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An Analytical Model for Prevention of Mother to Child Transmission of HIV through Breastfeeding.

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Abstract
In this paper, a non- linear model to study the dynamics of transmission of HIV from mother to child through breastfeeding was developed. Stability of the proposed method was established. Numerical Simulation were performed to show the level of effectiveness and accuracy of the model over existing ones in the Literature.
Keywords: *Analytical Model, Human Immune deficiency Virus, Mother to Child transmission*

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I. Introduction

Viral infections are infections caused by viruses. Viruses are tiny germs which cause severe illnesses such as hepatitis B and C, HIV/AIDS, small pox and Ebola, measles, mumps etc. They invade living normal cells and use those cells to multiply and produce other viruses like themselves. Viral infectious run their course and usually heals on their own but others like HIV, Hepatitis do not. They usually do not respond to antibiotics instead; treatment is focused on providing relief from their symptoms. Sub-Saharan Africa has continued to bear the greatest burden of the human Immuno deficiency virus and acquired immune deficiency syndrome HIV/AIDS epidemics (Louise et al, 2004). 90% of the 2.1million children infected globally with HIV in 2003 were as a result of mother to child transmission, MTCT (WHO, 2004). The high prevalence of MTCT of HIV is threatening to reverse the gains of child survival strategy in the African continent. It is currently estimated that there are 3.8million Nigerians living with HIV. About 90% of HIV infection in children < 15years is due to MTCT (WHO, 2002). Breastfeeding may thus be responsible for one-third to half of HIV infection in infants and young children in Africa (De Cock et al, 2002)

There are three major modes of transmission from HIV positive mother to child; during pregnancy, delivery or through breastfeeding. The rate of MTCT of HIV is affected by many factors including high viral load, mode of delivery, prolonged rupture of membrane, prematurity and breastfeeding. Of all the modes of transmission, transmission through breastfeeding is the objective of this research work. Use of anti-retroviral drugs, Cesarean section as a mode of delivery, active monitoring of labor and good antenatal care respectively will prevent transmission through other mode. Thus, with a disciplined way of life and professional care, other modes of transmission can be effectively controlled with a maximum success unlike the breastfeeding where an infant is forced to carry a burden, he has no direct responsibility to. Given the risk from breastfeeding, reduction of such HIV transmission is one of the most pressing public health challenges confronting researchers, health care professionals, health policy makers and HIV infected women in Africa.

Currently, World Health Organization, WHO advocates six months exclusive breastfeeding but there is a strong evidence however that the longer the duration of breastfeeding, the greater the risk of transmission. In other words, the risk is cumulative (Leroy et al, 1998, Miotti et al, 1999, Leroy et al, 2002, The Petra study Team, 2002, Read et al, 2003). In the developed countries, mothers who are HIV positive avoid breastfeeding completely and thus feed their babies with formula because they either can afford it or have support from agencies funded by the government or individuals and are not faced with other killer diseases of children like diarrhea etc. but in many low and middle income countries, formula feeding of infants is neither feasible nor safe due to the poverty level of women. Sanitation is lacking and clean water to mix formula is often not available. Many families cannot also afford infant formula. It may also not be easy to afford enough fuel to boil water to prepare the formula safely as constant power is still a challenge.

In this paper, we intend to develop a model which will prevent vertical transmission of HIV. it is assumed that HIV positive mothers will breastfeed their babies and all babies will not be positive at birth

II. Development of the Model

$$N - Total\ population\ of\ Infants$$
$$P - Pre\ HIV\ class$$
$$I - infective\ class\ (HIV\ positive\ class)$$
$$\beta - Probability\ of\ Transmission\ in\ a\ suckle$$
$$\theta - Rate\ of\ movement\ from\ pre\ HIV\ class\ to\ HIV\ class$$
$$\alpha - Frequency\ of\ suckling$$
$$\eta - length\ of\ each\ feeding\ time$$
$$\delta - Induced\ mortality\ rate$$
$$\gamma - Rate\ of\ dropping\ HIV\ staustus\ acquired\ at\ birth$$

Assume a population of $N(t)$ at time, *t* with a constant inflow of susceptible at rate, πN. We divide the population into 3 classes; susceptible, $S(t)$, pre- HIV class, $P(t)$and Infective, $I(t)$. We assume that the susceptible becomes HIV infected through suckling of infected milk during breastfeeding, there is no direct recruitment into the infective are not infected either during pregnancy or at delivery. Some of the susceptible join the pre-HIV class depending on the viral loadand then may either proceed at a rate of θ to join the infected (symptomatic) class or remain in the pre-HIV class (asymptomatic).

$$N = S + P + I$$
$$\frac{ds}{dt} = \gamma N - \alpha\beta\eta S$$
$$\frac{dp}{dt} = \eta\beta\eta S - \theta P$$
$$\frac{dI}{dt} = \theta P - \delta I$$
$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dP}{dt} + \frac{dI}{dt}$$

Thus,

$$\frac{dN}{dt} = \gamma N - \delta I$$

Normalizing the model we have the following systems of nonlinear Equations:

$$\frac{\boldsymbol{ds}}{\boldsymbol{dt}} = \gamma - (\alpha\beta\eta + \gamma)s + \delta is = 0$$
$$\frac{\boldsymbol{dp}}{\boldsymbol{dt}} = \alpha\beta\eta s - (\theta + \gamma)p + \delta ip = 0$$
$$\frac{\boldsymbol{di}}{\boldsymbol{dt}} = \theta p - (\delta + \gamma)i + \delta i^2 = 0 \quad \ldots 1.1$$

Such that

$$s + p + i \leq 1$$

For the model to be epidemiologically sensible, we prove that all the system variables are non-negative.
Theorem 1.1 (Positivity of Solution). Let $\tau = \{(x, y, z) \mid x, y, z \in \mathbb{R}^T \text{ and } x + y + z = 1$. Then for a solution $(s(t), p(t), i(t))$ to system (1.1), $\big(s(0), p(0), i(0)\big) \in \tau \Rightarrow \big(s(t), p(t), i(t)\big) \in \tau \ \forall\ t \geq 0$.
Proof:

$$\frac{ds}{dt} = -[\alpha\beta\eta + \gamma]s + \gamma + \delta is$$

$\frac{ds}{dt} \geq -[\alpha\beta\eta + \gamma]s$ …1.2

$$Integrating:$$
$$s(t) \geq s(0)\exp\{-(\alpha\gamma\eta + \gamma)t\}$$
$$hence\ s(t) \geq 0, for\ t \geq 0\ provided\ that\ s(0) \geq 0$$

Also,

$\frac{dp}{dt} \geq -(\theta + \gamma)p$ …1.3

$$so\ that\ p(t) \geq p(0)\ \exp\{-(\theta + \gamma)t\}$$

Thus $p(t) \geq 0 for\ t \geq 0\ provided\ that\ p(0) \geq 0$
Similarly,
$i(t) \geq i(0)\ \exp\{-(\delta + \gamma)t\}$ …1.4
Thus $i(t) \geq 0\ for\ t \geq 0\ provided\ that\ i(0) \geq 0$

This completes the proof

Theorem 1.2: Given the systems of nonlinear equations (1.1), the Disease- Free equilibrium, DFE is given by $E_0 = (\frac{\gamma}{\alpha\beta\eta+\gamma}, 0,0)$.

Proof:

At an equilibrium point,

$$\frac{ds}{dt} = 0 = \frac{dp}{dt} = \frac{di}{dt}$$

Also, at a Disease-free equilibrium, DFE, $i = 0 = p$

Then from (1.1):

$$\gamma - (\alpha\beta\eta + \gamma)s = 0$$

$\Rightarrow s^* = \frac{\gamma}{\alpha\beta\eta+\gamma} 1.$

$Thus\ the\ DFE\ is\ E_0 = (\frac{\gamma}{\alpha\beta\eta+\gamma}, 0,0).$

2.1 Stability of the nonlinear model defined by System (1.1)

We establish the stability of the DFE by the basic Reproduction number R_0 determined from the Next Generation Method on systems of nonlinear equations (1.1) in the form of matrices, F and V where F_i is the loss of the positive status acquired at birth and V_i is the rate of acquiring the positive status after a period of time. The higher the value of R_0, the greater the risk of transmission per feeding time. Basis Reproduction number is the effective number of new infections caused a HIV positive lactating mother.

2.2 Reproduction number of the Model defined by (1.1)

$$F = \begin{bmatrix} (\alpha\beta\eta + \gamma & 0 \\ -\alpha\beta\eta & 0 \end{bmatrix}, v = \begin{bmatrix} -(\theta+\gamma) & 0 \\ \theta & -(\delta+\gamma) \end{bmatrix}$$

$$F * v^{-1} = \begin{bmatrix} \frac{-(\alpha\beta\eta+\gamma)}{\theta+\gamma} & 0 \\ \frac{\alpha\beta\eta}{\theta+\gamma} & 0 \end{bmatrix}$$

Thus $R_0 = \frac{\alpha\beta\eta}{\theta+\gamma}$

If $R_0 > 1$, there will be an increase in the rate of transmission and if $R_0 < 1$, the prevalence is zero

2.3 Endemic Equilibrium and local stability of the model defined by (1.1)

Theorem 1.3: Given the systems of nonlinear equations (1.1), the Endemic Equilibrium point $E_1^* = (s^*, p^*, i^*) is\ asymptotically\ stable$

Proof:

Given the system of Equations (1.1) below

$\frac{ds}{dt} = \gamma - (\alpha\beta\eta + \gamma)s + \delta is = 0 ...(\text{i})$

$\frac{dp}{dt} = \alpha\beta\eta s - (\theta + \gamma)p + \delta ip = 0 ...(\text{ii})$

$\frac{di}{dt} = \theta p - (\delta + \gamma)i + \delta i^2 = 0 ...(\text{iii})$

Let $s + p + i = 1, substitue\ i = 1 - s - p\ into\ eqns\ (i - ii) above$ and from (iii):

$$i = \frac{(\delta+\gamma) + \sqrt{(\delta+\gamma)^2 - 4\delta\theta p}}{2\delta} ...(iv)$$

$$\gamma - (\alpha\beta\eta + \gamma - \delta)s - \delta sp - \delta s^2 = 0 \quad ...(v)$$

$$\alpha\beta\eta s - (\theta + \gamma - \delta)p - \delta sp - \delta p^2 = 0 \quad ...(vi)$$

$$(\delta+\gamma)^2 - 4\delta p > 0 \implies (\delta+\gamma)^2 \geq 4\delta\theta p$$

$$Thus, p \leq \frac{(\delta+\gamma)^2}{4\delta\theta} \quad ...(vii)$$

$$Let\ p^* = \frac{(\delta+\gamma)^2}{4\delta\theta} - \varepsilon \quad ...(viii)$$

$$then\ from\ (v):$$

$$s = \frac{\alpha\beta\eta + \gamma - \delta\left[1 - \left(\frac{(\delta+\gamma)^2}{4\delta\theta} - \varepsilon\right)\right] - \sqrt{(\alpha\beta\eta + \gamma - \delta\left[1 - \left(\frac{(\delta+\gamma)^2}{4\delta\theta} - \varepsilon\right)\right]^2 + 4\delta y}}{-2\delta} ...(ix)$$

$$provided\ that,$$

$$\sqrt{(\alpha\beta\eta+\gamma-\delta\left[1-\left(\frac{(\delta+\gamma)^2}{4\delta\theta}-\varepsilon\right)\right]^2+4\delta\gamma}\ \geq \alpha\beta\eta+\gamma-\delta\left[1-\left(\frac{(\delta+\gamma)^2}{4\delta\theta}-\varepsilon\right)\right]$$

$$s^*=\frac{M-\sqrt{(M^2+4\delta\gamma}}{-2\delta}$$

$$where\ M=\alpha\beta\eta+\gamma-\delta\left[1-\left(\frac{(\delta+\gamma)^2}{4\delta\theta}-\varepsilon\right)\right]$$

$$from\ (v)-(vi):$$

$$(2\alpha\beta\eta+\gamma-\delta)s-(\theta+\gamma-\delta)p+(s^2-p^2)\delta=\gamma\quad\ldots(x)$$

$$from\ (v)+(x):$$

$$-\delta p^2-\big(\theta+\gamma-\delta(1-s)\big)p+(\alpha\beta\eta)s=0\quad\ldots(xii)$$

$$p=\frac{\theta+\gamma-\delta(1-s)\pm\sqrt{[\theta+\gamma-\delta(1-s)]^2+4\delta\alpha\beta\eta}}{-2\delta}$$

$$p^*=\frac{\theta+\gamma-\delta(1-s)-\sqrt{[\theta+\gamma-\delta(1-s)]^2+4\delta\alpha\beta\eta}}{-2\delta}\ldots(xiii)$$

$$provided\ that,$$

$$\sqrt{[\theta+\gamma-\delta(1-s)]^2+4\delta\alpha\beta\eta}>\theta+\delta-\delta(1-s)$$

$$from\ (iv):$$

$$i=\frac{(\delta+\gamma)+\sqrt{(\delta+\gamma)^2-4\delta\theta p^*}}{2\delta}$$

$$i^*=\frac{\delta+\gamma+\sqrt{4\delta\theta\varepsilon}}{2\delta}\quad\ldots(xv)$$

$$The\ Endemic\ Equilibrium\ is\ thus\ given\ by$$

$$(s^*,p^*,i^*)=(\frac{M-\sqrt{(M^2+4\delta\gamma}}{-2\delta},\frac{\theta+\gamma-\delta(1-s)-\sqrt{[\theta+\gamma-\delta(1-s)]^2+4\delta\alpha\beta\eta}}{-2\delta},\frac{\delta+\gamma+\sqrt{4\delta\theta\varepsilon}}{2\delta}\ )$$

$$where\ M=\alpha\beta\eta+\gamma-\delta\left[1-\left(\frac{(\delta+\gamma)^2}{4\delta\theta}-\varepsilon\right)\right]$$

Given $R_0=\frac{\alpha\beta\eta}{\theta+\gamma}>1$, then EE is asymptotically stable.

2.4 Bifurcation of the Model

Theorem (1.4): Solutions to the Systems of linear Equations (1.1)Bifurcate

Proof:

Critic al points of the solutions of eqn(1.1) are found from

$$\gamma-(\alpha\beta\eta+\gamma-\delta)s-\delta sp-\delta s^2=0$$

With the solutions:

$$(s_1,s_2)=(\frac{\alpha\beta\eta+\gamma-\delta(1-p^*)\pm\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}}{-2\delta})$$

$$thus\ s_1=(\frac{\alpha\beta\eta+\gamma-\delta(1-p^*)+\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}}{-2\delta})$$

$$s_2=(\frac{\alpha\beta\eta+\gamma-\delta(1-p^*)-\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}}{-2\delta})$$

$$This\ is\ a\ Tangent\ bifurcation$$

For $\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}>\alpha\beta\eta+\gamma-\delta(1-p^*)$,

$(\frac{\alpha\beta\eta+\gamma-\delta(1-p^*)-\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}}{-2\delta})$is unstable and is the separatrix while

$(\frac{\alpha\beta\eta+\gamma-\delta(1-p^*)+\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}}{-2\delta})$ is stable and is the attractor.

Both points merge if $\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}=0$ making the equilibrium point to be at $\left(\frac{\alpha\beta\eta+\gamma-\delta(1-p)}{-2\delta}\right)$.

If $\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}<0$, the equilibrium disappears.

However from the nature of the discriminant which is obviously positive and given that $\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}>\ \alpha\beta\eta+\gamma-\delta(1-p^*)$ then the epidemiologically sensible solution, $(\frac{\alpha\beta\eta+\gamma-\delta(1-p^*)-\sqrt{\alpha\beta\eta+\gamma-\delta(1-p^*))^2+4\delta\gamma}}{-2\delta})$ is unstable and so the virus survives.

2.5 Simulation of the Model:
The Numerical Simulation of the developed models is done to study the dynamical behaviors using standard parameters and some peculiar parameters of the feeding time based on the average age of the infants using MATLAB. To perform the simulation, the following data obtained from WHO statistics are used. $\alpha = 8, \beta = 0.3, \theta = 0.9, \eta = 0.37, \delta = 0.05, \gamma = 0.4, \mu = 0.5$ (based on the age of the child).
The Endemic equilibrium values are simulated using MATLAB as earlier stated and shown graphically in the figures below which show the proportion of variations of the various classes

Fig 1. Proportion of Susceptible class to Pre HIV CLASS

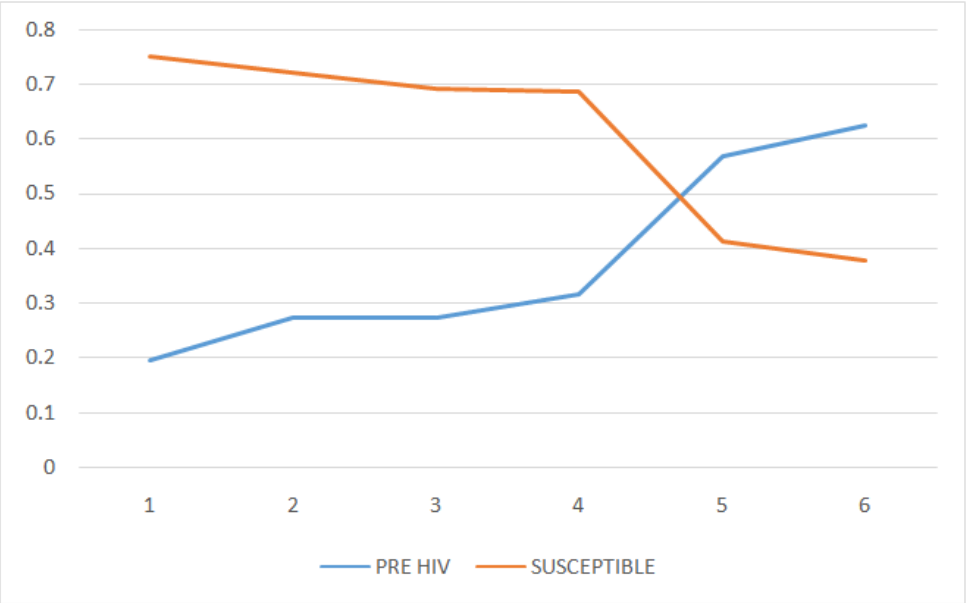


Fig 2:

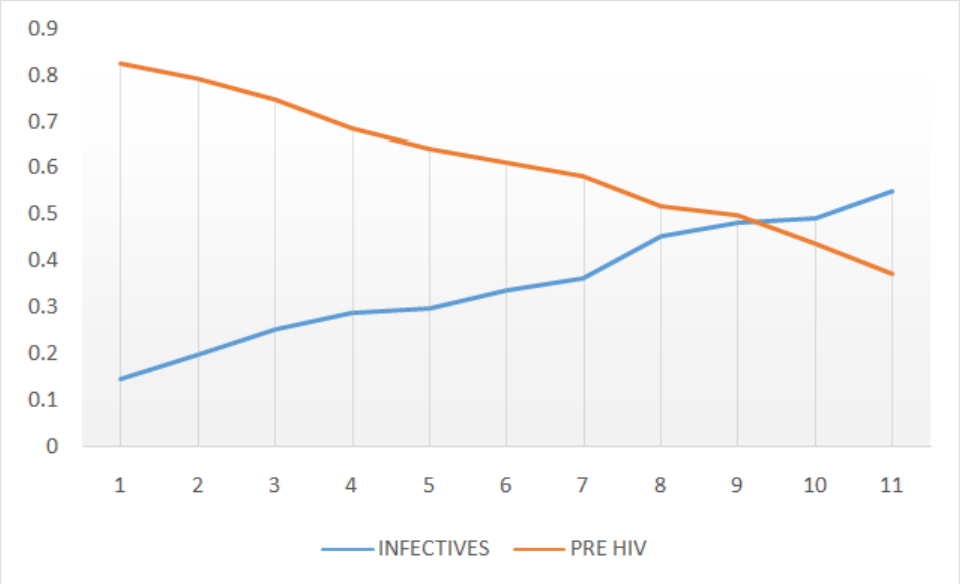
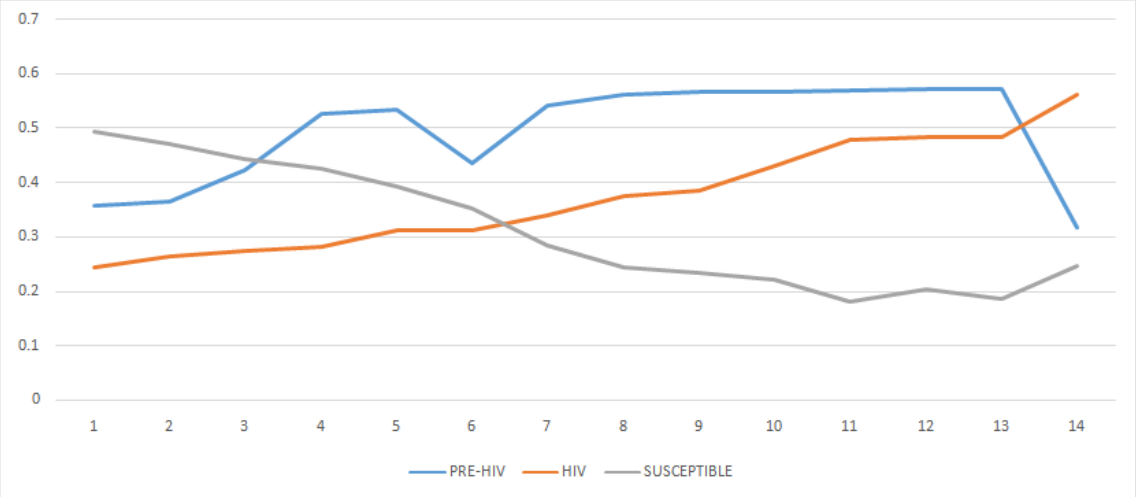


Fig 3:



III. Results and Discussion

A non- linear model has been developed and was used to study the dynamics of transmission of HIV from mother to child through breastfeeding under the assumption that the number of suckling determines the probability of transmission of HIV to the infant. The Reproduction number, R_0 was derived to show that if $R_0 < 1$, the virus dies off while if $R_0 > 1$, the virus thrives and becomes endemic. Two equilibrium points exist; the Disease-free equilibrium, DFE $E_0 = (1,0,0)$ and the Endemic Equilibrium, EE, $E_1 = (s^*, p^*, i^*) = \left(\frac{M-\sqrt{(M^2+4\delta\gamma}}{-2\delta}, \frac{\theta+\gamma-\delta(1-s)-\sqrt{[\theta+\gamma-\delta(1-s)]^2+4\delta\alpha\beta\eta}}{-2\delta}, \frac{\delta+\gamma+\sqrt{4\delta\theta\varepsilon}}{2\delta}\right)$. The Disease- free Equilibrium is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. While the Endemic Equilibrium exists when $R_0 > 1$ and is always locally asymptotically stable.

The Simulation shows a rise in the number of Pre-HIV as there was a decline in the number of pre-HIV cases. Similarly, the number of HIV case had a steady increase as the pre- HIV cases were reducing.Thus there is transmission of HIV from an infected mother to her infants through breastfeeding if there is no effective intervention made to prevent it

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