NIGERIAN STATISTICAL ASSOCIATION

2006 CONFERENCE PROCEEDINGS

THEME:

STATISTICAL SUPPORT FOR MEASURING THE MILLENNIUM DEVELOPMENT GOALS (MDGS) IN NIGERIA.

VENUE:

ROYAL TROPICANA HOTEL, KANO, KANO STATE.

DATE:

13th - 15th September, 2006.

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MATHEMATICAL MODELLING OF THE EFFECT OF PUBLIC HEALTH CAMPAIGN AGAINST THE SPREAD OF HIV/AIDS EPIDEMICS

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ABSTRACT

Mathematical modeling is increasingly being applied to interpret and predict the dynamics and control of infectious diseases. Applications include predicting the impact of vaccination strategies against common infections such as measles and malaria and determining optimal control strategies against HIV and vector-borne diseases.
In this paper, differential equations were employed to model the dynamic spread of HIV/AIDS epidemics and the intervention campaigning programmes against the future pandemic of HIV/AIDS. The model will provide a veritable tool to estimating the level of effectiveness of mounting advertising campaigns against disease spread and adverse health outcomes within a given population. The goal of campaign programmes are to change people's behavior in order to minimize the spread of AIDS, to reduce the percentage of people who smoke, to cut alcohol and drug abuse, Indian helm and so on.

Key words: differential equations, limiting factors, HIV/AIDS dynamic, SIR model

1. INTRODUCTION

Unlike infectious diseases that were first recognized in the 20th century, AIDS has had not only the most profound effect on human illness and death, it ended the developed world's complacency about infectious diseases. Caused by HIV, AIDS is, as far as we know, always fatal, even with effective therapy. In the last 50 years, HIV went from being maintained primarily, if not exclusively, in sooty mangebeys (HIV-2) and chimpanzees (HIV-1) to being the etiologic agent of a worldwide pandemic.(see Gao F, et al., 1999; Korber B, et al., 2000; and Robertson DL, et. al.1999). AIDS was not recognized as a specific disease until 1980, and HIV was not identified as the etiologic agent until 1983. Nevertheless, an estimated 16 million persons have died from AIDS worldwide with 50 million currently infected with HIV.

In this paper, a differential equation was employed to describe the population dynamics of HIV/AIDS. A simple time dependent factor was later incorporated into the spread model as a result of the effect of intervention campaign programmes against spread of a disease or any other anti-social vices. This model can be used to provide feedback information to the government or sponsoring agency and to measure the relative effectiveness of such campaign. It can also be used to estimated the proportion of the susceptible population (risk population) against its attack, therefore minimize the cost committed to HIV/ AIDS programmes. . The goal of campaign programmes are to change people's behavior in order to minimize the spread of AIDS, to reduce the percentage of people who smoke, to cut alcohol and drug abuse, and so on

This paper is organized in the following manner; it firstly explains the dynamics of HIV/AIDS epidemics. Secondly, it gives deterministic model for the disease spread, and discusses factor limiting the spread of HIV/AIDS. A time dependent term was later incorporated into the model to quantify the intervention campaigns against its spread. It illustrates the model using simple evaluation and finally concludes.

In this paper, a susceptible population will be assumed. This is the group of people who get the disease, like potential smoker, and so on. The percentage of susceptible that are affected (the percentage who become infected with the disease, who begin smoke, etc) is the risk population denoted by y(t), which model estimates.

2. HIV DYNAMICS

When an infectious disease is first introduced into a population, it has the greatest opportunity to spread because all hosts are susceptible. Thus, suppose a single infected person is introduced into a wholly susceptible population, the maximum opportunity exists for producing secondary infections, the number of which is traditionally designated as R_0 , Gao F, et al (1999).). Although R_0 is a measure of the potential of a disease to spread in a population, it not a measure of the rate at which that disease will spread.

One way to measure the rate at which a disease spreads in a wholly susceptible host population is used by demographers to represent the geometric expansion rate of populations, the intrinsic rate of increase, r ($r \geq 0$). N persons at time 0 become Ne^{rt} persons at time t, where e is the base of the natural logarithm (if N were an amount of money, r would be the compound interest rate; in this case, r is the intrinsic rate of increase in the number of infected persons). With age structure in the model, a certain "settling out" period occurs in which the ratios of numbers of infections at different stages oscillate. As these oscillations decay, r approaches its steady state value, which can be calculated from the rate of change in any of the age categories of the Age of Infection (Aol) model proposed by Levin BR, et. al.(1996) .

The long duration of infection is important in understanding the intrinsic rate of increase of HIV and its dissemination through a population. During the epidemic phase of the disease, when there are many susceptible hosts and the number of new infections is increasing geometrically, the contribution of transmissions occurring at later stages of the infection to the spread of the virus is severely discounted Thus, new infections transmitted by recently infected persons, at initial stage, contribute much more to the spread of HIV than infections from persons in final stage (say 12 years later). (for detail see Levin BR, et. al.(1996) and May RM, et.al (1990)).

3 DETERMINISTIC MODELS FOR HIV/ AIDS DYNAMICS

Consider a simple differential equation proposed by Muench (1959) for spread of a disease:

$$\frac{dy}{dt} = r(1 - y), \quad 0 \leq y \leq 1 \qquad (1)$$

where r is the infection rate, usually a positive constant. Note that as y increases, the rate of increase of y decreases. The solution to this linear equation is

$$y(t) = 1 - [1 - y(0)]e^{-rt} \qquad (2)$$

As t approaches infinity, e^{-rt} approaches zero. Thus $y(t) \rightarrow 1$; that is , eventually all the susceptible become infected. The data generated using equation(2) is shown in the appendix 1

To illustrate this principle, let's use the model by Levin BR, et. al.(1996), and assume that all transmission of the virus is confined to just one of the four stages. A rate of increase of HIV of 0.50 per year (HIV infections doubling every 1.4 years) would require (a) 1.1 secondary infections if transmission occurred solely in stage 1 (between weeks 6 and 12 after the host is infected in this example); (b) 5.2 secondary infections if transmission occurred solely during the asymptomatic period will require an average of 10 years; and (c) 72 secondary infections if transmission occurred solely during the period after the onset of AIDS (at an Aol between 10 and 12 years in this examples.

This conclusion has a number of implications, the most immediately practical of which is that public health and education procedures to control the epidemic will fail if they are based on using serologic test results to identify infected persons. That is infected persons may well have transmitted the virus before they seroconverted.

4. FACTORS LIMITING THE RATE OF SPREAD OF HIV/AIDS

Although at least 50 million persons have been infected with HIV, the human population (more than 6 billion persons) consists almost entirely of susceptible persons, and the global rate of increase in new HIV infections does not appear to have abated. However, unlike the case with influenza and measles, considerable geographic and cultural variation exists in the epidemiology of HIV/AIDS. In effect, the HIV pandemic has been largely restricted to subpopulations—risk groups within which the likelihood of infection is substantially greater than that in the population at large, e.g., gay men, injection drug users, and sex workers, their patrons, and their spouses (or other sex partners).

It seems reasonable as well as hopeful to expect that the rate of increase in new HIV infections will decline in a number of different populations. Several processes can be undertaken to account for the declines in the incidence of new HIV infections and reductions in the rate of spread of this virus. Such programmes or campaigns may reflect the efficacy of public health measures, education programs leading to more prudent sexual e.g. use of condom and needle use behaviour. Chemotherapy can also reduce the transmissibility of the virus. The rate of infection can possibly be brought down through evolution, which makes these viruses less transmissible or humans less susceptible to HIV infections. More so, it may well be that the dominant reason for observed declines in the rate of spread of this retrovirus lies in the progression (and confinement) of the epidemic in particular subpopulations (risk groups). The reductions in the spread of HIV in these subpopulations could be due to the saturation of the pool of susceptible hosts in these groups rather than to successful intervention programmes or behavioral changes (see Koopman J. S, Pollock SM, et al.(1997)).

5. MODEL WHEN INCORPORATING INTERVENTION CAMPAIGN FACTOR

Mathematical models have been used to address several questions concerning the epidemiologic and evolutionary future of HIV/AIDS in human populations. It has been shown that when HIV first enters a human population, and for many subsequent years, the epidemic is driven by early transmissions, possibly occurring before donors have sero-converted to HIV-positive status. Also, evolution of the virus or to the efficacy of intervention, education, and public health measures are likely to reduce the rate of spread of the disease. HIV chemotherapy will reduce the transmissibility of the virus and treating individual patients can reduce the frequency of HIV infections and AIDS deaths in the general population.(for more detail see Koopman JS et.al 1997).

To avoid this dire outcome, public health officials launch a series of advertising campaigns with all designed to increase the public awareness and suggest ways to protect against the disease (to persuade people not to smoke, not to involve in anti-social vices etc) . If the campaign is mounted at time t and effective from then on, then the rate of increase of y(t) is reduced by some amount α from time t onwards, and the differential equation modeling in (1) becomes

$$\frac{dy}{dt} = r(1-y) - \alpha H(t-t_1), \quad 0 \le y \le 1 \qquad (3)$$

where α is effective rate of the campaign, taking to be a positive constant and $0 \le \alpha \le r$,

To proffer a solution to equation (3), apply Laplace transforms:

$$sL\{y\} - y(0) = \frac{r}{s} - r\,L\{y\} - \frac{\alpha e^{-t_1 s}}{s}$$

$$(s+r)L\{y\} - y(0) = \frac{\beta}{s} + y\{0\} - \alpha e^{-t_1 s}$$

$$L\{y\} = \frac{r}{s(s+r)} + \frac{y\{0\}}{s+r} - \frac{\alpha e^{-t_1 s}}{s+r}$$

Using partial fraction

$$\frac{r}{s(s+r)} = \frac{1}{r}\left(\frac{1}{s} - \frac{1}{s+r}\right)$$

so

$$L\{y\} = \frac{1}{r}\left(\frac{1}{s} - \frac{1}{(s+r)}\right) + \frac{y\{0\}}{s+r} - \frac{\alpha e^{-t_1 s}}{r}\left(\frac{1}{s}\right) + \frac{\alpha e^{-t_1 s}}{r(s+r)}$$

Taking the inverse Laplace of $e^{-tI\,s}/s = H(t-t_1)$ *by equation (3), we obtain*

$$L\{H(t-t_1)e^{-r(t-t_1)}\} = e^{-t_1 s} L(e^{-rt}) = \frac{e^{-t_1 s}}{(s+r)}$$

Thus, $L^{-1}\{e^{-r(t-t_1)}/(s+r)\} = H(t-t_1)e^{-t_1 s} e^{-r(t-t_1)}$ and

$$y(t) = 1 - e^{-rt} + y(0)\, e^{-rt} - \alpha H(t-t_1) + \alpha H(t-t_1) e^{-r(t-t_1)}$$

$$= 1 - \frac{1}{r} - y(0)\, e^{-rt} - \frac{\alpha}{r} H(t-t$$

Figure 1. The spread of HIV/AIDS in a steady population of 10,000. In this figure, HIV-positive includes all persons infected with this virus, but not manifesting the symptoms of AIDS....

Figure 1, A

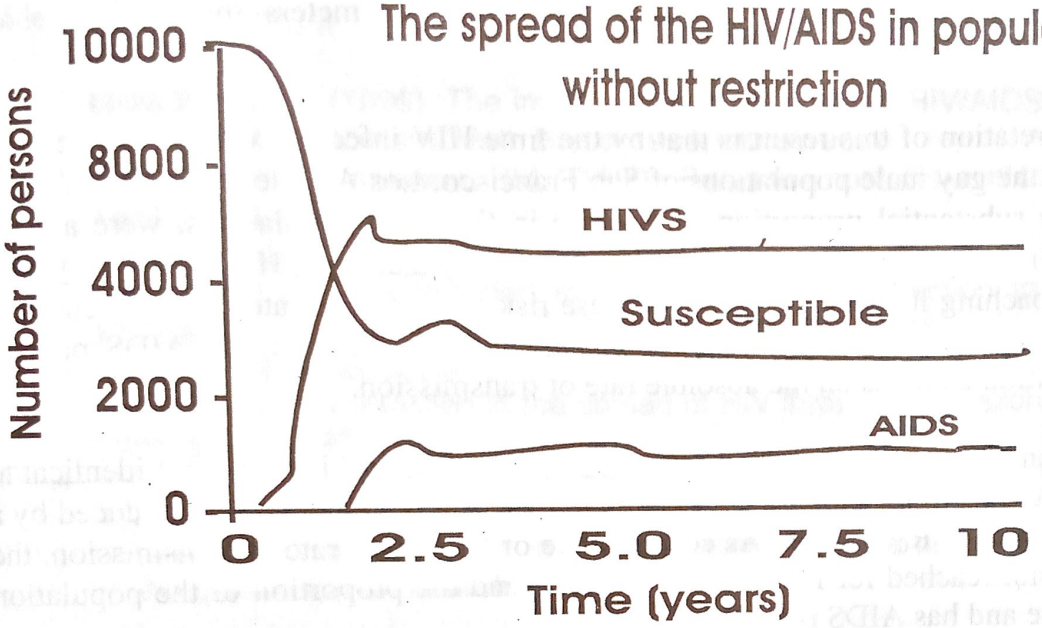

Figure 1, B

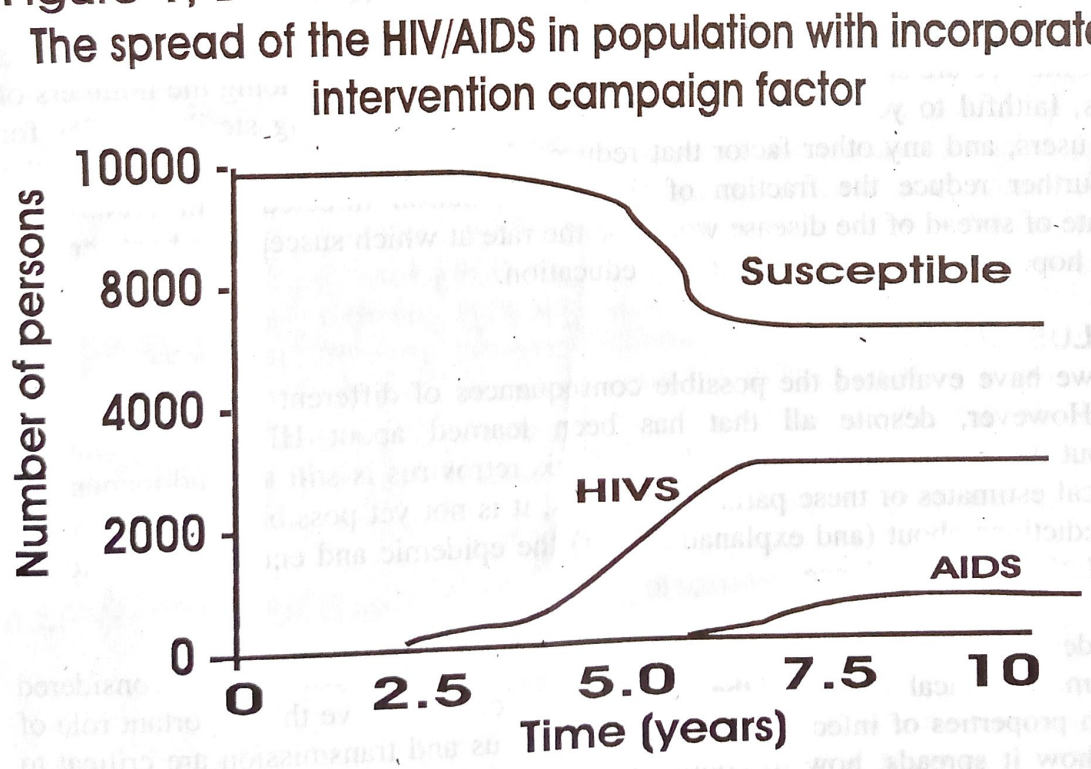

Figure 1A shows how the densities of susceptible hosts, HIV-positive persons without AIDS, and persons with AIDS change over the course of time in a population with an initial number of 10^4 susceptible hosts and two HIV-positive persons at the earliest age of

the infection (week 1). The virus rapidly spreads through the host population who exhibit no sign of AIDS for the first 10 years. By the time the first AIDS cases are recognized, more than half of the original population of 10,000 hosts are infected with the virus. Because of the relative dearth of susceptible hosts, the rate of spread of HIV to new hosts has already declined. Eventually, equilibrium is achieved and the infection maintains a steady state. In this endemic phase, the densities of susceptible hosts, HIV-positive hosts not manifesting the symptoms of AIDS, and AIDS patients level off. With these parameters, this endemic phase is reached in about 30 years.

A historical interpretation of this result is that by the time HIV infection was recognized as a specific disease in the gay male populations of San Francisco, Los Angeles, and New York in the early 1980s, a substantial proportion of persons in those subpopulations, were already infected with the virus, Koopman JS,et.al (1997). Moreover, by that time, HIV/AIDS may have already been approaching its endemic phase in these risk groups. The rate at which endemic phase is approached as well as the frequency of HIV-positive persons and AIDS patients within a subpopulation depends on the absolute rate of transmission.

This is illustrated in **Figure IB**, the parameters used for generating this figure are identical to those in **Figure 1A**, except for the maximum rates of increase, which have been reduced by a factor of two, $R_{01} = R_{02} = R_{03} = 0.50$. As consequence of this lower rate of transmission, the endemic phase is not reached for more than 100 years, and the proportion of the population that is HIV-positive and has AIDS is markedly reduced.

The simple explanation of these results is that an epidemic cannot continue forever because the number of uninfected hosts eventually declines, which stops the expansion of infections. At equilibrium, the fraction of infected versus uninfected hosts depends on various parameters that may be are subsumed in the model. Using condoms, reducing the numbers of sexual partners, faithful to your partners (in case of couples), providing sterile needles for injection drug users, and any other factor that reduces the likelihood of transmission of the virus would further reduce the fraction of the subpopulation infected with HIV. Also affecting the rate of spread of the disease would be the rate at which susceptible hosts enter a risk group. We hope this rate can be reduced by education.

7. CONCLUSION

In this paper, we have evaluated the possible consequences of different properties of HIV transmission. However, despite all that has been learned about HIV/AIDS, existing knowledge about the biology and epidemiology of this retrovirus is still too rudimentary to employ empirical estimates of these parameters. Thus, it is not yet possible to make robust, quantitative predictions about (and explanations for) the epidemic and endemic behavior of HIV or the evolution of its virulence.

Towards these desired ends, however, it is believed that the mathematical models considered here and other mathematical models of the epidemiology of HIV serve the important role of revealing which properties of infections with this retrovirus and transmission are critical to understanding how it spreads, how to control that spread, and to predict the direction of evolution of its virulence.

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Appendix;: Data generated for equation (2) at different rate of infection

y (0) =10,000 t = 0 e=2.7178

$y(t) = 1 - [1 - y(0)]e^{-rt}$

t(weeks)	r =0.001	r=0.02	r=0.05
0	10000	10000	10000
1	9990.006	9802.007	9512.343
5	9950.13	9048.469	7788.229
10	9900.508	8187.489	6065.7
s20	9802.007	6703.53	3679.427
45	9560.019	4066.29	1054.887
52	9493.339	3535.193	743.6615
100	9048.469	1354.217	68.37273
250	7788.229	68.37273	1.037263
300	7408.441	25.78504	1.003059
520	5945.611	1.304294	1