

Modeling the probability distribution model for controlling the prevalence of disease outbreak

Akintunde Oyetunde A

¹ Department of Mathematics, Faculty of Science, Federal University Oye-Ekiti, Ekiti State, Nigeria

Abstract

This research work investigates the development and benefits of model for disease transmission through a population. Demonstration on how to characterize a disease as it is spread in terms of the portions of the population that it affects, as well as how to evaluate and explore preventative and controlled measures to limit the disease's effects are well examined. For the purposes of this work, exploration into the sensitivity of the framework that involves using a probabilistic model checker has to changes in the underlying disease transmission model was shown. The use of publicity campaigns to expose high proportions of large populations to messages through routine uses of existing media, such as television, radio, and newspapers was considered and found to be effective in controlling the transmission of the prevalence of disease among human population.

Keywords: disease transmission, control, prevalence, probability, human population

1. Introduction

The 20th century saw a marked decline in infectious disease deaths and an impressive eradication of some infectious diseases; however the current population is still faced with outbreaks of new diseases and the resurgence of some previous declining diseases. Disease control in the 20th century resulted from improved sanitation and hygiene, the discovery of antibiotics and the introduction of worldwide childhood vaccination programs. Science and technology played major roles in these improvements. In the 21st century, scientists, researchers, public health officials and governments continue efforts to control infectious diseases such as HIV, West Nile virus, Ebola virus, Lassa fever, various strains of influenza, severe respiratory syndrome (SARS) and encephalopathy.

The mathematical techniques used to understand, forecast, and control the spread of infectious diseases like influenza are diverse and growing rapidly. Some techniques have been newly developed, whereas others build upon existing methods from diverse fields including dynamical systems, stochastic processes, statistical physics, graph theory, statistical theories, operations research, and high-performance computing. The use of mathematical theory enables clarity in understanding and communicating the generalized processes of explaining and/or predicting the observed phenomenon of a complex real-world event or structure, and in doing so glean the fundamental processes (the essential features) that underpin the system. Different types of mathematical models as discussed by Sharmistha Mishra *et al* are used to capture the essential behavioral and demographic characteristics of a population and the biological features (natural history) of the infection among which are:

1. Transmission dynamic model
2. Static model
3. Compartmental SIR model
4. Deterministic model
5. Stochastic model
6. Individual-based (agent-based, micro-simulation) model
7. Network model, and many others.

2. Compartmental Models of Epidemiology

The first differential equation models of infectious disease dynamics go back as far as 1766, to the work of Daniel Bernoulli, which has been recently republished (Bernoulli and Blower). Modern differential equation models of epidemics were introduced by Kermack and McKendrick and later expanded by Anderson and May.

One basic but well-characterized model of the spread of disease through a population is known as the Susceptible, Infectious and Recovered Model, commonly referred to as the SIR model. The SIR model for any specific disease assumes that there are three distinct divisions of a given population comprising of N people, specifically at some time instance t , they are:

- $S(t)$ - The number of people who are susceptible (have not yet been infected) to the disease under study at time instance t .
- $I(t)$ - The number of people who are actively infected and can spread the disease under study to members of the susceptible population at time instance t .
- $R(t)$ - The number of people who were infected but have now recovered from the disease under study by time instance t .

Because these three sub-populations form a partition of the overall population, we have that:

$$\forall t, S + I + R = N \quad (1)$$

This model assumes a unidirectional progression of the population, namely each member of the populations follows the flow diagram:

$$\text{Susceptible} \rightarrow \text{Infectious} \rightarrow \text{Recovered}$$

The SIR model is thus more well-suited for all diseases to which members of the population cannot be re-infected (perhaps due to such factors as an inherent resistance built up during a recovery period). It is easy to extend or adjust the

model to allow for alternative disease patterns. Such models of this sort are termed *compartmental models*, due to their subdividing of the population into characteristic groups. Some other common compartmental models are described in the Table below.

Table 1: Common compartmental models of disease spread in a population.

Model	Flow Diagram
SIR	<i>Susceptible</i> → <i>Infectious</i> → <i>Recovered</i>
SEIR	<i>Susceptible</i> → <i>Exposed</i> → <i>Infectious</i> → <i>Recovered</i>
SIS	<i>Susceptible</i> ↔ <i>Recovered</i>

The *Exposed (E)* population models a latency in becoming infected and becoming infectious.

In addition to the modeling of the population partitions, compartmental models also include rules about how members of the population transition from state to state. A common approach is to take the time component *t* to be a continuous variable, and assume that each edge of the flow diagram in the table above has a transition rate associated with it. Such rates are represented as ordinary differentiable equations (ODEs), which allow for compact transition rules. Thus,

$$\frac{ds}{dt} = \beta_1 - \mu_1 S - (\alpha_1 I + \alpha_2 R)S + (\gamma + (1 - \theta)\beta_1) \quad (2)$$

$$\frac{\delta I}{\delta t} = (\alpha_1 I + \alpha_2 R)S - (\mu_1 + \delta_1 + \delta)I + \theta\beta_1 I \quad (3)$$

$$\frac{\delta E}{\delta t} = \delta S - kE \quad (4)$$

$$\frac{\delta R}{\delta t} = (\beta_2 - \mu_2 - \delta_2)R \quad (5)$$

The above models are solved to obtain equilibrium stable by setting them all equal to zero, thus:

$$\frac{ds}{dt} = \frac{dI}{dt} = \frac{dE}{dt} = \frac{dR}{dt} = 0 \quad (6)$$

Where, *S* is the Susceptible class of human population, *I* is the Infected class of human population, *E* is the Exposed class of human population, and *R* is the Recovered class of human population. Also, β_1 is the Natural Birth rate for the human population, β_2 is the Natural Birth rate for the vector population, δ_1 is the Death rate for the human population due to infection, and δ_2 is the Death rate of the vector population due other factors like harvesting, fire etc contracting rate for the susceptible human population. In addition, α_1 is the Contracting rate for the susceptible human population as a result of interaction with infected human population, α_2 is the Contracting rate for the susceptible human population as a result of interaction with faeces and urine of infected /reservoir population, γ is the rate at which the susceptible human population join the exposed human population, and *k* is the rate at which the exposed human population moves to the infected human population.

However, the SIR Model is used in epidemiology to compute the amount of Susceptible, Infected, and Recovered people in a population. This model is an appropriate one to use as stated by Hackborn Bill (2008) [13] under the following assumptions:

1. The population is fixed.
2. The only way a person can leave the susceptible group is to become infected. Also, the only way a person can leave

the infected group is to recover from the disease. Once a person has recovered, the person received immunity.

3. Age, sex, social status, and race do not affect the probability of being infected.
4. There is no inherited immunity.
5. The member of the population mix homogeneously (have the same interactions with one another to the same degree).

Since age, sex, social status and race do not affect the probability of an individual being infected by a disease, that do not indicate that there are not other factors that do affect the probability of an individual being infected by a disease.

3. Probabilistic Model for Control

Once a particular chosen model for disease spread over a population, be it as simple as an SIR model, or utilizing a more complex approach such as a contact network, has been done, a natural question to ask is “What sorts of characteristics can it model?”

Perhaps more to the point, one will like the chosen model to be able to answer questions that are relevant to the inspection and understanding of disease spread. In particular, one might be interested in questions related to prevention and/or control of the disease. Such questions may be:

1. “At any time instance *t*, what fraction of the total population is infected?”
2. “Allowing the disease to run its full course, what fraction of the total population will become infected?”
3. “Will person *p* (or group of people *g*) become infected within *t* time units of the onset of the disease?”

If the second question above is considered, it might seem strange at first, because in both the common SIR model and contact networks, it is quite possible that the entire population will become infected (and then recover) exactly once. However, this brings the issue of disease prevention. However, the introduction of such things as vaccinations and public enlightenment/education to the model would result in individuals being initialized to be in the “recovered” state. Of course, some models (including formulations of contact networks) are probabilistic and will not always give the same answer for deterministic questions of this sort. Then one can relax the queries somewhat by rephrasing them with a probabilistic flavor:

1. “At any time instance *t*, what is the likelihood that the fraction of the total population is infected is at least *c*?”
2. “Allowing the disease to run its full course, what is the likelihood that the fraction of the total population becomes infected is at least *c*?”
3. “What is the likelihood that person *p* (or group of people *g*) becomes infected within *t* time units of the onset of the disease?”

The realm of model checking allows one to pose questions of the sort above, both probabilistic and non-probabilistic.

3.1 Introduction of New Probability Distribution for Disease Control

Like existing probability distributions are developed among which are Bernoulli probability distribution, Poisson probability distribution, Gamma probability distribution, Weibull probability distribution, Nakagami probability

distribution, Gamma probability distribution, Beta probability distribution and many others, this research paper presents a new probability distribution function which is just being formulated.

Definition: A random variable x is having a probability density function with two parameters (α and β) given as:

$$f(x/\alpha, \beta) = \frac{1}{2^\alpha \pi^\beta} (x - \beta)^{\alpha-1} e^{-(x-\beta)^\alpha} \begin{matrix} 0 \leq x \leq \infty \\ 0 \leq \alpha \leq \infty \\ 0 \leq \beta \leq \infty \\ x > \beta \end{matrix} \quad (7)$$

And cumulative distribution function given as:

$$Pr(X = x/\alpha, \beta) = e^{-(x-\beta)^\alpha} \quad (8)$$

It should be noted that α is denoted as shape parameter and β is denoted as location parameter.

3.2 Real Life Uses of π

π is introduced into the probability distribution function because π is one of the most extremely useful and fundamental quantities we know of. It is defined as the ratio of area of a circle to its diameter. The applications of π in real life include several areas like geometry, science, trigonometry, nature, and engineering, among others. In nature, for instance, π can measure things like ocean waves, light waves, river bends, radioactive particles distribution, probabilities like the distributions of pennies, the gird of nails and mountains by using a series of circles, among others.

Some examples of daily uses of π include, but not limited to:

1. Electrical engineers use π to solve problems for electrical applications.
2. Statisticians use π to track population dynamics.
3. Medicine benefits from π when studying the structure of the eye and other circular parts of human body.
4. Biochemists use π when trying to understand the structure and function of DNA.
5. Physicists use π when looking into the behavior of fluid ripples and use it in their calculations like in quantum Physics.
6. Clock designers use π when designing pendulums for clocks.
7. Aircraft designers use π to calculate areas of the skin of the aircraft.
8. Signal processing and spectrum analysis (finding out what frequencies are in the wave you are using) use π since fundamental period of a sine wave is 2π .
9. Navigation experts make use of π when locating global positioning (GP).
10. π is used in most calculations for building and construction works.
11. π is used in communication and music theory.
12. π is used in medical procedures, air travels and space flight.

3.3 Estimation of the K-Th Raw Moment

By the method of raw moments, the k-th raw moment of the probability density function given in equation (7) above is

obtained as:

$$E(x^k) = \int_{-\infty}^{\infty} x^k f(x) dx \quad k = 1, 2, 3, \dots \quad (9)$$

$$= \int_0^{\infty} x^k \frac{1}{2^\alpha \pi^\beta} (x - \beta)^{\alpha-1} e^{-(x-\beta)^\alpha} dx$$

$$= \frac{1}{2^\alpha \pi^\beta} \int_0^{\infty} x^k (x - \beta)^{\alpha-1} e^{-(x-\beta)^\alpha} dx$$

$$\text{If } t = (x - \beta)^{\alpha-1} \Rightarrow t^{1/\alpha} = x - \beta \Rightarrow x = t^{1/\alpha} + \beta \text{ and } dx = \frac{1}{\alpha} t^{-(1-1/\alpha)} dt$$

Thus,

$$E(x^k) = \frac{1}{2^\alpha \pi^\beta} \int_0^{\infty} (t^{1/\alpha} + \beta)^k t^{(1-1/\alpha)} e^{-t} \frac{1}{\alpha} t^{-(1-1/\alpha)} dt$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \int_0^{\infty} (t^{1/\alpha} + \beta)^k e^{-t} dt \quad (10)$$

When $k=1$ in equation (10),

$$E(x) = \frac{1}{\alpha 2^\alpha \pi^\beta} \int_0^{\infty} (t^{1/\alpha} + \beta)^1 e^{-t} dt$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \left\{ \int_0^{\infty} t^{1/\alpha} e^{-t} dt + \int_0^{\infty} \beta e^{-t} dt \right\}$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{1}{\alpha}\right) + \beta \right]$$

$$= \frac{\beta + \Gamma\left(1 + \frac{1}{\alpha}\right)}{\alpha 2^\alpha \pi^\beta} \quad (11)$$

This is the **mean** of the distribution.

When $k=2$ in equation (10),

$$E(x^2) = \frac{1}{\alpha 2^\alpha \pi^\beta} \int_0^{\infty} (t^{1/\alpha} + \beta)^2 e^{-t} dt$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\int_0^{\infty} t^{2/\alpha} e^{-t} dt + 2\beta \int_0^{\infty} t^{1/\alpha} e^{-t} dt + \beta^2 \int_0^{\infty} e^{-t} dt \right]$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{2}{\alpha}\right) + 2\beta \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^2 \right] \quad (12)$$

When $k=3$ in equation (10),

$$E(x^3) = \frac{1}{\alpha 2^\alpha \pi^\beta} \int_0^{\infty} (t^{1/\alpha} + \beta)^3 e^{-t} dt$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\int_0^{\infty} t^{3/\alpha} e^{-t} dt + 3\beta \int_0^{\infty} t^{2/\alpha} e^{-t} dt + 3\beta^2 \int_0^{\infty} t^{1/\alpha} e^{-t} dt + \beta^3 \int_0^{\infty} e^{-t} dt \right]$$

$$= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{3}{\alpha}\right) + 3\beta \Gamma\left(1 + \frac{2}{\alpha}\right) + 3\beta^2 \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^3 \right] \quad (13)$$

When k=4 in equation (10),

$$\begin{aligned} E(x^4) &= \frac{1}{\alpha 2^\alpha \pi^\beta} \int_0^\infty (t^{1/\alpha} + \beta)^4 e^{-t} dt \\ &= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\int_0^\infty t^{4/\alpha} e^{-t} dt + 4\beta \int_0^\infty t^{3/\alpha} e^{-t} dt \right. \\ &\quad \left. + 6\beta^2 \int_0^\infty t^{2/\alpha} e^{-t} dt + 4\beta^3 \int_0^\infty t^{1/\alpha} e^{-t} dt \right. \\ &\quad \left. + \beta^4 \int_0^\infty e^{-t} dt \right] \\ &= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{4}{\alpha}\right) + 4\beta \Gamma\left(1 + \frac{3}{\alpha}\right) + 6\beta^2 \Gamma\left(1 + \frac{2}{\alpha}\right) + 4\beta^3 \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^4 \right] \quad (14) \end{aligned}$$

And so on for other values of k.

3.4.1 Estimation of-The Variance of The Probability Distribution

$$\begin{aligned} Var(x) &= E(x^2) - [E(x)]^2 \\ &= \frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{2}{\alpha}\right) + 2\beta \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^2 \right] \\ &\quad - \left[\frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{1}{\alpha}\right) + \beta \right] \right]^2 \\ &= \frac{\alpha 2^\alpha \pi^\beta \Gamma\left(1 + \frac{2}{\alpha}\right) - \left(\Gamma\left(1 + \frac{1}{\alpha}\right) + \beta\right)^2 (\alpha 2^\alpha \pi^\beta - 1)}{\alpha^2 2^{2\alpha} \pi^{2\beta}} \quad (15) \end{aligned}$$

3.4.2 Estimation of the Skewness of the Probability Distribution

$$\begin{aligned} skewness &= \frac{E(x^3)}{[\sqrt{E(x^2)}]^3} \\ &= \frac{\frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{3}{\alpha}\right) + 3\beta \Gamma\left(1 + \frac{2}{\alpha}\right) + 3\beta^2 \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^3 \right]}{\left[\frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{1}{\alpha}\right) + \beta \right] \right]^{3/2}} \\ &= \alpha^{1/2} 2^{\alpha/2} \pi^{\beta/2} \frac{\left[\Gamma\left(1 + \frac{3}{\alpha}\right) + 3\beta \Gamma\left(1 + \frac{2}{\alpha}\right) + 3\beta^2 \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^3 \right]}{\left[\Gamma\left(1 + \frac{1}{\alpha}\right) + \beta \right]^{3/2}} \quad (16) \end{aligned}$$

3.4.3. Estimation Of-The Kurtosis Of The Probability Distribution

$$\begin{aligned} kurtosis &= \frac{E(x^4)}{[E(x^2)]^2} \\ &= \frac{\frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{4}{\alpha}\right) + 4\beta \Gamma\left(1 + \frac{3}{\alpha}\right) + 6\beta^2 \Gamma\left(1 + \frac{2}{\alpha}\right) + 4\beta^3 \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^4 \right]}{\left[\frac{1}{\alpha 2^\alpha \pi^\beta} \left[\Gamma\left(1 + \frac{2}{\alpha}\right) + 2\beta \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^2 \right] \right]^2} \end{aligned}$$

$$= \alpha 2^\alpha \pi^\beta \frac{\left[\Gamma\left(1 + \frac{4}{\alpha}\right) + 4\beta \Gamma\left(1 + \frac{3}{\alpha}\right) + 6\beta^2 \Gamma\left(1 + \frac{2}{\alpha}\right) + 4\beta^3 \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^4 \right]}{\left[\Gamma\left(1 + \frac{2}{\alpha}\right) + 2\beta \Gamma\left(1 + \frac{1}{\alpha}\right) + \beta^2 \right]^2} \quad (17)$$

3.4.4 Graphical Representation of the Probability Distribution Function

The graph below shows the curve of the probability distribution function (pdf) in equation (7) of the two parameters (α and β) at several values of the two parameters (α and β) as shown below:

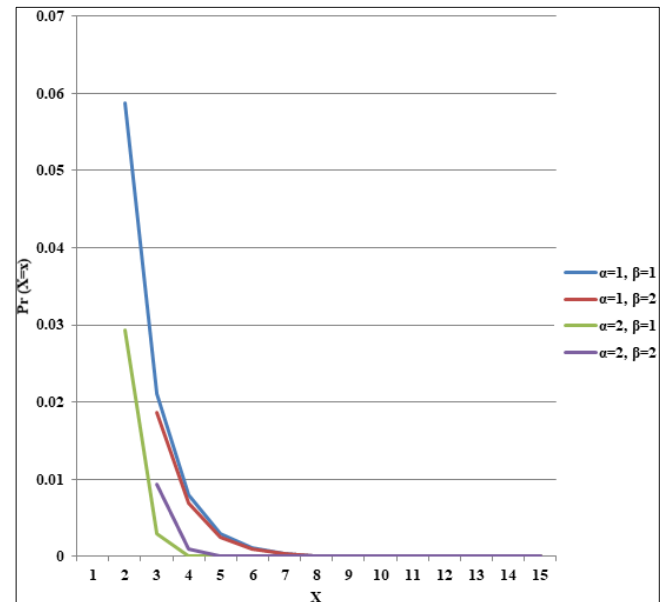


Fig 1: Graphical representation of the probability density function curve under different values of (α and β)

4. Application of the Probability Distribution to Disease Control

If the two parameters in the probability distribution are explained and termed in the terms of sociological factors for the controlling of disease among human population, like making α to be number of occurrence or outbreak of any disease in any geographical location and β to be the number or rate of awareness or publicity or enlightenment about the disease in the geographical environment. One will observe that the possibility of having the disease will drastically reduce among the human population, the values of the two parameters (α and β) are increased.

Most diseases are preventable to a greater or lesser degree. In the case of diseases resulting from environmental exposures, prevention is a matter of eliminating or sharply reducing the factors responsible for in the environment. However, the infectious diseases may be prevented in one of the two general ways: by preventing contact and transmission of infection between the susceptible host and the source of infection, and by rendering the host unsusceptible, either by selective breeding or by induction of an effective artificial immunity.

As emphasized by Centre for Disease Control (CDC) and World Health Organization (WHO), over the past few decades, raising awareness through media campaigns have been used in an attempt to affect various health behaviours in human populations. Such campaigns have most notably been aimed at tobacco use and illicit-drug prevention, but have also addressed alcohol and illicit drug use, cancer screening and prevention, sex-related behaviours, child survival, and

many other health-related issues. Typical campaigns have placed messages in media that reach large audiences, most frequently via television or radio, but also outdoor media, such as billboards and posters, and print media, such as magazines and newspapers. Exposure to such messages is generally passive, resulting from an incidental effect of routine use of media. Some campaigns incorporate new technologies like the internet, mobile phones and personal digital assistants. (Kotler P. and Lee N. R., 2008) ^[1]

Media campaigns can be of short duration or may extend over long periods. They may stand alone or be linked to other organized programme components, such as clinical or institutional outreach and easy access to newly available or existing products or services, or may complement policy changes. Multiple methods of dissemination might be used like peer-to-peer campaign, campaigns at religious centres, market places, recreation centres, and many others. The great promise of these campaigns, awareness and enlightenment lies in their abilities to disseminate well defined behaviour ally focused messages to large audiences repeatedly, over time, in an incidental manner, and at a low cost per head.

5. Conclusion

The presented probability model to be known as *Tunde-Akintunde Probability Distribution function* can be used to estimate the size/location and speed of spread of any disease, irrespective of the nature of the infectious agent or the size and spatial distribution of the affected population. The probability model will not only help in the development of policy options for containment but can also assist in the development of guidelines for effective population treatment programs. The newly developed probability distribution is hereby presented for further used and analysis.

6. References

1. Kotler P, Lee NR. Social Marketing: influencing behaviors for good. 3. Thousand Oaks, CA: Sage, 2008.
2. Hornik R, Yanovitzky I. Using theory to design evaluations of communication campaigns: the case of the National Youth Anti-Drug Media Campaign. Commun Theory. 2003; 13:204-24.
3. Fishbein M, Azjen I. Predicting and changing behaviour: the reasoned action approach. New York: Psychology Press, 2010.
4. Bernoulli D, Blower S. An attempt at a new analysis of the mortality caused by smallpox and of the advantages of inoculation to prevent it. Reviews in Medical Virology. 2004; 14:275-288.
5. Anderson RM, May RM. Population biology of infectious diseases: Part I. Nature, 1979; 280:361-367.
6. Anderson RM, May RM. Population biology of infectious diseases: Part II. Nature. 1979; 280:455-461.
7. Anderson R, May R. Infectious Diseases of Humans: Dynamics and Control. Oxford: University of Oxford Press, 1991.
8. Anderson RM, Donnelly CA, Ferguson NM, Woolhouse MEJ, Watt C, *et al.* Transmission dynamics and epidemiology of BSE in British cattle. Nature. 1996; 382:779-788.
9. Ferguson NM, F de Wolf, Ghani AC, Fraser CC, Donnelly *et al.* Antigen driven CD4+ T-cell and HIV-1 dynamics: Residual viral replication under HAART. Proceedings of the National Academy of Sciences. 1999; 21-96(26):15167-15172.
10. Ho DAU, Neumann AS, Perelson W, Chen JM, Leonard, Markowitz M. Rapid turnover of plasma virions and CD4 lymphocytes in HIV-1 infection. Nature. 1995; 373:123-126.
11. Nissinen AM, Leinonen P, Huovinen E, Herva ML, Katila S, Kontiainen O, *et al.* Antimicrobial resistance of *Streptococcus pneumoniae* in Finland, 1987-1990. Clinical Infectious Diseases. 1995; 20(5):1275-1280.
12. Schwartländer B, Garnett G, Walker N, Anderson R. AIDS in the new millennium. Science. 2000; 289:64-67.
13. Hackborn, Bill. Susceptible, Infected, Recovered: the SIR model of an Epidemic. University of Alberta: Augustana. Fall, 2008. <<http://www.augustana.ab.ca/~hackw/mat332/exhibit/sir.ppt>>.
14. Keeling Matt. The mathematics of diseases. Plus Magaazine: Living Mathematics. Mar. 2001. Fall, 2008. <<http://plus.maths.org.uk/issue14/features/diseases/index.html>>
15. Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society A. 1927; 115:700-721.
16. Sharmistha Mishra, David N Fisman, Marie Claude Boily. The ABC of terms used in mathematical models of infectious diseases. Journal Epidemiol Community Health. 2011; 65:87e94. doi:10.1136/jech.2009.097113
17. Dodge KA. Framing public policy and prevention of chronic violence in American youths. Am Psychol. 2008; 63:573-90.
18. Swaim RC, Kelly K. Efficacy of a randomized trial of a community and school-based anti-violence media intervention among small-town middle school youth. Prev Sci. 2008; 9:202-14.
19. Mikton C, Butchart A. Child maltreatment prevention: a systematic review of reviews. Bull World Health Organ. 2009; 87:353-61.