# Mathematical Modeling of Optimal Control of Hiv/Aids Prevalence among Fisherfolk in Lake Victoria Region

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Abstract: To date, HIV/AIDS has remained the most widespread viral STD with no cure and the leading cause of death. All what is available are viral load control regimen and treatment of opportunistic diseases. The highly advocated control measure remains as Abstinence, Being faithful and use of Condom, popularly denoted as (ABC) method. The ABC control measures relate directly to individual behavior, and the success depends on individual commitment and self-discipline. However, environmental and cultural practices have for a long time been blamed for observed high levels of prevalence, especially among the fisher folk community. In this paper, we consider the interaction dynamics of sub communities, their coupling strength viz a viz transmission rates and propose optimally strategies of curbing the pandemic. A mathematical compartmental model is formulated using ODE's for the distinct disease characteristic classes, then coupled via transmission rates and analyzed for the effect of disease control parameters. It was found that there exist a stable, positive and bounded invariant manifold, characterized by disease free equilibrium and endemic equilibrium points. Using data collected from Fisher folk around Lake Victoria region in Kenya, the equilibrium state is found to have a reproductive ratio of  $R_0 = 21.4965752$  and  $R_{ov} = 21.5412683$ . Control strategies studied include treatment  $\tau$ , public health education campaign to reduce sexual partners  $\phi$  and reduce recruitment to high risk class  $\lambda$  and reduce infectivity rate  $\beta$  through the use of abstinence, contraceptives or being faithful. It was found that the optimal control values to reduce the vector population reproductive ratio to values less than one  $R_{0v} < 1$  are;  $\tau > \tau$ 0.67,  $\beta \leq 0.000665$ ,  $\phi \leq 1$  and  $\lambda = 0$ . The achievement of these however depends on the budgetary allocation, which was not considered in this paper.

Key Words: Metapopulation, Disease Free Equilibrium, Endemic Equilibrium, Optimal Control, Stability, Boundedness, Positivity, Elasticity, Reproductive Ratio.

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# I. Introduction.

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Mathematical modeling in epidemiology has gained prominence since its introduction in 1040 by Malthus [1]. To date, mathematical modeling together with the use of computers in simulation has been of help in scientific investigations, thus shedding light on expected dynamics, and provides knowledge on the sensitivities and the range of values of parameters whose control can help avert epidemics. The most common and simple approach in epidemiological modeling is the use of compartments linked by transmission probabilities or transfer rates between the compartments. The population dynamics are then modelled using differential equations [2]. In this paper, the study of HIV dynamics is modelled using ODE's with individuals placed into two structures; general community (low risk) and the fisher folk community (high risk), who are differentiated by their economic activity and associated HIV prevalence [3]. Since time immemorial, it has been noted that fisher folk community has remained distinct, intact, with their own culture, social structures, and censored membership recruitment strategies and insignificant dismissal of members. This body has led to creation of a unique group with unique disease dynamics. These homogeneous groups of people are here referred to as a metapopulations. This paper looks at interacting metapopulations around Lake Victoria region in Kenya, which include; Homabay, Mbita, Samia and Kisumu. These groups are distinct from the rest of the population because of their extreme prevalence rate of over 14 times higher than the normal community [4]. This characteristic makes it possible to consider fisher folk as a HIV/AIDS vector, in relation to the rest of the people.

# II. Literature Review.

HIV/AIDS was discovered in 1981 and has remained the leading sexually transmitted disease causing death globally, affecting mostly impoverished people already suffering from poor nutrition and health [5]. As the name suggests, HIV is a virus that attacks the immune system, leaving the immune system incapacitated, allowing opportunistic diseases to significantly hamper the body's defensive mechanism and therefore lead to deteriorated body's health status called AIDS. According to [6], HIV/AIDS has killed an estimate of 25 million people globally. The HIV/AIDS epidemic has had a major impact throughout the world. In December 2007, the World Health Organization (WHO) and joint United Nations Program on HIV/AIDS (UNAIDS) estimated that there are 33 million people living with HIV. Most of these people are unaware of their HIV status and infectiousness, and as a result unknowingly contribute to the spread of the infection [5]. The epidemic has disproportionately affected people residing in areas of the world that have fewer resources to combat the disease. WHO [5] also estimated that 2.7 million people were newly infected with HIV in 2007 and greater than 95% of these new infections occurred among persons residing in Low and Middle Income Countries (LMIC). Sub-Saharan Africa accounts for an estimated 22 million cases of HIV/AIDS with an estimated prevalence of 5% in adults ages 15-49. Of these LMIC countries, HIV/AIDS epidemic has often over-burdened the underresourced health care infrastructure worsening the situation.

In the past decade, it is evident that HIV/AIDS-related illnesses and mortality are devastatingly high in some fishing communities. A synthesis surveys conducted since 1992 in ten low or middle-income countries in Africa, Asia and Latin America revealed that HIV/AIDS prevalence among fishers or fishing communities are between 4 and 14 times higher than the National average prevalence rate for adults aged 15-49 years. These considerable rates of HIV/AIDS infection place fisher folks among groups that are usually identified as being at high risk [4].

HIV/AIDS was first described from a Ugandan fishing Village on the shores of Lake Victoria in 1982 [7]. The vulnerability of fishing communities to HIV/AIDS stems from complex interactions, mobility of many fishers, the time they spend away from home, their access to cash income, demographic profile (they are often young and sexually active), low level of education (especially sex education), and readily available commercial sex hawkers in most of the fishing ports and shores of fishing grounds [4]. The sub-ordinate economic and social positions of women in many fishing communities make them even more vulnerable to the infection [8]. It has been reported that due to poverty women fishmongers have become victims of fishermen who demand for sexual favor on top of supplying fish [9, 10]. It is no longer gain saying that people exchange sex for gift or economic gain for their up keep, commercial sex activities are thriving in the area which may be one of consequences of effect of global warming on the water bodies which the desired attention has not been offered [11].

A comparative data for ten low and middle income countries on HIV prevalence among fisher folks with both the wider population and with other groups generally considered fisher folk at high risk of HIV infection [8]. In nine of those countries, fisher folks were more likely to have HIV than the general population, by factors ranging between 4.4 and 14.0. Three of the studies were conducted in Africa. Prevalence rates for fisher folks were 20.3% in the Democratic Republic of Congo (DRC), 30.5% in Kenya and 24.0% in Uganda, representing respectively a 4.8, 4.5 and 5.8 times higher than in the general population [9]. Moreover, in Kenya and Uganda, this incidence was respectively 2.1 and 1.8 times higher than that of truck drivers, another known high-risk group [12]. The actual numbers of people infected were: 44,000 Kenyan fisher folk infected as compared with 8,000 truck drivers, and 33,000 Ugandan fisher folk compared with 5,000 truck drivers [12]. The Kenyan study suggests that rates of HIV infection are even slightly higher for fisher folks than for sex workers. Work elsewhere in Africa (e.g., Senegal, Ghana, Tanzania, Zambia, South Africa, Nigeria and Benin) indicates that many fishing communities have high HIV/AIDS prevalence rates [13]. Cash income, poverty, irregular working hours and being away from home places fishermen in a group with disposable income and time off, that favours the consumption of alcohol and prostitution; the corollary of this is that low-income women are drawn to fish landings or ports precisely because of the opportunities to sell food, alcohol or sex. The chance of being exposed to HIV is increased where a small number of women have unprotected sex with a larger number of men, or vice versa [14]. In places where the amount of fish is limited, women compete intensely for the fish catch and are willing to have "fish for sex", so as to secure desired amount of fish. African traditions and gender inequality places women at risk of exploitation. Augmented with poverty, it becomes difficult for women to insist on safe sex using condom. These contraceptives per se are not available, and the community have low sex knowledge, and therefore fishing communities have limited access to sexual health services [15]. Africa's industrial fisheries and fish processing sub-sectors are also affected by HIV/AIDS, through the loss of skilled labor and high levels of absenteeism due to sickness or compassionate leave to attend funerals. In addition, Allison and Seeley [7] highlight potential impacts on natural resource management, pointing out that: HIV/AIDS undermines the long-term perspective needed for successful co-management in fisheries, whilst deepening and desperate poverty may drive fisher folk towards increasingly short-sighted and unsustainable practices; and indigenous knowledge about resource management may also be lost, along with crucial capacities in universities and public services.

HIV/AIDS in the fisheries sector has much wider impacts too. Mobile and part-time fishing populations, moving in and out of the sector, along with interactions through trade and markets, permit HIV and its impacts to be spread outside the sector. The multiplier effects of the loss of productive labor and declining productivity may affect rural incomes more broadly. HIV/AIDS, moreover, threatens the ability of the fisheries sector to supply fish and fish products to the low-income groups for whom it represents an important and affordable source of animal protein and micronutrients.

HIV/AIDS among Fishers Studies done in Tanzania, Africa, found that fishers were most likely to die from any cause - AIDS or Non-AIDS. In fact, this study found that they were five times more likely to die of AIDS and of other causes than are farmers in the same region [16]. Their entire lifestyle also makes them vulnerable to death due to infection from sexually transmitted diseases, including HIV/AIDS. One study among Malaysian fishers in the state of Kedah reported that 18.1% of the subjects had sex with commercial sex workers, 19.2% used various drugs and 14.4% consumed alcohol, all behaviors which put them at risk of being infected [17].

There is also an immediate need for action to tackle HIV/AIDS in fishing communities: to develop and implement policies; to translate the emerging lessons and approaches into school programs and activities on the ground, making sure that some of the key foundations are in place, including the availability of condoms, VCT centers, workplace policies, and other sensitization and education programs; and to engage with donors, governments, the private sector and communities to harness the commitment and resources needed to fight the problem. Such action implies a variety of roles appropriate to different groups and professions working at different levels, from policy right down to the communities themselves.

## **III.** Model formulation.

In order to formulate the model, the following variables, parameters and assumptions are made. Due to observed significant difference in the way of living, in relation to disease transmission, and a long historical difference in HIV prevalence, we define two distinct groups of people in the society, namely, the Fisher folk and the general community. We denote the fisher folk community by a subscript v and the general community with no subscript. Consider a purely susceptible population at any time t denoted by S(t). Because of the distinct structures, we have  $S(t) = S(t) + S_v(t)$ . With the introduction of HIV/AIDS disease, other epidemiological compartments are created, namely; Infectives  $I(t) = I(t) + I_v(t)$ , these are people exposed to the virus, and without knowledge, progressed to symptomatic infective class. The next compartment is the treatment class  $T(t) = T(t) + T_v(t)$ , which comprises of infectious and symptomatic individuals who seek treatment, and assumed to get effective treatment, and recover fully, so that they move out of this class through death or remain as people living with HIV/AIDS (PLWHA). The complement category who may have not received effective treatment progress to AIDS class  $A(t) = A(t) + A_v(t)$ . This yields a *SITA* compartmental model, which can be represented diagrammatically as shown in Figure 1 below.



Figure 1 SITA model flow chart showing Compartments and respective flow rates

The following assumptions are made; that individuals in each structure are homogeneously mixed, and each has equal opportunity as their counterparts. Also, it is assumed that the recruitment of members in each stratum is known, and their interaction flow rates are instantaneous and constant. With these assumptions and the variables defined, the following model equations are formulated.

## **3.1.** Model Equations

The following is a system of ordinary differential equations, representing the transmission dynamics of the model in Figure 1. Above.

$\frac{dS}{dt} = \lambda - \beta SI - \beta_v \phi SI_v - \mu S$	(1)
$\frac{dI}{dt} = \beta SI + \beta_v S_v I \phi_v - \mu I - \tau I$	(2)
$\frac{d\tilde{T}}{dt} = \tau I - (\mu - \delta)T$	(3)
$\frac{dA}{dt} = \delta T - (\mu + \eta)A$	(4)
$\frac{dS_v}{dt} = \lambda - \beta_v S_v I_v - \beta_v S_v \phi_v I - \mu S_v$	(5)
$\frac{dI_v}{dt} = \beta_v S_v I_v + \beta \phi S I_v - (\mu + \tau) I_v$	(6)
$\frac{dT_v}{dt} = \tau I_v - (\mu + \delta_v) T_v$	(7)
$\frac{dA_v}{dA_v} = \delta_v T_v - (\mu + \eta_v) A_v$	(8)

where:  $\beta$  is the force of infection, with  $\beta_v$  being the vector folk infection rate,  $\lambda$  is the natural recruitment rate to the susceptible group.  $\lambda_v$  is the corresponding recruitment rate to the vector folk group,  $\mu$  natural death rate. It is here assumed to be equal in all compartments,  $\theta$  is the modification parameter accounting for the difference in the infection rate by the infected class.  $\psi$  is the modification parameter accounting for the difference in the infection rate by the treated class,  $\phi$  is the modification parameter accounting for the difference in the infection rate by the treated class,  $\phi$  is the modification parameter accounting for the difference in the infection rate by the treated class,  $\phi$  is the modification parameter accounting for the difference in the infection rate by the AIDS class,  $\tau$  is the rate at which infected class seek treatment,  $\delta$  is the proportion of those under treatment, who will not be cured and therefore progress to AIDS class,  $\omega$  is the rate at which people with AIDS seek treatment.  $\omega_v$  is the corresponding rate for vector folk class,  $\rho$  is the recovery rate from AIDS status, back to treatment class. Note that treatment for HIV is a life-long process and therefore forms PLWHA,  $\omega$  is the rate at which infected individuals progress to AIDS class without seeking treatment, and  $\eta$  is the accelerated death rate due to opportunistic diseases or AIDS.

**3.2 Model Analysis**. The model (1-8) is analyzed for its positivity, boundedness and stability of equilibrium points.

**3.2.1 Positivity and Boundedness.** Since the model relates to human population, it is expected that the solutions remain positive and bounded always. To guarantee this, we define an octagonal positive space, where all solutions stay for all future time. Consider the positive invariant space  $\mathbb{R}^8 = \{S(t) \ge 0, I(t) \ge 0, T(t) \ge 0, A(t) \ge 0, S_v(t) \ge 0, I_v(t) \ge 0, T_v(t) \ge 0, A_v(t) \ge 0\}$  for all initial conditions  $(S(0) = S_0, I(0) = I_0, T(0) = T_0, A(0) = A_0 S_v(0) = S_{v0}, I_v(0) = I_{v0}, T_v(0) = T_{v0}, A_v(0) = A_{v0}) \in \mathbb{R}^+_0 \ge 0$  and time  $t \ge 0$ . Using tis conditions, we have from equation (1),  $\frac{ds}{dt} = \lambda - \beta SI - \beta_v \phi SI_v - \mu S \le \lambda - (\beta I - \beta_v \phi I_v - \mu)S \in \mathbb{R}^+_0$  with the solution

 $S(t) \leq \frac{\lambda}{A} + \left(S_0 - \frac{\lambda}{A_0}\right)e^{-At}$ , where  $A(t) = \beta I(t) + \beta_v \phi I_v(t) + \mu$  and  $A_0 = \beta I_0 + \beta_v \phi I_{v0} + \mu$ . Note that for  $A(t) \geq 0$ , the solution S(t) is always positive and bounded. The same analysis can be shown for positivity and boundedness of equations (2 - 8).

**3.2.2. Fixed Points.** In order to study the qualitative dynamics of the system, we look at the behavioral dynamics locally, around the fixed points. There are two sets of fixed points; disease free equilibrium (*DFE*) and endemic equilibrium points (*EEP*).

**Disease Free Equilibrium**: DFE is defined as the equilibrium point in absence of the disease. At this point, all members of the population is susceptible and no invectives, no treatments and no AIDS cases. Thus we have;  $DFE := (S^0, I^0, T^0, A^0, S_v^0, I_v^0, T_v^0, A_v^0) = \left\{\frac{\lambda}{\mu}, 0, 0, 0, \frac{\lambda_v}{\mu}, 0, 0, 0\right\}$ (9)

#### **Endemic Equilibrium Point**

EEP is the equilibrium, where the pandemic exist in the population, and therefore we have nonzero population of people in each of the epidemiological compartment. EEP is evaluated and obtained as;

$$EEP \coloneqq (S^e, I^e, T^e, A^e, S^e_v, I^e_v, T^e_v, A^e_v)$$

Where,  

$$S^{e} = \frac{(1-\phi_{v})(\mu+\tau)}{\beta(1-\phi\phi_{v})}, I^{e} = \frac{\lambda(\beta_{v}-\sigma\phi\beta)-\mu\sigma(\beta_{v}-\phi\beta)S_{v}^{e}}{\sigma\beta\beta_{v}(1-\phi\phi_{v})S_{v}^{e}}, T^{e} = \frac{\tau I^{e}}{(\mu+\delta)}, A^{e} = \frac{\tau\delta I^{e}}{(\mu+\delta)(\mu+\eta)}, S_{v}^{e} = \frac{(1-\phi)(\mu+\tau)}{\beta_{v}(1-\phi\phi_{v})}, \text{ and } I_{v}^{e} = \frac{\lambda(\sigma\beta-\phi_{v}\beta_{v})-\mu\sigma(\beta-\phi_{v}\beta_{v})S_{v}^{e}}{\sigma\beta\beta_{v}(1-\phi\phi_{v})S_{v}^{e}}, T_{v}^{e} = \frac{\tau I_{v}^{e}}{(\mu+\delta)}, \text{ and } A_{v}^{e} = \frac{\tau\delta I_{v}^{e}}{(\mu+\delta)(\mu+\eta)} \text{ with } \sigma = \frac{\beta_{v}(1-\phi_{v})}{\beta(1-\theta)}.$$

**3.2.3 Stability of Fixed Points:** Stability of the system can be implied by studying the stability of the system around the fixed points. In order to analyze stability of the system, we evaluate stability linearization matrix defined by;

M =	$\begin{pmatrix} -A \\ \beta I \\ 0 \\ 0 \\ \phi I_{\nu} \\ 0 \\ \rho I_{\nu} \end{pmatrix}$	$-\beta S$ $B$ $\tau$ $0$ $-\beta_v S_v \phi_v$ $0$ $0$	$ \begin{array}{c} 0 \\ 0 \\ -(\mu + \delta) \\ \delta \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{array} $	$ \begin{array}{c} 0 \\ 0 \\ -(\mu + \eta) \\ 0 \\ 0 \\ 0 \\ 0 \end{array} $	$0\\ \beta_{\nu}I\phi_{\nu}\\ 0\\ 0\\ -A_{\nu}\\ \beta_{\nu}I_{\nu}\\ 0\\ 0$	$-\beta S\phi$ 0 0 0 -\beta_v S_v $B_v$ $\tau$	$ \begin{array}{c} 0 \\ 0 \\ 0 \\ 0 \\ -(\mu + \delta) \end{array} $	0 0 0 0 0 0 0 0	(10)
	$\begin{pmatrix} 0 \\ 0 \end{pmatrix}$	0	0	0	0	0	δ	$-(\mu + \eta)/$	

Where  $A = \beta I + \beta \phi I_v + \mu$ ,  $B = \beta S + \beta_v S_v \phi_v - \mu - \tau$ ,  $A_v = \beta_v I_v + \beta_v \phi_v I + \mu$ ,  $B_v = \beta_v I_v + \beta S \phi - \mu - \tau$ . Stability of DFE is determined by the sign of the eigenvalues of the stability matrix  $\sigma(M)$ , linearized about DFE.

Stability of Disease Free Equilibrium: The eigenvalues of linearization matrix M in equation (10) about the DFE is given by,  $\Lambda_1 = -\mu$ ,  $\Lambda_2 = (\beta + \phi_v \beta_v) \frac{\lambda}{\mu} - (\mu + \tau)$ ,  $\Lambda_3 = -(\mu + \delta)$ ,  $\Lambda_4 = -(\mu + \eta)$ ,  $\Lambda_5 = -\mu$ ,  $\Lambda_6 = -(\mu + \eta)$  $(\beta_v + \phi \beta) \frac{\lambda}{\mu} - (\mu + \tau_v), \Lambda_7 = -(\mu + \delta_v), \Lambda_8 = -(\mu + \eta).$  Note that all eigenvalues  $\Lambda_s, s = 1, 3, 4, 5, 7, 8$  are clearly negative. For stability, we require that  $\Lambda_2, \Lambda_6$  are also negative. This is satisfied if the following conditions are satisfied.

 $\frac{(\beta + \phi_{\nu} \beta_{\nu})\lambda}{\mu(\mu + \tau)} < 1 \coloneqq R_{0\nu} \text{ and } \frac{(\beta_{\nu} + \phi_{\beta})\lambda}{\mu(\mu + \tau_{\nu})} < 1 \coloneqq R_{0}$ (11) The condition in equation (11) is called the basic reproductive ratio. It is a measure of the ratio of newly infected

individuals over those transferred by other terms. If the ratio is greater than one, the disease replaces itself, and thus remains persistent in the community. If the ratio is less than one, the disease will die off with time.

Stability of Endemic Equilibrium Point: The persistence of the disease in the population refers to continued multiplication and reproduction of the disease in the population. Stability of EEP of this system is derived from linearizing the community matrix M in equation (11) about the EEP. The following eigenvalues are obtained. The arizing the community matrix *M* in equation (11) about the EEP. The following eigenvalues are obtained.  $S^{e} = \frac{(1-\phi_{v})(\mu+\tau)}{\beta(1-\phi\phi_{v})}, I^{e} = \frac{\lambda(\beta_{v}-\sigma\phi\beta)-\mu\sigma(\beta_{v}-\phi\beta)S_{v}^{e}}{\sigma\beta\beta_{v}(1-\phi\phi_{v})S_{v}^{e}}, T^{e} = \frac{\tau I^{e}}{(\mu+\delta)}, A^{e} = \frac{\tau\delta I^{e}}{(\mu+\delta)(\mu+\eta)}, S^{e}_{v} = \frac{(1-\phi)(\mu+\tau)}{\beta_{v}(1-\phi\phi_{v})}, I^{e}_{v} = \frac{\lambda(\sigma\beta-\phi_{v}\beta_{v})-\mu\sigma(\beta-\phi_{v}\beta_{v})S_{v}^{e}}{\sigma\beta\beta_{v}(1-\phi\phi_{v})S_{v}^{e}}, T^{e}_{v} = \frac{\tau\delta I^{e}_{v}}{(\mu+\delta)(\mu+\eta)} \text{ where } \sigma = \frac{\beta_{v}(1-\phi_{v})}{\beta(1-\theta)}. \text{ Clearly, EEP can be expressed in terms of the reproductive number } R_{0} \text{ as; } S^{e} = \frac{\lambda}{\mu R_{0}}$ 

3.2.4. Positivity and boundedness of solutions: Modeling transmission dynamics of infectious disease in human population require that the model represents solutions in a feasible domain. This must be a positive ndimensional space denoted by  $\mathbb{R}^{n+1}_+$ , where n is the number of compartments under study, and the additional dimension is the temporal space with  $0 \le t < \infty$ . The following analysis shows that the solutions are always positive and bounded.

From equation (1), we have 
$$\frac{dS}{dt} \leq -\beta SI - \beta_v \phi SI_v - \mu S \leq -(\beta I + \phi I_v + \mu)S$$
, so that we obtain  
 $\dot{S} \leq -(\beta I + \phi I_v + \mu)S \Longrightarrow \frac{\dot{S}}{S} \leq -(\beta I + \phi I_v + \mu)$ . Integrating both sides gives  
 $lnS(t) \leq -(\beta I + \phi I_v + \mu)t + K \Longrightarrow S(t) \leq S_0 e^{-(\beta I + \phi I_v + \mu)t}$  where  $S_0 = e^K$ .

Clearly, the solution  $S(t) \le S_0 e^{-(\beta I + \phi I_v + \mu)t}$  is positive and bounded for all  $t \ge 0$ . Similarly, equation (2) yields

$$\frac{dI}{dt} = (\beta S + \beta_v S_v \phi_v - \mu - \tau)I \Longrightarrow I(t) = I_0 e^{(\beta S + \beta_v S_v \phi_v - \mu - \tau)t}$$

To guarantee positivity and boundedness, we require that  $(\beta S + \beta_v S_v \phi_v - \mu - \tau) < 0 \implies \beta S + \beta_v S_v \phi_v < \delta S + \delta_v S_v \delta_v < \delta S + \delta_v S_v \phi_v < \delta S + \delta_v S_v \delta_v < \delta S + \delta_v S +$  $\mu + \tau$ . This is the condition of reproductive ratio  $R_0$ .

It is therefore concluded that equation (2) is positive and bounded for all time  $iff R_0 < 1$ . In the same way, equation (3) and (4) reduces to  $T(t) \le T_0 e^{-(\mu+\delta)t}$  and  $A(t) \le A_0 e^{-(\mu+\eta)t}$  respectively. These are clearly positive and bounded for all  $t \ge 0$ .

The analysis of the second set of equations representing Fisher-folk gives the same results. With the prove above, the model represented by equation (1) - (8) is positive and bounded for all time.

**3.2.5. Elasticity:** Elasticity is a measure of how much change a quality of interest is changing with every small perturbation of a parameter. This will help identify the parameters whose small perturbation leads a big change in the quantity of interest. In this study, the quantity of interest is the reproductive ratio. Elasticity of the parameters  $P_i$ , i = 1,2,3,4,... on the reproductive ratio  $R_0$  is defined as  $\frac{1}{P_i} \frac{\partial R_0}{\partial P_i}$ , which is a proportional measure describing the level of influence of each parameter in the interval [0, 1] on the reproductive ratio .

The parameters of interest are  $\mu, \tau, \phi, \phi_{\nu}, \beta, \beta_{\nu}, \lambda$ . The significance of these parameters on the influence on reproductive number  $R_0$  over a measure of 0.5 will be analyzed further. Using the reproductive ratios  $R_{0\nu}$  and  $R_0$  in equation (11), we obtain;

Sensitivity of  $\beta$  on  $R_{0v} = \frac{1}{\beta} \left( \frac{\lambda}{\mu(\mu+\tau)} \right)$  and the sensitivity of  $\beta$  on  $R_0 = \frac{1}{\beta} \left( \frac{\phi \lambda}{\mu(\mu+\tau)} \right)$ . Similarly, the sensitivity of  $\beta_v$  on  $R_0 = \frac{1}{\beta_v} \left( \frac{\lambda}{\mu(\mu+\tau)} \right)$ . Sensitivity of  $\phi$  on  $R_{0v} = \frac{1}{\phi} \left( 0 \right) = 0$  while the sensitivity of  $\phi$  on  $R_0 = \frac{1}{\phi} \left( \frac{\beta \lambda}{\mu(\mu+\tau)} \right)$ . Also the sensitivity of  $\phi_v$  on  $R_{0v} = \frac{1}{\phi_v} \left( \frac{\beta_v \lambda}{\mu(\mu+\tau)} \right)$  and the sensitivity of  $\phi_v$  on  $R_0 = 0$ . The sensitivity of  $\lambda$  on  $R_{0v} = \frac{1}{\lambda} \left( \frac{(\beta+\beta_v\phi_v)}{\mu(\mu+\tau)} \right)$  and the sensitivity of  $\lambda$  on  $R_0 = \frac{1}{\lambda} \left( \frac{(\beta_v+\beta\phi)}{\mu(\mu+\tau)} \right)$ . The treatment rate  $\tau$  has sensitivity on  $R_{0v} = \frac{-\lambda(\beta+\beta_v\phi_v)}{\mu^2(\mu+\tau)^2}$  and that on  $R_0 = \frac{-\lambda(\beta_v+\beta\phi)(2\mu+\tau)}{\mu^2(\mu+\tau)^2}$ , while the natural mortality  $\mu$  has sensitivity on  $R_{0v} = \frac{-\lambda(\beta+\beta_v\phi_v)(2\mu+\tau)}{\mu^3(\mu+\tau)^2}$  and that on  $R_0 = \frac{-\lambda(\beta_v+\beta\phi)(2\mu+\tau)}{\mu^3(\mu+\tau)^2}$ .

# IV. Numerical Solutions and Simulation:

Numerical solutions support analytic solutions and depicts the specific picture of a particular zone or area. In this section, numerical results are presented using data collected from Kisumu, Homabay, Siaya and Busia. The following table summarizes the parameter values as per the data collected.

Table 1. Parameter values from data collected from Samia, Kisumu, Hom	abay and Mbita.	
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No	Symbol	Description	Value
1	λ	Recruitment rate of normal community	0.7
2	$\lambda_{v}$	Recruitment rate of individuals into the fisher folk	0.9
3	μ	Natural death rate	0.00124
4	β	Probability of infectivity given sufficient contact	0.00033
5	$\hat{\beta}_{v}$	Probability of infectivity by fisher folk community	0.00133
6	$\phi$	Number of sexual partners or contact rate of normal population	0.18624
7	$\phi_v$	Number of sexual partners or contact rate of fisher folk population	0.20835
7	τ	Rate of seeking treatment by HIV infected patients	0.024
8	$\tau_v$	Rate of seeking treatment by fisher folk HIV patients	0.021
9	σ	Progression rate of HIV patients to AIDS status	0.023
10	$\sigma_v$	Fisherfolk Progression rate of HIV patients to AIDS status	0.032
11	'n	Accelerated death rate due to HIV/AIDS	0.00124

Using the parameter values in Table 1. above, we obtain the following HIV/AIDS dynamics of the normal population and the fisher folk population.

**3.2.** Basic reproductive number. Using the data in Table 1 above, the basic reproductive number for the general community and the fisher folk  $R_0$  and  $R_{0\nu}$  is given as  $R_0 = 21.4965752$  and  $R_{0\nu} = 21.5412683$  respectively. These values are equal correct to one decimal place, since both represents reproductive ratios when one infective is introduced into a purely susceptible population, in absence of any control intervention strategy.

**3.3.** Sensitivity of parameters to Reproductive Ratios  $R_0$  and  $R_{ov}$ . Before the analysis of parameters, it is necessary to check which of them significantly affects the basic reproductive ratio. This will guide the amount of effects applied to various intervention strategies, which include restriction of interaction, treatment and use of contraceptives. The following table shows the sensitivity values in percentage, of various parameters to reproductive ratios.

No	Parameter	Symbol	Sensitivity to R <sub>0</sub>	Sensitivity to R <sub>0v</sub>
1	Infectivity rate of normal population	β	1.94E+08	0
2	Infectivity rate of the fisher folk population	$\beta_v$	1.94E+08	0
3	Interaction parameter contributing to infection	$\phi$	00	0
4	Increased interaction causing infection	$\phi_v$	0	00
5	Natural recruitment rate into the subpopulation	λ	21.49658	21.54127
6	Natural attrition rate	μ	-1.82E+04	-1.82E+04
7	Treatment rate, assumed to be equal for both groups	τ	-6.85E+05	-6.88E+05

It is clearly seen that some parameters are highly elastic while some are inelastic. The same scenario is represented graphically in the figure below. It is clearly shown on Table 2 and in Figure 2 below that some parameters  $\beta$ ,  $\phi_v$  and  $\tau$  have no effect on the fisher folk population, and the significant parameters to be targeted include;  $\beta_v$ ,  $\phi$ ,  $\lambda$ ,  $\mu$  and  $\tau_v$ .



Figure 2 Effects of Control parameters on Reproductive Ratio

Description of the controls for the said parameters are; prevention of transmission measures for  $\beta_v$ , public health education for  $\phi$  and  $\lambda$ , and treatment parameter  $\tau_v$ . Considering each of the four parameters mentioned, the maximum implementation of treatment will see reproductive ratio only reduced to 1, but achieving 100% treatment is impossible. Public health campaigns reduce interaction or sexual contacts/partners rate  $\phi$  and recruitment to high risk class rate  $\lambda$ . Variation of these parameters are illustrated in Figure 2 below.

The dynamics of HIV/AIDS for the normal population and for the high risk fisher folk population in absence of any control, and with free interaction is illustrated in Figure 3 below. It is noted that from the onset of HIV pandemic, both the normal population and the fisher folk are affected. After the first quarter, wild dynamics gently begin to vanish so that by one year, the dynamics begin to settle and steady state is achieved after two years (700 days).

The parameters with positive effect, and which need to be controlled to as minimum as possible are infective rate  $\beta$ , interaction/number of sexual partners  $\phi$  and recruitment rate  $\lambda$ . On the other hand, the negative effect parameters which need to be increased are treatment rate  $\tau$ , and elimination rate  $\mu$ . However, it is against human rights to accelerate the death rate or cause a person to die earlier, and therefore elimination rate may be used to mean removing from the others, though isolation, quarantine or other forms of withdrawal from interacting with the rest.



Figure 3 HIV/AIDS dynamics in Lake Victoria region for the Normal and the Fisherfolk Population.

With the consideration of fisher folk as HIV vector, the parameters representing the interaction between the two communities can be controlled using the parameter  $\phi_v$ . If adjusted to zero, the dynamics of the new system as shown in Figure 4 below. Variation of public health parameter  $\lambda$  yields the same dynamics as shown in Figure 4.



Figure 4 HIV/AIDS dynamics among normal and fisher folk communities with no interaction

It is here depicted that the disease dynamics of the fisher folk community are prolonged and intensified, while the normal population experience improved health status, with lesser infectivity and accelerated achievement of a higher steady state.

# V. Conclusion

The target of every public health worker is to reduce the reproductive ratio to less or equal to one, especially for the high risk fisher folk population. This can be achieved through a combination of various strategies. From simulation results, it is noted that increasing treatment rate to a minimum of  $\tau = 0.67$ , and increasing public education campaigns so as to reduce infectivity rate by 50% to  $\beta_v = 0.000665$ , which is still twice as high as infectivity rate of the normal population  $\beta = 0.00033$ . Public health education is also expected to influence people to be faithful and have one sexual partners  $\phi = 1$ , while recruitment rate to high risk class is reduced to zero  $\lambda = 0$ .

There is need to consistently have a repeat of treatment and public health campaigns as much as the budget allows, because of complacency. People tend to forget and go back to their old lifestyle, hence leading to an upsurge of the epidemic. Alternative fishing methods and control of fish market, by putting up structures and policies or creating fish brokers, private middle men or government intermediaries like the Kenya National Trading Cooperation (KNTC) will bridge the gap between the fisher folk and the rest of the public, thus reducing direct interaction and use of sex in exchange of fish.

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