vascular geometry ($\pi r^2 l$) and the unit is (m^3), μ is the dynamic viscosity of blood (kgm^{-1}s^{-1}), and $\sigma = \upsilon w_b$ is the product of the kinematic viscosity of blood (υ) whose unit is (m^{2}s^{-1}) and the arterial blood perfusion (w_b) having a unit of kgm^{-3}s^{-1}, σ is the elasticity of the blood medium. The influence of gravity on the flow of blood is assumed to be negligible. Hence for maximum value of the carrier wave we then rewrite (2.36) as

$$\rho V \frac{d^2 y_m}{dt^2} + 2\mu\, y_m \left(\frac{dy_m}{dt}\right) + \sigma\, y_m^2 = 0$$

(2.37)

Now with the following boundary conditions that at time $t = 0$, $\lambda = 0$, in (2.32) – (2.34)

$$y_m = \left(a^2 - 2a^2 \cos(-\varepsilon)\right)^{\frac{1}{2}}$$

(2.38)

$$y_m^2 = \left(a^2 - 2a^2 \cos(-\varepsilon)\right)$$

(2.39)

$$\frac{dy_m}{dt} = n\, a^2 \sin(-\varepsilon)\left(a^2 - 2a^2 \cos(-\varepsilon)\right)^{-\frac{1}{2}}$$

(2.40)

$$\frac{d^2 y_m}{dt^2} = n^2\, a^2 \cos(-\varepsilon)\left(a^2 - 2a^2 \cos(-\varepsilon)\right)^{-\frac{1}{2}} - n^2\, a^4 \sin^2(-\varepsilon)\left(a^2 - 2a^2 \cos(-\varepsilon)\right)^{-\frac{3}{2}}$$

(2.41)

By substituting (2.38) – (2.41) into (2.37) we get

$$\rho V \left(\frac{n^2 a^2 \cos(-\varepsilon)}{(a^2 - 2a^2 \cos(-\varepsilon))^{1/2}} - \frac{n^2 a^4 \sin^2(-\varepsilon)}{(a^2 - 2a^2 \cos(-\varepsilon))^{3/2}}\right) + 2\mu \left((a^2 - 2a^2 \cos(-\varepsilon))^{1/2} \times \frac{na^2 \sin(-\varepsilon)}{(a^2 - 2a^2 \cos(-\varepsilon))^{1/2}}\right)$$

$$+ \sigma\left(a^2 - 2a^2 \cos(-\varepsilon)\right) = 0$$

(2.42)

$$\rho V \left(\frac{n^2 a \cos(-\varepsilon)}{(1 - 2\cos(-\varepsilon))^{1/2}} - \frac{n^2 a \sin^2(-\varepsilon)}{(1 - 2\cos(-\varepsilon))^{3/2}}\right) + 2\mu \left(na^2 \sin(-\varepsilon)\right) + \sigma\left(a^2(1 - 2\cos(-\varepsilon))\right) = 0$$

(2.43)

To linearize (2.43) we multiply through it by $\left(1 - 2\cos(-\varepsilon)\right)^{\frac{3}{2}}$ so that

$$\rho V \left(n^2 a \cos(-\varepsilon)(1 - 2\cos(-\varepsilon)) - n^2 a \sin^2(-\varepsilon)\right) + 2\mu \left(na^2 \sin(-\varepsilon)(1 - 2\cos(-\varepsilon))^{3/2}\right) + \sigma\left(a^2(1 - 2\cos(-\varepsilon))^{5/2}\right) = 0$$

(2.44)

We are going to utilize two types of approximation to linearize (2.44). The first approximation is the usual binomial expansion of the fractional terms in (2.44) while we stop at the second term and the second type of approximation is the 'third world approximation'. Consequently, upon using the fact that $\cos(-\varepsilon) = \cos\varepsilon$ (even and symmetric function) and $\sin(-\varepsilon) = -\sin\varepsilon$ (odd and screw symmetric function), equation (2.44) yields after the binomial expansion the following result.

$$\rho V\, n^2 a \left(\cos\varepsilon(1 - 2\cos\varepsilon) - \sin^2\varepsilon\right) + 2\mu \left(-na^2 \sin\varepsilon(1 - 3\cos\varepsilon) + \ldots\right) + \sigma\left(a^2(1 - 5\cos\varepsilon) + \ldots\right) = 0$$

(2.45)

$$\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right)\rho V\, n^2 a + \left((1 - 5\cos\varepsilon)\sigma + \left(6\mu n \sin\varepsilon \cos\varepsilon - 2\mu n \sin\varepsilon\right)\right)a^2 = 0$$

(2.46)

$$\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right)\rho V\, n^2 a = 0$$

(2.47)

$$\left((\sigma - 5\sigma\cos\varepsilon) - 2\mu n (\sin\varepsilon - 3\sin\varepsilon\cos\varepsilon)\right)a^2 = 0$$

(2.48)

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2.2 Calculation of the phase angle (ε) of the host wave

It can be gathered from (2.47) and (2.48) by simply equating coefficients of the left side to zero, hence

$$\cos \varepsilon - 2\cos^2 \varepsilon - \sin^2 \varepsilon = 0$$

(2.49)

$$\sin \varepsilon - 3\sin \varepsilon \cos \varepsilon = 0$$

(2.50)

$$1 - 5\cos \varepsilon = 0$$

(2.51)

Let us now solve for the critical value of the phase angle of the 'host wave' by using the relation $\cos \varepsilon = 1 - \dfrac{\varepsilon^2}{2}$

and $\sin \varepsilon = \varepsilon$ in (2.49), (2.50), and (2.51) so that we get respectively

$$\varepsilon^4 - \varepsilon^2 + 2 = 0$$

(2.52)

$$3\varepsilon^2 - 4 = 0$$

(2.53)

$$5\varepsilon^2 - 8 = 0$$

(2.54)

By solving for ε in (2.52) we get

$$\varepsilon_1 = -0.9783 + 0.6761i \quad ; \quad \varepsilon_2 = -0.9783 - 0.6761i \quad ; \quad \varepsilon_3 = 0.9783 + 0.6761i \quad ; \quad \varepsilon_4 = 0.9783 - 0.6761i$$

(2.55)

However, a more realistic complex value of ε is $\varepsilon_3 = 0.9783 + 0.6761i$ and by converting the result from complex variable value to degree we get

$$\tan \varepsilon = \frac{0.6761}{0.9783} = 0.6911 \implies \varepsilon = \tan^{-1}(0.6911) = 35^0 \ (0.6109 \ rad.)$$

(2.56)

Equations (2.53) and (2.54) yield, $\varepsilon = 1.15 \ rad.$ and $\varepsilon = 1.26 \ rad.$, respectively. In this work, we are going to implement $\varepsilon = 0.6109 \ rad.$. The two dimensional nature of ε which is given by (2.56) makes it more a suitable and realistic value for application in this study.

2.3 Calculation of the angular frequency (n) of the 'host wave'

Now the kinematic viscosity of blood is given by the equation

$$\text{Kinematic} \qquad \text{viscosity} \qquad \upsilon = \frac{\mu}{\rho} = \frac{0.004 kgm^{-1}s^{-1}}{1050 kgm^{-3}} = 3.8095 \times 10^{-6} m^2 s^{-1}$$

(2.57)

The elasticity σ of the blood medium is calculated from the arterial blood perfusion (which is a measure of the ability of blood to spread). Thus given that the arterial blood perfusion $w_b = 0.5 \ kg \ m^{-3} s^{-1}$ we can write

$$\sigma = \upsilon \, w_b = 1.9048 \times 10^{-6} \ kg \ m^{-1} s^{-2}$$

(2.58)

$$n = \frac{-(1 - 5\cos \varepsilon) \, \sigma}{(6\sin \varepsilon \cos \varepsilon - 2\sin \varepsilon)\mu}$$

(2.59)

$$n = \frac{-(1 - 5\cos(0.6109)) \, 1.9048 \times 10^{-6} kgm^{-1}s^{-2}}{(6\sin(0.6109)\cos(0.6109) - 2\sin(0.6109))0.004 kgm^{-1}s^{-1}} = 0.000882 \ rad./s$$

(2.60)

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2.4   Calculation of the amplitude (a) of the 'host wave'

The second approximation to (2.44) is the 'third world approximation'. This is the differential minimization of the binomial expansion of a given variable function. It states that for any variable function

$$(1 + \xi f(\phi))^{\pm n} = \frac{d}{d\phi}\left(1 + n\,\xi f(\phi) + \frac{n(n-1)}{2!}(\xi f(\phi))^2 + \frac{n(n-1)(n-2)}{3!}(\xi f(\phi))^3 + \dots\right) - n\frac{d}{d\phi}(\xi f(\phi))$$

(2.61)

Here ϕ is a variable function and ξ is any scalar number. The 'third world approximation' enhances minimum functional value and easy convergence of result. Hence

$$(1 - 2\cos\varepsilon)^{3/2} = (1 + (-2\cos\varepsilon))^{3/2} = (0 + 3\sin\varepsilon - 3\cos\varepsilon\sin\varepsilon + \dots) - 3\sin\varepsilon = -3\cos\varepsilon\sin\varepsilon$$

(2.62)

$$(1 - 2\cos\varepsilon)^{5/2} = (1 + (-2\cos\varepsilon))^{5/2} = (0 + 5\sin\varepsilon - 15\cos\varepsilon\sin\varepsilon + \dots) - 5\sin\varepsilon = -15\cos\varepsilon\sin\varepsilon$$

(2.63)

Substituting (2.62) and (2.63) into (2.44) we get after simplification

$$\rho V n^2 a\left(\cos\varepsilon(1 - 2\cos\varepsilon) - \sin^2\varepsilon\right) + 2\mu\left(na^2\sin\varepsilon(3\cos\varepsilon\sin\varepsilon)\right) + \sigma\left(a^2(-15\cos\varepsilon\sin\varepsilon)\right) = 0$$

(2.64)

$$a = \frac{-\left(\cos\varepsilon - 2\cos^2\varepsilon - \sin^2\varepsilon\right)\rho V n^2}{6\mu n\sin^2\varepsilon\cos\varepsilon - 15\sigma\cos\varepsilon\sin\varepsilon}$$

(2.65)

$$a = \frac{-\left(\cos(0.6109) - 2\cos^2(0.6109) - \sin^2(0.6109)\right)\rho V n^2}{6(0.000882)(0.004)\sin^2(0.6109)\cos(0.6109) - 15(1.9048\times10^{-6})\cos(0.6109)\sin(0.6109)}$$

(2.66)

$$a = \left(-129538.0767\right)\rho V n^2$$

(2.67)

Now for the human Aorta the radius $r = 0.015m$, $l = 0.07m \Rightarrow V = \pi r^2 l = 3.142\times(0.015)^2\times0.07$

$$a = \left(-129538.0767\right)\times1050\times3.142\times(0.015)^2\times0.07\times(0.000882)^2 = 0.00524\,m$$

(2.68)

Also the amplitude of the carrier wave in the human capillary can be calculated from (2.68). Thus for the human capillary whose length is about 600 X10^{-6}m and diameter d= 10X10^{-6} (radius r = 5X10^{-6} m), the approximate value of the amplitude of the 'host wave' in the capillary is $a = 4.99\times10^{-12}\,m$.

2.5   Calculation of the wave number or the spatial frequency (k) of the 'host wave'.

We have made the assumption that for the carrier wave to have a maximum value then the spatial oscillating phase must be equal to 1, as a result

$$\cos\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - E\right) = 1$$

(2.69)

$$\left((\vec{k} - \vec{k}'\lambda).\vec{r} - (n - n'\lambda)t - E\right) = 0$$

(2.70)

$$(\vec{k} - \vec{k}'\lambda) = (k - k'\lambda)_x i + (k - k'\lambda)_y j + (k - k'\lambda)_z k$$

(2.71)

$$\vec{r} = xi + yj + zk$$

(2.72)

If we assume that the motion is constant in the z-direction and the wave vector mode is also the same for both x and y plane in the cylindrical system then

$$(\vec{k} - \vec{k}'\lambda) = (k - k'\lambda)_x i + (k - k'\lambda)_y j$$

(2.73)

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$$\vec{r} = r\cos\theta\, i + r\sin\theta\, j$$

(2.74)

where $\theta = \pi - (\varepsilon - \varepsilon'\lambda)$ is the variable angle between y_1 and y_2 ,please appendix for details.

$$(\vec{k} - \vec{k}'\lambda).\vec{r} = \qquad\qquad = \qquad\qquad (k - k'\lambda)\, r\, (\cos\theta + \sin\theta)$$

(2.75)

$$\big((k - k'\lambda)r(\cos\theta + \sin\theta) - (n - n'\lambda)t - E\big) = 0$$

(2.76)

with the boundary conditions that at time $t = 0$, $\lambda = 0$, $E = \tan^{-1}(\tan\varepsilon) = \varepsilon = 0.6109\, rad.$

$$\theta = \pi - (\varepsilon - \varepsilon'\lambda) = \pi - \varepsilon = 3.142 - 0.6109 = 2.5311\, rad.$$

(2.77)

$$k\, r\big(\cos(2.5311) + \sin(2.5311)\big) - 0.6109\, rad. = 0$$

(2.78)

$$k = \frac{0.6109\, rad.}{r\,(-0.2460945)} = \frac{0.6109\, rad.}{0.015m \times (-0.2460945)} = 166\, rad./m$$

(2.79)

Note that the radius of the human aorta r = 0.015m and also we are working with the absolute value of k .

2.6   Determination of the HIV/AIDS parameters (b , n' , ε' and k')

The gradual deterioration of the biological system of a HIV/ AIDS infected candidate would make us believe that after a sufficiently long period of time all the active constituents of the human system would have been completely eroded by the influence of the HIV, on the basis of this argument the below relation holds

$$\left.\begin{array}{l} a - b\lambda = 0 \;\; \Rightarrow 0.00524 = b\lambda \\[4pt] n - n'\lambda = 0 \;\; \Rightarrow 0.000882 = n'\lambda \\[4pt] \varepsilon - \varepsilon'\lambda = 0 \;\; \Rightarrow 0.6109 = \varepsilon'\lambda \\[4pt] k - k'\lambda = 0 \;\; \Rightarrow 166 = k'\lambda \end{array}\right\}$$

(2.80)

Upon dividing the sets of relations in (2.80) with one another with the view to first eliminate λ we get

$$\left.\begin{array}{l} 5.94104\, n' = b \\[4pt] 0.0085775\varepsilon' = b \\[4pt] 0.000031566k' = b \\[4pt] 0.00144377\varepsilon' = n' \\[4pt] 0.00000531325k' = n' \\[4pt] 0.00368k' = \varepsilon' \end{array}\right\}$$

(2.81)

A more realistic and applicable relation is when: $0.0085775\,\varepsilon' = 0.000031566k'$, from which based on simple ratio

$$\left.\begin{array}{l} \varepsilon' = 0.0000316\, rad. \\[4pt] k' = 0.00858rad./m \\[4pt] n' = 4.56\times10^{-8}\, rad./s \\[4pt] b = 2.71\times10^{-7}\, m \end{array}\right\}$$

(2.82)

Any of these values of the HIV parameters shall produce a corresponding approximate value of lambda $\lambda = 19332$ upon substituting them in (2.80). Note that for the interest of uniformity and anticipated

complications we are using the minimum value which is 19332. Hence; $0 \leq \lambda \leq 19332$. Hence, so far, we have determined the basic intrinsic vibratory characteristics of both the human 'host wave' and those of the HIV 'parasitic wave' both contained in the carrier wave.

2.7 Determination of the attenuation constant (η)

Attenuation is a decay process. It brings about a gradual reduction and weakening in the initial strength of the intrinsic parameters of a given active system. In this study, the parameters are the amplitude (a), phase angle (ε), angular frequency (n) and the spatial frequency (k).

The dimension of the attenuation constant (η) is determined by the system under study. However, in this work, attenuation constant is the relative rate of fractional change (FC) in the basic parameters of the carrier wave function. There are 4 (four) attenuating parameters present in the carrier wave. Now, suppose a, n, ε, k represent the initial intrinsic parameters of the 'host wave' that is present in the carrier wave and $a - b\lambda$, $n - n'\lambda$, $\varepsilon - \varepsilon'\lambda$, $k - k'\lambda$ represent the intrinsic parameters of the 'host wave' that survives after a given time. Hence, the FC is

$$\sigma = \frac{1}{4} \times \left(\left(\frac{a - b\lambda}{a} \right) + \left(\frac{\varepsilon - \varepsilon'\lambda}{\varepsilon} \right) + \left(\frac{n - n'\lambda}{n} \right) + \left(\frac{k - k'\lambda}{k} \right) \right)$$

(2.83)

$$\eta = \frac{FC\big|_{\lambda=i} - FC\big|_{\lambda=i+1}}{unit\ time\ (s)} \qquad = \qquad \frac{\sigma_i - \sigma_{i+1}}{unit\ time\ (s)}$$

(2.84)

The dimension is *per second (s^{-1})*. Thus (2.84) gives $\eta = 0.0000517\,s^{-1}$ for all values of $\lambda\,(i = 0, 1, 2, \ldots, 19332)$.

2.8 Determination of the time (t)

We used the information provided in section 2.7, to compute the various times taken for the carrier wave to decay to zero. The maximum time the carrier wave lasted as a function of the raising multiplier λ is also calculated from the attenuation equation shown by (2.84). The reader should note that we have adopted a slowly varying regular interval for the raising multiplier since this would help to delineate clearly the physical parameter space accessible to our model.

However, it is clear from the calculation that the different attenuating fractional changes contained in the carrier wave are approximately equal to one another. We can now apply the attenuation time equation given below.

$$\sigma = e^{-(2^\gamma \eta t)/\lambda}$$

(2.85)

$$t = -\left(\frac{\lambda}{2^\gamma \eta} \right) \ln \sigma$$

(2.86)

The index γ is the HIV factor. Thus, for HIV $\gamma = 3$, and it is different for any other human diseases that are not localized. The equation is statistical and not a deterministic law. It gives the expected intrinsic parameters of the 'host wave' that survives after time t. Clearly, we used (2.86) to calculate the exact value of the decay time.
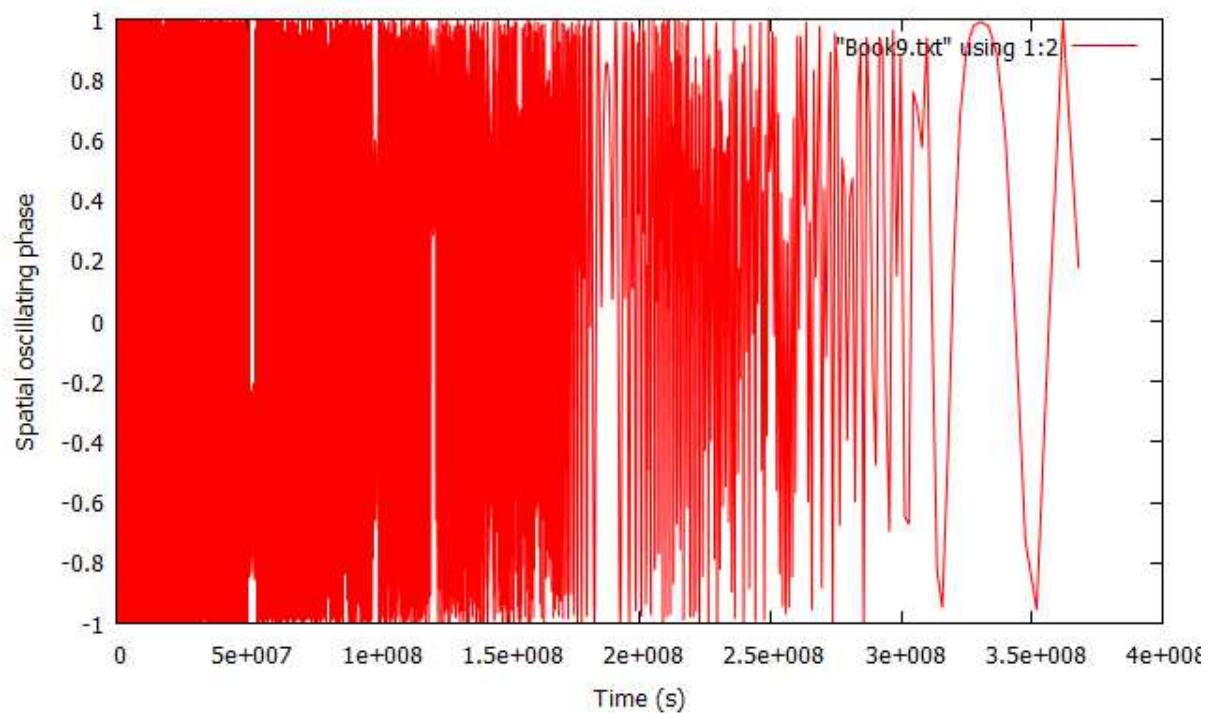
3.0 PRESENTATION OF RESULTS

Fig. 3.1: This **r**epresents the graph of the spatial oscillating phase of the constitutive carrier wave against time t .
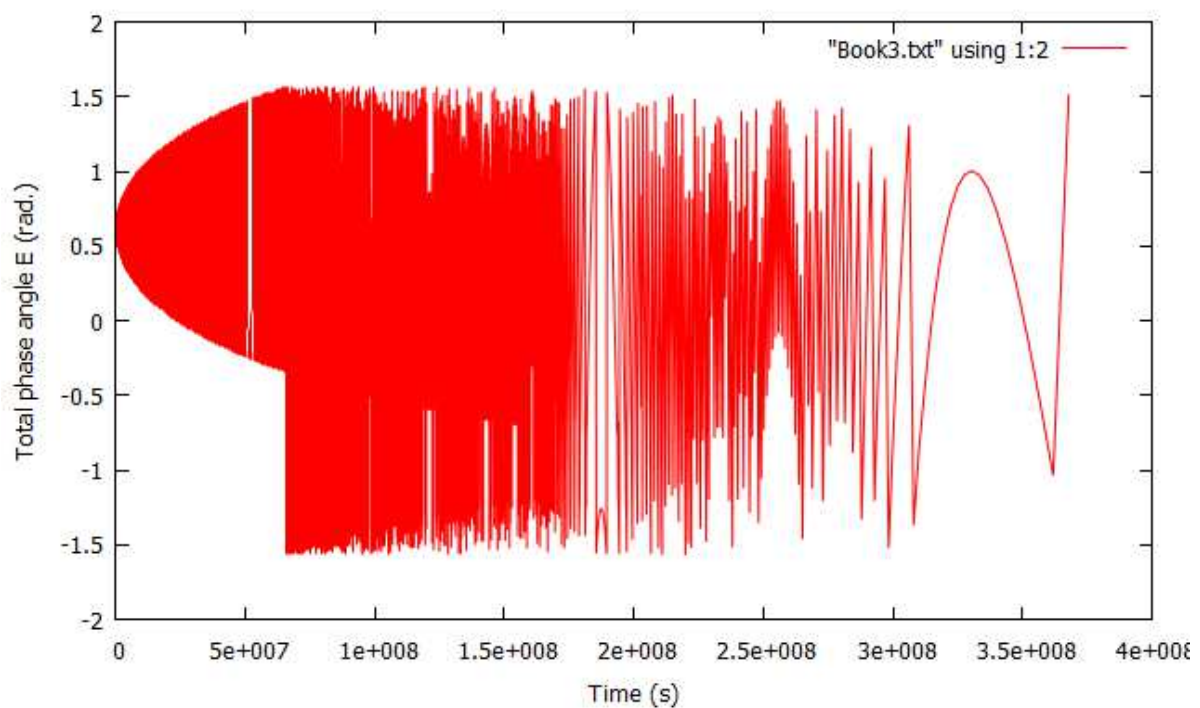

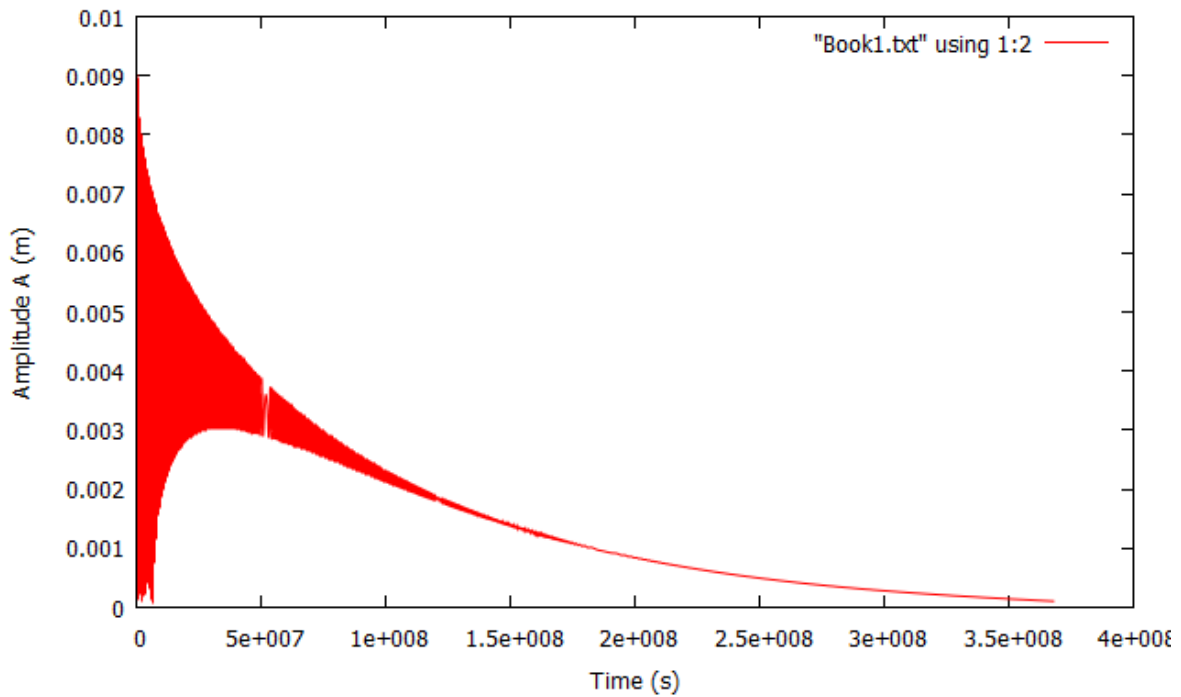**Fig. 3.2:** This **r**epresents the graph of the total phase angle E of the constitutive carrier wave against time t .

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Fig. 3.3: This **r**epresents the graph of the amplitude A of the constitutive carrier wave against time t .

Fig. 3.4: This **r**epresents the graph of the constitutive carrier wave displacement against time t .

4.0 DISSCUSION OF RESULTS

The fig. 3.1 represents the variation of the spatial oscillating part of the carrier wave with time. It oscillates between the values of ± 1 and converges to zero when the multiplier has attained its maximum value $\lambda = 19332$. Generally, the first significant feature of the spectra given by the figures is the definite singularity at time t = 5 x 10^7 s (19 months) or about 2 years. This definite singularity is an indication of the attenuating constituent parameters of the biological system of Man due to the presence of the HIV. However, the host system further renormalizes to cancel the systems defect and hence the continuous bold spectra.

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The second obvious significant feature common to the figures is the depletion in the spectra at t = 1.7 x 10^8 s (65 months) or about 5 years. The interpretation of this depletion is that the HIV is now taking active dominant control of the host biological system. Thus the constituent parameters of the HIV wave function are gradually becoming equal to those of the host. This results to the decay in the active constituents of the Host system. The destructive effect of the HIV in the host system is now becoming very intense and difficult to control.

Finally, the graphs show that the spectra of the infected biological system of the host degenerate into AIDS if uncontrolled around $t \geq 2.5 \times 10^8 s$ (96 months) or about 8 years. This is marked by the faint and sharp separations in the spectra lines of figs 3.1, 3.2 and 3.4. The biological system of Man goes to zero when $t = 367894196 s$ which is about 12 years.

The Fig. 3.2 shows that within the first 2 years (t = 5 x 10^7s) when the multiplicative factor $0 \leq \lambda \leq 14633$, the total phase angle E experiences both positive and negative increase in values. Also the figure reveals a bold spectrum in the total phase angle of the constituted carrier wave in the interval of the multiplicative factor $0 \leq \lambda \leq 18874$ and time $0 \leq t \leq 1.7 \times 10^8 s$ ($0 \leq t \leq 5$years). In this regime the phase angle of the 'host wave' is fluctuating between both positive and negative values thereby undergoing constructive and destructive

interactions with that of the 'parasitic wave'. Beyond this interval, that is, when the time $t \geq 1.7 \times 10^8 s$ and the multiplier $18875 \leq \lambda \geq 19332$, the spectrum lines of the total phase angle becomes faint and sharp showing a steady depletion in the total phase angle of the carrier wave. The total phase angle of the constitutive carrier wave has maximum and minimum value of ± 1.5468 rad. However, the spectrum does not finally go to zero rather it diverges even when the multiplier has attained its maximum value $\lambda = 19332$.

In Fig. 3.3, the amplitude of the carrier wave is initially imaginary in the interval $0 \leq \lambda \leq 1866$ and $0 \leq t \leq 122$s while the amplitude of the carrier wave is $i\,0.00418631m \leq A \leq i\,0.00405311m$. However, it is the absolute values of the amplitude that we used in the graphical presentation. Subsequently, the amplitude is made up of the imaginary and real part, $A = A_1 + iA_2$ in the interval of the multiplier $0 \leq \lambda \leq 6432$, and the time $0 \leq t \leq 6287092 s$ and the amplitude of the carrier wave is $i\,0.00418631m \leq A \leq i\,0.00018918m$. This shows

that the motion is actually two-dimensional (2D). Thus A_1 and A_2 are the components of the amplitude in x and y - directions, and A is tangential to the path of the moving amplitude in the carrier wave. This region of real and imaginary values of the amplitude is an indication of the human system to guild and renormalizes the system against the effect of the interfering HIV 'parasitic wave'.

There is constant agitation by the intrinsic parameters of the 'host wave' to suppress the destructive influence of the interfering 'parasitic wave' in this region. The intrinsic parameters of the 'host wave' are posing a serious resistance to the destructive tendency of the 'parasitic wave'. The effect of the imaginary decay in the amplitude is unnoticeable or inadequately felt by the human system in this interval. Although, unnoticeable as it may, but so much imaginary destructive harm would have been done to the intrinsic constituent parameters of the 'host wave'.

Beyond this interval the amplitude of the carrier wave begins to fluctuate with only real values in the interval of the multiplier $6433 \leq \lambda \leq 19233$ and the time 6289274 s $\leq t \leq 241907463$ s or (73 days $\leq t \leq 8$ years) and the amplitude of the carrier wave is $0.00337988m \leq A \leq 0.00054097m$. In this region the HIV infection is now

taking absolute effect on the biomechanical system of the infected candidate. In other words, counting from the moment one takes in the HIV, there would be absolute indication and manifestation of its presence after 73 days. This in the clinical literature of diseases is regarded as the window period.

The non-consistent attenuating behavior in this interval is a consequence of the fact that the amplitude of the carrier wave do not steadily go to zero, rather it fluctuates. The fluctuation is due to the constructive and destructive interference of both the 'host wave' and the 'parasitic wave'. In the regions where the amplitude of the carrier wave is greater than either of the amplitude of the individual wave, we have constructive interference, otherwise, it is destructive.

Our calculation shows that in the absence of specific treatment, the infection degenerates into AIDS after 8 years. In this case, it is observed that there is a steady exponential decrease in the values of the amplitude in the interval $242359220 s \leq t \leq 367894196 s$ (8 years $\leq t \leq 12$ years) when raising multiplier $19234 \leq \lambda \leq 19332$.

This consistent decrease leads to a gradual reduction and weakening in the initial strength of the active constituents of the host biological system. Consequently, the amplitude of the constituted carrier wave consistently attenuates to zero when the raising multiplier $\lambda \geq 19332$ and the time $t \geq 367894196$ s or about 12 years. The amplitude of the carrier wave thus varies asymptotically between $0.00053612m \leq A \leq 0.00010378m$.

The graph of the carrier wave against time is shown in fig. 3.4. Of course we know that the carrier wave is the product of the amplitude and the spatial oscillating phase. Consequently, the calculated values of the amplitude which is the maximum displacement from some origin are usually greater those of the carrier wave.

As we have said before now, the spectrum of the carrier wave is similar to those of figs, 3.1 and 3.2. The carrier wave experiences a steady damping process and it is consistently attenuated to zero. The spectrum of the carrier wave was initially bold with some regular discontinuities in the time interval 1.5×10^8 s $\leq t \leq 2 \times 10^8$ s or about 5 years. One could even suspect that with the nature of the depletion, this interval should mark the beginning of AIDS after infection. The attenuation of the carrier wave to zero is rapid in the interval when the raising multiplier $19234 \leq \lambda \leq 19332$ and the time 2.5×10^8 s $\leq t \leq 4 \times 10^8$ s. This of course represents the interval of the predominance of the dynamic constituents of only the interfering HIV 'parasitic wave', that is existing AIDS, while those of the resident 'host wave' are critically undergoing damping.

Thus the phenomenon of AIDS actually occurs in the interval when the raising multiplier $19234 \leq \lambda \leq 19332$ and the time 242359220 s $\leq t \leq 367894196$ s or (8 years $\leq t \leq 12$ years). This interval revealed that in the absence of specific treatment, people infected with HIV develop AIDS within eight years. Consequently, within this interval the system of the infected candidate can no longer recover from the HIV infection irrespective of any treatment administered. The carrier wave which describes the coexistence of the biological system of Man and the HIV ceases to exist –the phenomenon called death, around 12 years after infection. This is as a result of the fact that all the active constituents of the 'host wave' would have been completely attenuated by the influence of the interfering HIV 'parasitic wave'.

5.0   CONCLUSION

It seems from the results, that the actual dynamic components of the HIV responsible for their destructive tendency are $b\lambda$, $n'\lambda$, $\varepsilon'\lambda$ and $k'\lambda$. The result shows that every active system has an inbuilt parameter that enables it to correct any anomaly that affects it. This study revealed that in the absence of specific treatment, people infected with HIV develop AIDS within eight years and the average survival time after infection with HIV is found to be about 8 to 12 years. Consequently, within this time interval the biological system of the infected candidate can no longer recover from the HIV infection irrespective of any treatment administered. Also the cessation of the carrier wave which describes the coexistence of the system of Man and the HIV is not instantaneous but gradual. Initially the biological system of Man tends to annul the effect of the presence of the HIV starting from the moment the person contacted it. Consequently, the existence or the life span of any physically active system is determined by the resistance of its basic intrinsic parameters to the destructive influence of any external factor. Thus this study has to some extent provided the means of determining the activity and performance of HIV in the human blood circulating system. As a result, the HIV can be selectively and discriminately destroyed from the human biological system by anti-vibrating component without causing the slightest harm to the body mechanics of Man. This work thus identifies the matrix of scientific priorities that should bring us measurably closer to our vision of developing a cure for HIV/AIDS infection. Finally, the significance of the HIV pandemic has been scientifically exaggerated, since the search strategy for the cure may lie outside the complex scientific ideas and approach currently propounded.

5.1   Limitations

Limitations in this work would include the use of some physical data such as the dynamic viscosity of blood whose values may not be exact based on small variations during experimental measurement. Secondly, the study relied on measurements of some sensitive physical parameters such as the radius and length of the human aorta, which may differ slightly between individuals. Consequently, the findings presented here may not be generalizable to the greater sense except if one can be provided with uninterrupted physically measured data. However, variations in the results or not, the average statistical difference in the results that can be obtained will be very small and tolerable.

5.2   Suggestions for further work

This study in theory and practice can be extended to investigate wave interference and propagation in two- and three- dimensional systems. The carrier wave function we developed in this work can be utilized in the deductive and predictive study of wave attenuation in exploration geophysics and telecommunication engineering. This work can also be extended to investigate energy attenuation in a HIV/AIDS patient.

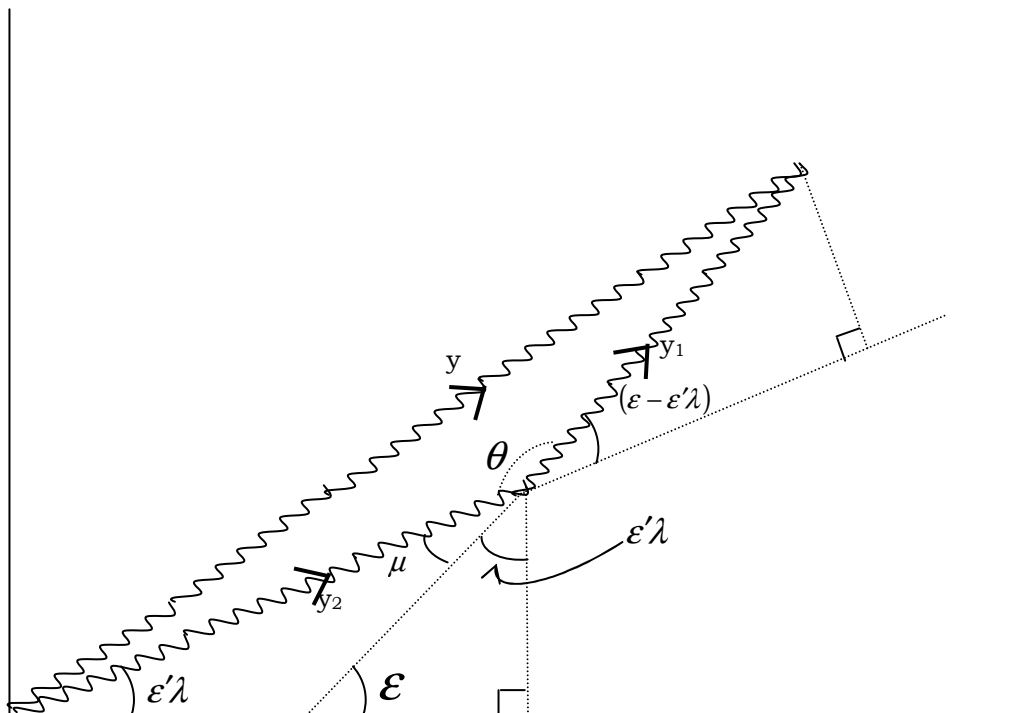**APPENDIX :** Vector representation of the superposition of the wave function of Man and that of HIV.

Fig. A1. Represent the resultant carrier wave y after superposing the HIV wave function y_2 on the Host wave function y_1. Where, $\mu + \mathcal{E}'\lambda + 180^0 - \varepsilon = 180^0$; $\mu = \varepsilon - \mathcal{E}'\lambda$; $\theta = 180^0 - (\varepsilon - \mathcal{E}'\lambda)$; $\theta = \pi - (\varepsilon - \mathcal{E}'\lambda)$.

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(iii)   Member of the International AIDS Society in 2013.

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