

On The Effect of Affinity Hemodialysis on HIV/AIDS

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Abstract

We use the Lyapunov theory to confirm observations of eigenvalue theory and experiments that affinity hemodialysis could prevent HIV positiveness developing into full blown AIDS.

Keywords: , Affinity hemodialysis, HIV/AIDS, Lyapunov function, stability of critical points..

1. Introduction:

HIV is a deadly disease and so there is a need to control the spread of the disease. Many researchers studied the modes of spreading the disease. Among the researchers are mathematicians, medical scientists and biologists..

Castillo and Carlos [7] developed a series of mathematical models which view AIDS as an exclusively sexually transmitted disease. Their objective was to evaluate the role of long periods of infectiousness in the dynamics of HIV. These models are natural extensions of those developed by [2]. They divided the population under consideration into social group defined by criteria such as sex and sexual behavior.

Gani and Yakowitz [8] studied modeling the spread of HIV among intravenous drug users, they considered a random allocation model for the transmission of HIV by needle sharing among a group of intravenous drug users who are friends or relatives (buddy-users). A Markov chain approach was used to track the increase in infective in a stable group of such IVDUs, some of whom are HIV positive. The model is modified to allow the replacement of infection in the group, with the group size remaining constant.

Greenhalgh and Hay [10] studied mathematical modeling of the spread of HIV/AIDS among injecting drug users. The work is based on a model originally due to Kaplan (1989). The Kaplan model was an outline on a more realistic extension. It was examined that there is a critical threshold parameter R_0 which determines the behavior of the model. $R_0 \leq 1$ there is a unique disease-free equilibrium and if $R_0 < 1$ the diseases die out. If $R_0 > 1$, then these diseases free equilibrium is unstable. There is a unique endemic equilibrium which is locally stable in the model.

Masayuki [14] presented a mathematical analysis of the spread of HIV/AIDS in Japan. A mathematical model incorporating pair formations between adults and sexual contacts with commercial sex workers was used. According to the analyses of the model it was concluded that the actual situation of HIV spread in Japan, should lie very near the critical point that determines whether the explosive HIV spread actually takes place.

Hyman et al [11] developed some risk-based model on the assumption that individuals with multiple sexual partners are usually infected first and tend to become the major source of spread into those groups with fewer sexual partners. In addition, they also explored the role of variable infectivity in the context of their model. They observed the number of contacts and probability of infection from contact with an infected, then partners are chosen at random from the population. Hence the model reduced to that of [2].

Hyman et al [11] presented a simple differential susceptibility (DS) model in which the infected population is homogenous, but the susceptible population is divided into n groups according to their susceptibilities. They also combined the DS and DI models. In each of these models, they obtained the reproductive number.

Kimber and Aboiyar [12] studied a mathematical model for the prevention of HIV/AIDS in varying population.

Tullis, [18] presented mathematical model of the effect of affinity hemodialysis on the T-cell depletion leading to AIDS. Deterministic mathematical models based on the well-known Perelson formulations were used. The two models predicted a rapid and sustained reduction, in gp 120 levels. The calculations support the contention that affinity hemodialysis is a potentially useful adjunctive therapy which can be employed to treat. HIV – infected patients in conjunction with drug therapy but affinity hemodialysis treatment may become a viable option for those patients resistant to anti-retroviral drugs or those unable to take the drugs due to the side effects of those medications.

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Journal of the Nigerian Association of Mathematical Physics Volume 18 (May, 2011), 299 – 302

Kimbir [13] studied a two-sex model for HIV/AIDS transmission dynamic in a polygamous female dominant population. It was observed that a disease – free equilibrium state exists which is locally and asymptotically stable (LAS) if the parameter $R_0 < 1$. The author concluded that it is possible to eradicate HIV/AIDS in polygamous growing population.

Ayodele and Ayeni [5] worked on some remarks on quantification of intrinsic residual viral replication in treated HIV infected patients.

Ayeni et al [3] presented effect of positive initial HIV-specific CTLs on intrinsic viral replication in treated HIV infected patients.

Akinwande [1] presented paper on a mathematical model of the dynamics of the HIV/AIDS disease pandemic. The model used consists of ordinary and partial differential equations. The population was partitioned into three compartments these are the susceptible $S(t)$ –virus free members, the removed $R(t)$ –no susceptible infections members and the infected $I(t)$ –infected at various stages. It was observed that a low level of k indicates high rate of death among the infected while high level of k indicates longer life span for the infected, where k is the measure of the effectiveness of efforts at slowing down the death of infected members.

Oluyo et al [15] discussed mathematical analysis of the global dynamics of a model of HIV infection of CD_4^+ T-cells. Using Rene Descartes’ theory of solutions it was shown that if

the so called basic reproduction $R_0 < 1$, the infection will eventually die out but if $R_0 > 1$ then the infection will lead to full blown AIDS.

Wang and Li [19] discussed mathematical analysis of the global dynamics of the model for HIV infection of CD_4^+ T cells. In their model they proved that if the basic reproduction $R_0 \leq 1$, the infection is cleared from the T-cell production, if $R_0 > 1$, the infection persist. This result is similar to [15] using contact tracing as a method of controlling the spread of HIV/AIDS observed that contact tracing could be used to control the spread of the virus.

Oluyo and Ayeni [16] discussed a mathematical model of virus neutralizing antibody response. It was shown that the spread of the disease can be controlled if the critical parameter $R = \frac{\delta B^*}{\mu_6} < 1$, where δB^* is the scale initial value of B cells and μ is the death rate of the virus.

Wen and Lou [19] worked on the global dynamics of a model about HIV-1 infection in vivo. The model includes four components: the healthy T cells, the latent-infected T cells, the active-infected T cells and the HIV virus. Two equilibria were discovered: the healthy equilibrium which is globally stable and the infected equilibrium which is also globally stable.

Bhunu et al [6] worked on mathematical analysis of a two strain HIV/AIDS model with antiretroviral treatment. The system of non – linear ordinary differential equations was presented. The disease- equilibrium is shown to be globally asymptotically stable when the associated epidemic threshold known as the basic reproduction number for the model is less than unity.

Smith et al [17] discussed an accurate two-phase approximate solution to an acute viral infection model. It was observed that data and numerical solutions suggest the growth and decay of phase are linear on a log scale which viral dynamic models are typically non linear with analytical solutions difficult to obtain the exponential nature of the solutions suggests approximations can be found.

Ayeni et al [4] worked on some new result on affinity hemodialysis and T-cell recovery.

A key factor in the analysis is μ . When μ is zero, the possibility of quasi-steady infected equilibrium does not exist. Thus a stable infected equilibrium does not arise. The paper shows further that affinity hemodialysis is a potentially useful adjunctive therapy which can be employed to treat HIV infected patients.

Garba and Gumae [9] used a deterministic model for assessing the impact of counseling, condom use and treatment strategies on the transmission dynamics of HIV/AIDS in Nigeria. The results show that whenever the associated reproduction number $R_0 < 1$, the disease free equilibrium is globally asymptotically stable (GAS).

1. Mathematical formulation and stability of equilibrium

We take the mathematical model as

$$\frac{dT}{dt} = \pi - d_1T - k_1TV + \mu T_i, T(0) = T_o \quad (i),$$

$$\frac{dT_i}{dt} = k_1TV - d_2T_i - \mu T_i, T_i(0) = T_{io} \quad (ii),$$

$$\frac{dV}{dt} = k_2T_i - cV, V(0) = V_o \quad (iii),$$

where the symbols are as in Ayeni et al. [10].

The two equilibrium for (i) – (iii) are

$$(T, T_i, V) = \left(\frac{\pi}{d_1}, 0, 0 \right) \text{ disease free equilibrium and}$$

$$(T_*, T_{i*}, V_*) = \left[\frac{\mu + d_2}{k_1 k_2} c, \frac{\pi}{d_2} - \frac{d_1(d_2 + \mu)c}{d_2 k_1 k_2}, \frac{k_2}{c} \left\{ \frac{\pi}{d_2} - \frac{d_1(d_2 + \mu)c}{d_2 k_1 k_2} \right\} \right] \text{ disease infected equilibrium}$$

Letting $x = T - T_*$, $y = T_i - T_{i*}$, $z = V - V_*$

Equation (i) – (iii) become (neglecting higher order terms)

$$\frac{dx}{dt} = -(d_1 + k_1 V_*)x + \mu y - k_1 T_* z \tag{iv}$$

$$\frac{dy}{dt} = k_1 V_* x - (d_2 + \mu)y + k_1 T_* z \tag{v}$$

$$\frac{dz}{dt} = k_2 y - cz \tag{vi}$$

Ayeni et al [4] showed by eigenvalue technique that (T_*, T_{i*}, V_*) is asymptotically stable. In this paper, we shall use

Lyapunov technique. We let the Lyapunov function $v(x, y, z) = \frac{1}{2} \left[(x - \bar{x})^2 + (y - \bar{y})^2 + (z - \bar{z})^2 \right]$ (vii)

When $(\bar{x}, \bar{y}, \bar{z}) = (0, 0, 0)$ (vii) becomes

$$v(x, y, z) = \frac{1}{2} (x^2 + y^2 + z^2) \tag{viii}$$

$V(0,0,0) = 0$ and $V(x, y, z) > 0$ for every other point.

Then $\frac{dv}{dt} < 0$ if $2\sqrt{d_1 d_2} = \mu + k_1 V_*$, $2\sqrt{\frac{\mu c}{2}} = k_1 T_* + k_2$ and $\sqrt{\frac{c k_1 V_*}{2}} = k_1 T_*$

Hence (T_*, T_{i*}, V_*) is asymptotically stable under such conditions.

Result and Discussion

This means , biologically that an HIV positive cannot develop into full blown aids as [4] showed by another method and as [18] suggested. We conclude that all medical measures at curtailing the infected CD4+T cells should be encouraged.

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