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A Density–Dependent Epidemiological Model for the Spread of Infectious Diseases

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Abstract - A population consists of healthy (H), infected (I) and recovered (R) individuals at any time (t). The infected individuals I(t) are capable of infecting the healthy individuals (H(t)) but not the recovered ones. A density dependent population model is used for the healthy individuals within the environment with maximum carrying capacity M. The first model developed and used in this paper consists of a coupled system of differential equations. The model can be used to predict an outbreak or massive epidemic through its infection rate b. The second model is a simplification of the first model assuming a density-dependence structure in all the compartments or states of the disease. It is shown that with such simplification, it is possible to predict the trajectory of the infectious disease over time given the rate of population growth of the susceptible or healthy population, the rate of infection and the rate of recovery from the infectious disease.

Due to the density-dependence structure of both models, the birth and death rates are already implicitly factored into the epidemic model. Data for the spread of HIV across different countries of the world were used to illustrate the usefulness of the model.

Keywords - epidemic, non-linear differential equations, logistic curve, variation of parameters, chaotic dynamical systems, human immune-deficiency virus (HIV)

INTRODUCTION

Models of the spread of infectious diseases have always been considered useful in epidemiology. In particular, mathematical epidemiologic models allow medical practitioners to predict the onset of an outbreak, establish control measures to prevent such an outbreak and, more importantly, understand the dynamics of disease spread. Since the bubonic plague of the 19th century, man has become more wary of infectious diseases. To date, infectious diseases brought about by more virulent viruses like the AH1N1 virus, the HIV virus, and others are of greater interest to mankind particularly with the unsettling knowledge that human population increases while stretching earth's resource capacity to carry such a load.

Most mathematical models for the spread of diseases are stochastic. The McKendrick (1978) model is a classic example of such a model when the spread of the disease is modeled as a spatio-temporal Poisson process. Perhaps, the most common epidemic model is the Kermack – McKendrick model (*from Epidemic Modeling by Gani, 1982)*. In this model, there are no births, the population size N is fixed, and there are three states: susceptible (S), infected (I) and removed (R). People move from being susceptible to being infected and then to being removed.

The main problem with the Kermack – McKendrick model, even if it is the one often used in practice today, is the fact that it assumes a fixed population N and no births occur. It is similar to assuming a population that remains the same and where every infected individual

eventually recovers. On the other hand, models which account for the realities in epidemiology often result to extremely complicated mathematics, as will be later seen in this paper. A more realistic model must take into account the fact that population growth fluctuates (even if growing) and is naturally controlled by the maximum carrying capacity of the environment. Density-dependent models, generally, account implicitly for the births and deaths that take place in the population of organisms within a limited environment without explicit reference to both birth and death rates of the organisms. Such a model can, for instance, be used in reference to both the human (host) population and the vector population growth trajectories. The model can also be used to describe the movement from one epidemiological stage to another of individuals for directly transmissible diseases.

To this end, assume a maximum carrying capacity of M individuals in an environment. We introduce the basic logistic population model (*Anderson, 1982*) by letting H(t) be the population of healthy individuals at time t, I(t) the population of infected individuals and R(t) is the number of recovered individuals.

Let $x(t) = \frac{H(t)}{M}, y(t) = \frac{I(t)}{M}$ and $z(t) = \frac{R(t)}{M}$. It is clear that

$$0 < x(t) \leq 1,\ 0 < y(t) \leq 1,\ 0 < z(t) \leq 1.$$

The logistic model for population growth is:

$$(3)\ \frac{dx}{dt} = ax(1-x) \qquad ,a > 0 \text{ is a constant}$$

whose solution is:

$$(4)\ x(t) = \frac{K\exp(at)}{1+k\exp(at)}.$$

The discrete counterpart of (3) is a source of rich dynamical behavior:

$$(4)\ X_t = ax_{t-1}(1-x_{t-1}).$$

For $0 < \alpha \leq 1$, population becomes **extinct**; for $1 < a < 3.57$ the population tends to a **stable limit** while for $\alpha > 3.57$, the population experiences a series of busts and booms appearing like a **chaotic** process. We wish to exploit the variety of dynamical behaviors in the formulation of our epidemiological model.

BRIEF LITERATURE REVIEW

Epidemiological models for large populations require the use of deterministic or compartmental mathematical models. For example, this is done in the case of tuberculosis and HIV infection modeling. In the deterministic model, individuals in the population are assigned to different subcategories or compartments, each representing a specific stage of the epidemic. Letters such as M, S, E, I, and R are often used to represent different stages.

Individuals in a population move from one stage to another stage of the epidemic model. The rate at which such individuals move from one class to another are mathematically expressed as derivatives or rates of change with respect to time, hence the model is formulated using differential equations. While building such models, it must be assumed that the population size in a compartment is differentiable with respect to time and that the epidemic process is deterministic. In other words, in deterministic models, the notion of randomness or stochasticity does not factor in the model formulation, only the history of the movement of individuals from one stage to another (Brauerand Castillo-Chavez, 2001).

Terminology

- We survey some of the most common forms of deterministic epidemic model. In order to better appreciate the presentation, the following is a summary of the notation used in this paper:M -- Passively Immune Infants
- S -- Susceptibles
- E -- Exposed Individuals in the Latent Period

- I -- Infectives
- R -- Removed with Immunity
- β -- Contact Rate
- μ -- Average Death Rate
- B -- Averate Birth Rate
- $1/\varepsilon$ -- Average Latent Period
- $1/\gamma$ -- Average Infectious Period
- R_0 -- Basic Reproduction Number
- N -- Total Population
- f -- Average Loss of Immunity Rate of Recovered Individuals
- δ -- Average Temporary Immunity Period

The SIR Model

The simplest of all epidemiological models is the Kermarck-McKendrick Model. In 1927, W. O. Kermack and A. G. McKendrick developed a simple model in which only three categories or compartments are involved in a fixed population: susceptible: S(t), infected, I(t), and recovered, R(t).The compartments used for this model consist of three classes:

• S(t) is used to represent the number of individuals not yet infected with the disease at time t, or those susceptible to the disease

• I(t) denotes the number of individuals who have been infected with the disease and are capable of spreading the disease to those in the susceptible category

• R(t) is the compartment used for those individuals who have been infected and then recovered from the disease. Those in this

category are not able to be infected again or to transmit the infection to others.

The flow of this model is as follows:

Susceptible - Infected - Recovered or S I R.

Using a fixed population, N = S(t) + I(t) + R(t), Kermack and McKendrick derived the following equations:

The number of susceptible individuals decrease in proportion to the number of susceptible present who are in contact with infectious individuals:

$$\frac{dS}{dT} = -\beta SI$$

The number of infected individuals change according to the number of susceptible individuals infected (βSI) minus the number of individuals who recover from the disease (γI)

$$\frac{dI}{dT} = \beta SI - \gamma I$$

The number of individuals who recover from the disease is a fraction of the number of infected individuals (γI):

$$\frac{dR}{dT} = \gamma I$$

Certain assumptions were made in the formulation of these equations: First, individuals in the population are assumed to be equally likely to contract the disease depending on a rate β, which is considered the contact or infection rate of the disease. Therefore, an infected individual makes contact and is able to transmit the disease with βN others per unit time and the fraction of contacts by an infected with a susceptible is S/N. The number of new infections in unit time per infective then is $(\beta N)(S/N)$, giving the rate of new infections (or those

leaving the susceptible category) as $(\beta N)(S/N)I = \beta SI$ (Brauer and Castillo-Chavez, 2001). For the second and third equations, consider the population leaving the susceptible class as equal to the number entering the infected class. However, a number equal to the fraction (γ which represents the mean recovery rate, or $1/\gamma$ the mean infective period) of infectives are leaving this class per unit time to enter the removed class. These processes which occur simultaneously are referred to as the *Law of Mass Action*, a widely accepted idea that the rate of contact between two groups in a population is proportional to the size of each of the groups concerned (Daley andGani, 2005). Finally, it is assumed that there are no births nor deaths during the time scale of the epidemic. For a long time, this basic epidemiological model was used in analyzing the spread of infectious diseases until other more precise and realistic models were formulated. However, even the new models of the spread of infectious diseases base their initial formulation from this classic epidemiological model.

The SIR Model with Births and Deaths

It is possible to modify the basic McKendrick model to incorporate births and deaths. Using the case of measles, for example, there is an arrival of new susceptible individuals into the population. For this type of situation births and deaths must be included in the model. The following differential equations represent this model:

The number of susceptible individuals still decrease by the same amount as before but there is an arrival of new susceptible from the population i.e. from N-S:

$$\frac{dS}{dT} = -\beta SI + \mu(N - S)$$

Consequently, the rate at which the number of infected individuals change is the same as in the old classic model but decreases by a factor μI:

$$\frac{dI}{dT} = \beta SI - \gamma I - \mu I$$

Finally, the rate at which the number of recovered individuals change is the same as before but decreased by a factor μR:

$$\frac{dR}{dT} = \gamma I - \mu R$$

The SIS Model with Births and Deaths

The SIS model is an epidemiological model in which the population, once infected, does not gain any immunity from the disease. The SIS model can be easily derived from the SIR model by simply considering that the individuals recover with no immunity to the disease, that is, individuals are immediately susceptible once they have recovered.

Susceptible - Infected - Susceptible

It follows that the system of differential equations governing SIS is the same as SIR but with the last equation removed. There are only two compartments in this model, either one is susceptible to the disease or he is already infected:

$$\frac{dS}{dT} = -\beta SI + \mu(N - S) + \gamma I$$
$$\frac{dI}{dT} = \beta SI - \gamma I - \mu I$$

The SIRS Model

This model is simply an extension of the SIR model as we will see from its construction.

Susceptible-Infected-Recovered-Susceptible

The only difference is that it allows members of the recovered class to be free of infection and rejoin the susceptible class. For instance, this will be the model for infectious diseases like dengue where immunity from one serotype of the dengue flavivirus does not guarantee immunity from the other serotypes.

$$\frac{dS}{dT} = -\beta SI + \mu(N - S) + fR$$
$$\frac{dI}{dT} = \beta SI - \gamma I - \mu I$$
$$\frac{dR}{dT} = \gamma I - \mu R - fR$$

Models with More Compartments

The SEIS Model

The SEIS model takes into consideration the exposed or latent period of the disease, giving an additional compartment, E(t).

Susceptible-Exposed-Infected-Susceptible

In this model an infection does not leave a long lasting immunity thus individuals that have recovered return to being susceptible again, moving back into the S(t) compartment. The following differential equations describe this model:

$$\frac{dS}{dE} = B - \beta SI - \mu S + \gamma I$$
$$\frac{dI}{dT} = \beta SI - (\varepsilon + \mu)E$$
$$\frac{dT}{dT} = \varepsilon E - (\gamma + \mu)I$$

The SEIR Model

Many diseases have what is termed a latent or exposed phase, during which the individual is said to be infected but not infectious.

Susceptible-Exposed-Infected-Recovered

In this model the host population (N) is broken into four compartments: susceptible, exposed, infectious, and recovered, with the numbers of individuals in a compartment, or their densities denoted respectively by S(t), E(t), I(t), R(t), that is N = S(t) + E(t) + I(t) + R(t).

$$\frac{dS}{dE} = B - \beta SI - \mu S$$
$$\frac{dI}{dT} = \beta SI - (\varepsilon + \mu)E$$
$$\frac{dR}{dT} = \varepsilon E - (\gamma + \mu)I$$
$$\frac{dT}{dT} = \gamma I - \mu R$$

The MSIR Model

There are several diseases where an individual is born with a passive immunity from its mother.

Passive Immunity-Susceptible-Infected-Recovered

To indicate this mathematically, an additional compartment is added, M(t), which results in the following differential equations:

$\frac{dM}{dT} = B - \delta MS - \mu M$
$\frac{dS}{dT} = \delta MS - \beta SI - \mu S$
$\frac{dR}{dT} = \beta SI - \gamma I - \mu I$
$\frac{dT}{dT} = \gamma I - \mu R$

The MSEIR Model

For the case of a disease, with the factors of passive immunity, and a latency period there is the MSEIR model.

Passive Immunity-Susceptible Exposed-Infected-Recovered

$\frac{dM}{dT} = B - \delta MS - \mu M$
$\frac{dE}{dT} = \delta MS - \beta SI - \mu S$
$\frac{dI}{dT} = \beta SI - (\varepsilon + \mu)E$
$\frac{dR}{dT} = \varepsilon E - (\gamma + \mu)I$
$\frac{dT}{dT} = \gamma I - \mu R$

Reproduction Number

From every epidemiological model one can compute an epidemiological quantity called basic reproduction number or BRN. This is a threshold quantity which determines whether an epidemic occurs or the disease simply dies out. The BRN is defined as the number of secondary infections caused by a single infective introduced into a population made up entirely of susceptible individuals ($S(0) \approx N$) over the course of the infection of this single infective. This infective individual makes βN contacts per unit time producing new infections with a mean infectious period of $1/\gamma$. Therefore, the basic reproduction number is

$R_0 = BRN = (\beta N)/\gamma$

This value quantifies the transmission potential of a disease. If the basic reproduction number falls below one ($R_0 < 1$), i.e. the infective may not pass the infection on during the infectious period, the infection

dies out. If $R_0 > 1$ there is an epidemic in the population. In cases where $R_0 = 1$, the disease becomes endemic, meaning the disease remains in the population at a consistent rate, as one infected individual transmits the disease to one susceptible (Trottier and Philippe, 2001).
In cases of diseases with varying latent periods, the basic reproduction number can be calculated as the sum of the reproduction number for each transition time into the disease. An example of this is tuberculosis. (Blower, Mclean, Porco, Small, Hopewell, and Sanchez, 1995) calculated from a simple model of TB the following reproduction number:

$$R_0 = R_0^{\text{FAST}} + R_0^{\text{SLOW}}$$

In their model, it is assumed that the infected individuals can develop active TB by either direct progression (the disease develops immediately after infection) considered above as FAST tuberculosis or endogenous reactivation (the disease develops years after the infection) considered above as SLOW tuberculosis.

Other Considerations within Compartmental Epidemic Models

Vertical Transmission

In the case of some diseases such as AIDS and Hepatitis B, it is possible for the offspring of infected parents to be born infected. This transmission of the disease down from the mother is called Vertical Transmission. The influx of additional members into the infected category can be considered within the model by including a fraction of the newborn members in the infected compartment (Brauer and Castillo-Chavez, 2001). Hyman (1997) also developed an HIV model based on vertical transmission.

Vector Transmission

Diseases transmitted from human to human indirectly, i.e. malaria spread by way of mosquitoes, are transmitted through a vector.
In these cases, the infection transfers from human to insect and an epidemic model must include both species, generally requiring many more compartments than a model for direct transmission. For more information on this type of model see the reference *Population Dynamics of Infectious Diseases: Theory and Applications*, by R. M. Anderson (Brauer

and Castillo-Chavez, 2001)

Others

Finally, epidemiologists can develop more detailed and realistic models by considering other peculiar characteristics of the disease. Some of these considerations may include: (taken from *Mathematical Models in Population Biology and Epidemiology by Fred Bauer and Carlos Castillo-Chávez, 2001*):

- Nonhomogeneous mixing
- Age-Structured populations
- Variable infectivity
- Distributions that are spatially non-uniform
- Diseases caused by macroparasites
- Acquired immunity through vaccinations

FORMULATION OF THE MODEL

Let M be the maximum carrying capacity of an environment and as before:

(5) $x(t) = \frac{H(t)}{M}, y(t) = \frac{I(t)}{M}, z(t) = \frac{R(t)}{M}$

where $H(t), I(t)\ and\ R(t)$ are the number of healthy, infected and recovered individuals at time t respectively.

The rate at which the number of healthy individuals change depends on the number present based on the logistic model plus the number of individuals who recover less the number of individuals who get infected at that time: Moreover, the number of infected individuals change as a fraction of the healthy ones while the number of those who recover is a fraction of those who get infected.

(6) $\frac{dx}{dt} = a\,x(1-x) + z - y$

$\frac{dy}{dt} = b\,x(t)$

$\frac{dz}{dt} = c\,y(t)$

The system (6) defines our model. Differentiating once, we obtain:

(7) $\frac{d^2x}{dt^2} = a\,\frac{dx}{dt} - 2a\,x(t)\frac{dx}{dt} + \frac{dz}{dt} - \frac{dI}{dt}$

$$\frac{d^2x}{dt^2} = a\,\frac{dx}{dt} - 2a\,x(t)\frac{dx}{dt} + c\,y(t) - b\,x(t)$$

Differentiating again and substituting $\frac{dy}{dt}$:

(8) $\frac{d^3x}{dt^3} = a\,\frac{d^2x}{dt^2} - 2a\left(\frac{dx}{dt}\right)^2 - 2a\,x(t)\frac{d^2x}{dt^2} + bc\,x(t) - b\frac{dx}{dt}.$

We can rewrite (8) in differential operator form:

(9) $(D^3 + bD)\,x(t) = a\left(1 - 2x(t)\right)\frac{d^2x}{dt^2} - 2a\left(\frac{dx}{dt}\right)^2 + bc\,x(t).$

Let $Q(t) = bc\,x(t) - 2a\left(\frac{dx}{dt}\right)^2 + a\left(1 - 2x(t)\right)\frac{d^2x}{dt^2}.$

Equations (9) have a solution x(t) which can be written as:

$$X(t) = X_c + X_p$$

where X_c is the complementary solution to $(D^3 + bD)\,x(t) = 0$ and X_p is a particular solution to $(D^3 + bD)\,x(t) = Q(t)$. We attempt to use the method of variation of parameters.

First, the complementary solution is easy to obtain:

(10) . $X_c(t) = C_1 + C_2\cos\sqrt{b}\,t + C_3\sin\sqrt{b}\,t.$

We assume a particular solution of the form:

(11) . $X_p(t) = A(t) + B(t)\cos\sqrt{b}\,t + C(t)\sin\sqrt{b}\,t.$

The method of variation of parameters leads to the system:

(12)
$$B'\cos\sqrt{b}\,t + C'\sin\sqrt{b}\,t = \frac{-Q(t)}{\sqrt{b}}$$
$$-B'\sin\sqrt{b}\,t + C'\cos\sqrt{b}\,t = 0$$
$$A' + B'\cos\sqrt{b}\,t + C'\sin\sqrt{b}\,t = 0$$

with solutions given by:

(13)
$$A = \int \frac{Q(t)}{\sqrt{b}}\,dt$$

$$B = -\frac{1}{2\sqrt{b}}\int Q(t)\cos\sqrt{b}\,t\,dt$$

$$C = -\frac{1}{2\sqrt{b}}\int Q(t)\sin\sqrt{b}\,t\,dt$$

In order to evaluate (13), we need to find Q(t). Since we wish the population model to behave in a density –dependent way, we assume that:

(14)
$$x(t) = k\frac{\exp(at)}{1+k\exp(at)}$$

and evaluate:

(15)
$$Q(t) = bc\,x(t) - 2a\left(\frac{dx}{dt}\right)^2 + a\left(1-2x(t)\right)\frac{d^2x}{dt^2}.$$

The derivatives of x(t) are:

(16)
$$\frac{dx}{dt} = \frac{a\,k\exp(at)}{[1+\exp(at)]^2}$$

$$\frac{d^2x}{dt^2} = \frac{a^2K\exp(at)(1-\exp(at))}{[1+\exp(at)]^2}$$

which, when substituted to (15) yield:

(17)
$$Q(t) = \frac{m_1\exp(at)}{[1+\exp(at)]^4} + \frac{m_2\exp(2at)}{[1+\exp(at)]^4} + \frac{m_3\exp(3at)}{[1+\exp(at)]^4} + \frac{m_4\exp(4at)}{[1+\exp(at)]^4}$$

where:

$$m_1 = bck$$

$$m_2 = [3bc - 2a^3K + a^3 - 2k]k$$

$$m_3 = [3bc - a^3]\mathbf{k}$$

$$m_4 = [bc - 2a^3K + a^3]k$$

We can use (17) to compute: A(t) = $\frac{1}{b}\int Q(t)dt$. In particular:

$$\int Q(t)dt = m_1\int \frac{\exp(at)}{[1+\exp(at)]^4}\,dt + m_2\int \frac{\exp(2at)}{[1+\exp(at)]^4}\,dt + m_3\int \frac{\exp(3at)}{[1+\exp(at)]^4}\,dt$$
$$+m_4\int \frac{\exp(4at)}{[1+\exp(at)]^4}\,dt$$

and note that:

$$m_1\int \frac{e^{at}}{1+\exp(at)^4}dt = -\frac{m_1}{3a(1+\exp(at))^3}$$

$$m_2\int \frac{e^{2at}}{[1+\exp(at)]^4}dt = -\frac{m_2}{a[1+\exp(at)]^2}\left[-\frac{1}{2}+\frac{1}{3(1+\exp(at))}\right]$$

$$m_3\int \frac{e^{3at}}{[1+\exp(at)]^4}dt = -\frac{m_3}{a[1+\exp(at)]}\left[-1+\frac{1}{1+\exp(at)}-\frac{1}{3(1+\exp(at))^2}\right]$$

$$m_4\int \frac{e^{4at}}{[1+\exp(at)]^4}dt$$
$$= \frac{m_4}{a}\left[\ln(1+\exp(at))+\frac{3}{1+\exp(at)}-\frac{3}{2(1+\exp(at))^2}+\frac{1}{3(1+\exp(at))^3}\right]$$

The computation for B(t) and C(t) are shown in the Appendix.

SIMPLIFICATION OF THE MODEL

The extremely complicated function obtained in section 2 renders the model unusable for non- mathematicians. For this reason, we further simplify Model (6) yet retain most of its richness. To do so, we re-define Model (6) into:

(18) $\frac{dx}{dt} = a\,x(1-x) + \frac{dz}{dt} - \frac{dy}{dt}$

or which is the same thing as saying that the population of healthy individuals changes through the increment added by those who recovered at time t and decrement by those who get infected at that time.

Moreover:

(19) $\frac{dy}{dt} = ab\,x(1-x)$

$\frac{dz}{dt} = ac\,x(1-x).$

These together imply that:

(20) $\frac{dx}{dt} = a\,x(1-x) + ab\,x(1-x) - ac\,x(1-x)$ *or* $\frac{dx}{dt} = [a(1-b+c)]\,x(1-x).$

Equation (20) has a neat and rather simple solution:

(21) $x(t) = K\frac{\exp[a(1-b+c)t]}{1+K\exp[a(1-b+c)t]}$

The logistic curve tends to its upper limit (i.e. the healthy individuals continues to grow to the maximum carrying capacity) provided a (1 – b + c > 0) or b – c < 1 and decreases if b – c >1.

The quantity of interest is phrased as an index. Let:

(22) $x(t) = K\frac{\exp[a(1-b+c)t]}{1+K\exp[a(1-b+c)t]}$

if $\varphi \geq 1$, then an epidemic outbreak is likely to happen, otherwise, if $\varphi < 1$, then the epidemic is contained. It is interesting to note that the more complicated model (6) actually results to the same conclusion.

We can further explore the dynamics of the corresponding discrete epidemic growth model:

(23) $X_t = a(1-b+c)\,X_{t-1}(1-\ X_{t-1}), \qquad 0 < X_t \leq 1.$

Suppose first that the population progresses unhampered by diseases according to its natural law where $X_t = \alpha X_{t-1}(1-\ X_{t-1})$, $1< \alpha \leq 3$. Assuming regularity, the climax population is predicted to be $X_\infty = \frac{\alpha - 1}{\alpha}$. It is of interest to find out the climax population when infectious diseases are present. In this case:

$$X_\infty = \frac{a-ab+ac-1}{a-ab+ac.} \qquad as\ t \to \infty.$$

Extinction occurs if the numerator is zero or equivalently if :

(25) $\frac{c-b}{a} = \frac{1-a}{a^2}$ or $\frac{recovery\ rate - infection\ rate}{population\ growth\ rate} = \frac{1-population\ growth\ rate}{(population\ growth\ rate)^2}$

Just what effect have infection and recovery on the population dynamics of healthy individuals? If the natural growth rate is to produce a stable long run population, then $1 < a < 3.57$. With infection and recovery factored in, we need:

(24) $1 < a\,(1 + c - b) < 3.57$

or

(25) $\frac{1-a}{a} < c - b < \frac{3.57-a}{a},$

That is, the survival rate (c - b), which is the difference between the recovery rate and infection rate, must be between the prescribed

limits. For instance, if a = 2, then the survival rate must be such that $-0.5 < c - b < 0.785$ for a population that doubles once every generation. If c – b falls below -50% (i.e. more infection than recovery), then the population is in danger of extinction.

Can these natural processes of infection and recovery impact on a population experiencing periods of bust and booms (chaotic)? Chaos occurs when the population growth rate exceeds or is equal to 3.57. This means that if we wish to bring back the population to a stable population, we require that $a_0 > 3.57$ and $(1 + c - b) < 1$, or $c - b < 0$ (that is, the infection rate must exceed the recovery rate). *Nature has a way of dealing with run – away population growths*. There is a way to test this hypothesis by correlating the percentage of cases noted for the A(H1N1) virus to the population growth rates of various countries since 2005. A high positive correlation is an evidence for the hypothesis while a low non – significant correlation is an evidence against it.

There is yet another important insight that can be gained from this analysis. Since nature favors balance and stability, it follows that the spread of infection will be faster in areas of high population densities (and growth) such as the highly urbanized cities than in rural areas. Mathematically, this means that if $a_0 > 3.57$ then, to achieve balance, nature provides for $c - b < 0$. Thus, *one important disease control measure that can be implemented is to decongest urban centers by creating jobs in the rural areas so people will move out of the cities*.

NUMERICAL RESULTS

We provide several numerical simulations and one (1) real-life data to test the hypothesis that "nature has a way of dealing with run-away population growth rates".For the numerical simulations, we generated data starting from an initial population of $x_0 = 0.10$ (or 10% of the maximum carrying capacity) with various combinations of (a,b,c) = (population growth rate, infection rate, recovery rate). The combinations are chosen to provide the following scenario:

Run-Away Growth Rate with Nature Balance: (4, .75, .25)
Stable Growth Rate with Nature Balance: (3, .75, .70) Stable Growth Rate with Medical Intervention: (3, .50, .75)

The "run-away growth rate with medical intervention" scenario is omitted because that will certainly lead to a chaotic process. We generated 30 generations (roughly 30 lifetimes) of each to determine the long-run behavior of the population.

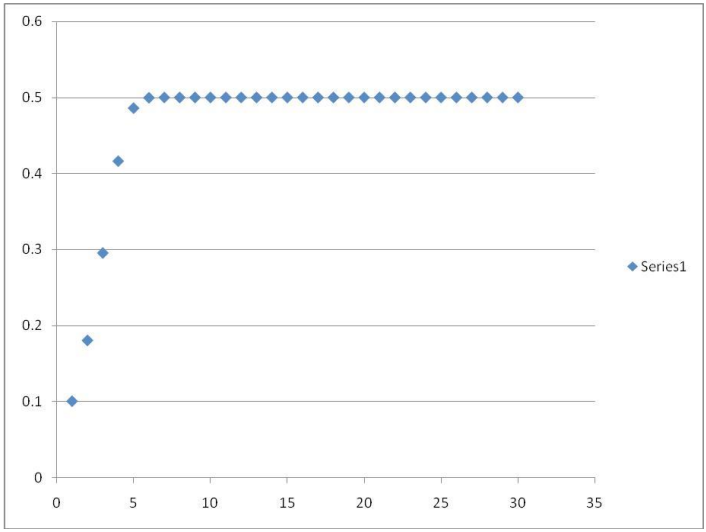

Figure 1: High Growth Rate with Natural Counterbalance by Nature through Disease Spread

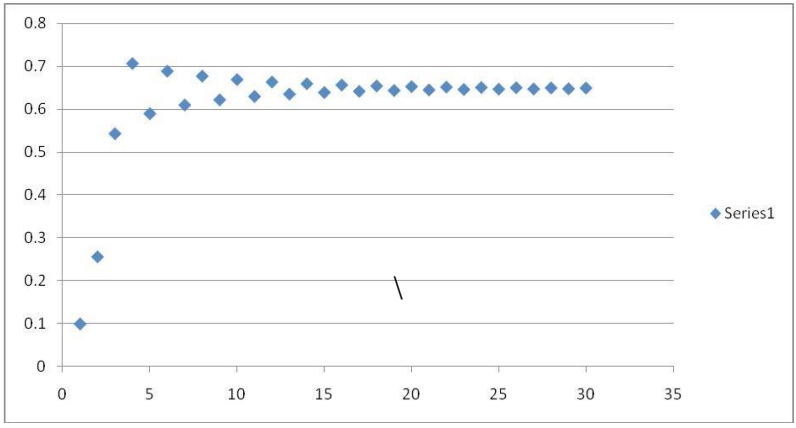

Figure 2: Stable Population Growth Rate with Natural Counterbalance by Nature

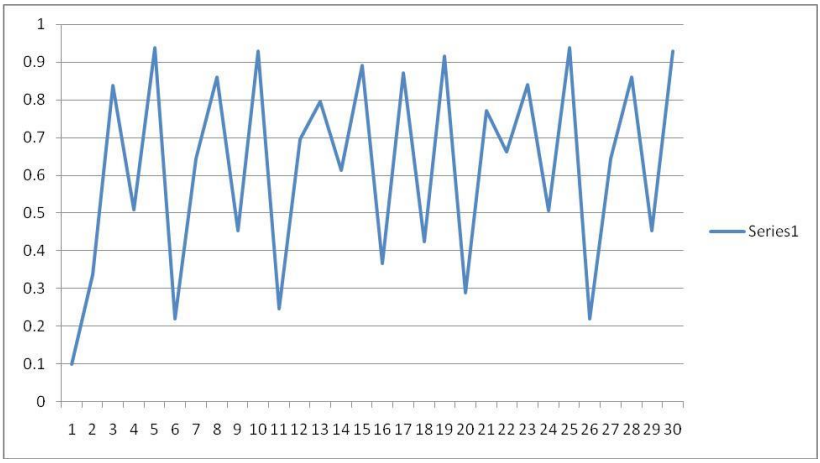

Figure 3: Stable Population Growth Rate with High Medical Intervention for Infection

Notice how the first two graphs tend to stability in the long run with the last graph (using high medical intervention on a stable population) resulting to a chaotic population dynamics instead. Table 1 shows the simulated values for the graphs above.

Table 1: Simulated population values for different scenarios

Paramet	**N**	**Param.**	**N**		**Param.**	**N**
a= 4	0.1	a= 3	0.1		a= 3	0.1
b= .75	0.18	b= .75	0.2565		b = .50	0.3375
c = .25	0.2952	c = .70	0.543517		c = .75	0.838477
High Growth Rate with Nature Balance	0.416114	Stable Growth Rate with Nature Balance	0.707103	Stable Growth Rate with High Medical Intervention		0.507876
	0.485926		0.590259			0.937267
	0.499604		0.689282			0.22049

0.5	0.610391	0.644527
0.5	0.677769	0.859169
0.5	0.622435	0.45374
0.5	0.669778	0.929475
0.5	0.63035	0.245817
0.5	0.664075	0.695216
0.5	0.635776	0.79459
0.5	0.65996	0.612063
0.5	0.639577	0.890407
0.5	0.656977	0.365933
0.5	0.642271	0.870098
0.5	0.654813	0.423854
0.5	0.644194	0.915757
0.5	0.653243	0.289299
0.5	0.645572	0.771019
0.5	0.652105	0.662058
0.5	0.646562	0.839015
0.5	0.65128	0.506508
0.5	0.647276	0.937341
0.5	0.650683	0.220248
0.5	0.64779	0.64402
0.5	0.650251	0.859719
0.5	0.64816	0.45226
0.5	0.649938	0.928953

DISCUSSIONS

The numerical simulations illustrate the effects of the spread of infectious diseases on the population dynamics with and without medical intervention. In particular:

1. We note that stable populations (those with low to medium population growth rates) will stay stable even without medical intervention. This may be attributed to the fact that for such populations, the maximum carrying capacity is well above the current levels of the population. This ,of course , means that contact and infection rates are minimized, effectively reducing the basic reproduction number to less than 1 indicating that the infectious disease will die out in the long run.

2. Unstable populations (those with high population growth rates) will tend to be stabilized through the spread of infectious diseases and low recovery rates.For these population types, the approach to the maximum carrying capacity of the environment is very fast. Once the population size reaches a high enough percentage of the maximum carrying capacity, contacts and infection rates become very high, effectively increasing the basic reproduction number of the infectious disease to a quantity greater than 1. Competition for a limited resource will become very intense with a few getting a good share while the majority barely surviving; high population density will cause congestion a situation ripe for infectious disease transmission. The situation will persist until the population size again falls well below the maximum carrying capacity. The cycle is repeated because of the high population growth rates and so, one observes a period of
"busts" and "booms" in the population of healthy individuals. The way to avoid this situation is really to establish a rational population growth rate policy that is consistent with available national resources.

3. On the other hand, with progress in medical science enabling scientists to arrest the spread of virulent diseases, even stable populations can go into a chaotic regime because of resource limitations (density-dependence). Consider a stable population with growth rate

a < 3.57. Because of the high recovery rates from infectious diseases (due to medical science), recovered individuals rejoin the population adding to the new births in the system. This situation effectively pushes the population growth rate past its stable threshold of 3.57, thereby, inducing a state of chaotic regime in the population of healthy individuals. This situation, therefore, cycles back to the situation in item 2 above.

Global Analysis of the Spread of HIV and the Relationship of Infection Rates of HIV and Population Densities

Using the model developed, we analyzed the spread of the Human Immune Deficiency Virus (HIV) across different countries in various continents of the world. Data were obtained from the *2010 CIA (International Atlas)* as published in the NET and supported by *UNESCO* for the following information: national population growth rates, estimated prevalence/infection rate of HIV as reported, the population densities of the countries, and the socio-economic development index based on UN published ranking of countries (2010). The objectives of the analysis are: (a.) to determine the population trajectories of the different countries given their population growth rates and HIV infection rates in the long run, and (b.) to determine the relationship of socio-economic development and infection rate to ascertain the impact of public health care on the spread of infectious diseases.

It is noted that HIV, unlike other infectious diseases , has no cure as of this writing. Thus, once infected there is no hope for recovery from the disease. It follows that for this type of infectious disease the parameter c = 0 and the population trajectory is determined solely by the growth rate ,a, and the infection rate, b in the population. Note likewise that the model is a **global model** and does not take into account the specific mode of transmission of HIV. The latter case is handled by more detailed models as in Hyman (1997).

Effect of Socio-Economic Development and other Factors on Infection Rates for HIV

We first examine the effect of socio-economic development (as a surrogate measure to public health care) and infection rate across 100 countries of diverse population densities. Table 2 shows the results of the analysis:

Table 2: Summary of the regression analysis with infection rate as dependent variable and SES as independent variable

The regression equation is
Infection rate% = 8.31 - 3.01 DEV.

Predictor	Coef	SE Coef	T	P
Constant	8.3106	0.9917	8.38	0.000
DEV.	-3.0063	0.4868	-6.18	0.000

S = 3.934 R-Sq = 28.0% R-Sq(adj) = 27.3%

The results indicate a very significant association between infection rate and the level of socio-economic development of a nation ($p < .01$). The higher the level of development, the smaller is the infection rate ($r = -0.529$ or $r2 \times 100\% = 28.0\%$). A graphical inspection of the scatterplot of the level of socio-economic development and infection rate suggests a reciprocal transformation. Table 3 shows the analysis using the reciprocal of the level of SES as the independent variable:

Table 3: Regression analysis using a reciprocal transformation for the independent variable: Level of SES

The regression equation is
Infection rate% = - 3.33 + 9.19 1/dev

Predictor	Coef	SE Coef	T	P
Constant	-3.3328	0.9371	-3.56	0.001
1/dev	9.193	1.309	7.02	0.000

S = 3.782 R-Sq = 33.5% R-Sq(adj) = 32.8%

A more detailed analysis suggested by the reciprocal transformation revealed that infection rate varies inversely as the level of development ($p<.01$) and that 33.5% of the variations in the infection rates of the countries can be explained by the inverse of the socio-economic development of these countries ($r = 0.578$).

DISCUSSIONS

1. A global analysis of the relationship between infection rate (as dependent variable) and the level of a country's socio-economic development revealed that a country's level of development significantly correlates with the spread of HIV. Public health care systems in more developed nations are superior to the corresponding public health care systems in less developed nations which explains why the HIV spread is easily arrested in more developed countries.

2. However, the level of a country's socio-economic development explains only a little over a third of the variances observed for the HIV infection rates in 100 countries. This means that other explanatory factors could account for the high infection rates even in some more developed nations such as their lifestyles, beliefs, traditions, and societal permissiveness given the nature of the infectious disease being examined.

3. Interestingly, the relationship between population growth rate and level of development was found to be highly significant ($r = -0.6403$, $t = -8.25$) so that roughly 41% of the countries' population growth rates are accounted for by their levels of development. More developed nations tended to have lower population growth rates as opposed to the less developed nations which registered high to very high population growth rates. That is, developed nations have population policies that tended to encourage lower population growth rates than the less developed nations (which do the opposite).

4. Finally, the relationship between population density and infection rate can be inferred indirectly. The relationship between population density and SES is roughly r = 0.1703 while the relationship between SES and infection rate is given by r = 0.5781. By the multiplication rule, the indirect effect of population density on the rate of infection or prevalence of HIV is about r = 0.0984. The low correlation between infection rate and population density in this particular case is an indication that the maximum carrying capacities(in terms of resources, space etc.) of the countries are still well above their current levels.

Population Trajectories of Selected Countries Based on Growth Rates and Infection Rates

We recomputed the new population growth rates based on the current growth rates of the country and the infection rates for HIV. The results are displayed in Table 4 for selected countries (the complete listing is placed in the appendix):

Table 4: Predicted long run populations of selected countries under an HIV regime

Stable Limit	growthrate	infection rate	new growth rate	Predicted Stable Limit
1. Brazil	1.2	0.7	1.1916	16%
2. Madagascar	3	1.7	2.949	66%
3. Uganda	2.69	4.1	2.57971	61%
4. Philippines	1.96	1.5	1.9306	48%
5. Cambodia	1.77	2.6	1.72398	42%
6. Brunei	1.76	0.2	1.75648	43%

Extinction				
1. Bahamas	0.93	3	0.9021	0%
2. Maldives	-0.17	0.1	-0.16983	0%
3. Tunisia	0.98	0.04	0.97961	0%
4. Norway	0.34	0.1	0.33966	0%
5. Singapore	1	0.2	0.998	0%
6. Canada	0.82	0.3	0.8175	0%
7. United States	0.98	0.6	0.97412	0%
Unstable Limits				
1. U. Arab Emirates	3.69	0.18	3.68336	Cycles
2. Niger	3.68	1.2	3.63584	Cycles

Discussions:

1. Results show that the Philippines would have a stable limit at 48% of its carrying capacity given its current HIV infection rate. Countries in the same stable limit category include Brazil, Madagascar, Cambodia and Brunei. Uganda with the highest infection rate in this category of countries is saved from oblivion because of its high population growth rate.

2. Developed countries, in general, with stringent population policies leading to very low growth rates and positive infection rates are in danger of extinction. These include: the United States of America, Singapore, Canada and others.

3. Only two countries: the United Arab Emirates and Niger will undergo cycles in their population sizes and will not achieve a stable long run population despite the HIV infection spreading in the population at the given rates.

CONCLUSION

The spread of infectious diseases in a given population is inextricably linked to the issue of population growth within a limited-resource environment. Nature has a way of maintaining the critical balance between population growth and resource availability by way of such infectious diseases. Even with advancements in medical science, the only rational way to deal with the spread of infectious diseases is to implement a population growth policy that is consistent with a limited-resource scenario since, as the current model predicts, a chaotic regime will be inevitable in the long run.

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