



ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 5.2
IJAR 2016; 2(10): 383-386
www.allresearchjournal.com
Received: 25-08-2016
Accepted: 26-09-2016

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Multiple aids susceptible population in infected partners

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Abstract

A Stochastic model of HIV high risk is proposed in realistic. The essential elements of the model are behavioral determinants of susceptibility, progression of the disease from early stage to disseminated disease, detection by various factors. The stochastic models with high risk are predicted under, a highest-risk indicator of susceptibility is sex-ratio. We propose a stochastic model to study the damage process acting on the immune system that is non-linear. The mean of seroconversion time of HIV and its Variance are derived. A numerical example is given to illustrate the seroconversion times of HIV transmission.

Keywords: Alpha poisson process, infection, Mittag-Leffler distribution, threshold, seroconversion

1. Introduction

The dynamic transmission of HIV is quite complex and there is no other human infection which has the same epidemiological characteristics with a similar mode of transmission. For instance, the incubation period after infection with HIV is known to be extremely long and is measured in years rather than days (such as in the case of measles, for example). During this period, the individuals stay healthy and can unknowingly transmit the disease to others. In addition, the incubation period after infection with HIV is known to be extremely long and is measured in years rather than days (such as in the case of measles, for example). During this period, the individuals stay healthy and can unknowingly transmit the disease to others. In addition, although the disease is known as a sexually transmitted disease, it is also passed on from infected mothers to their babies, and from sharing infected syringes, which is common among injecting drug users. All these factors have made it difficult to understand how this epidemic spreads in the population. The growth movement among populations further increases the contact between individuals in different patches and, consequently, it might trigger more epidemics. Thus, the migration of people among subgroups has many significant consequences for the outcome the epidemic spread [1,2]. Indonesia in particular, as one of the most populous countries in the world, with high population mobility among its regions³. Indonesia, seems to have a high risk for the spread of the epidemic [4]. The number infected has increased sharply, and the prevalence among provinces varies widely.

Mathematical models based on the underlying transmission mechanism of HIV might help the medical and scientific communities understand better how the disease spreads in the community. Even though the actual data needed for the models might not be accurate or even available, such modelling is still vital in investigating how changes in the various assumptions and parameter values affect the course of the epidemic [5]. Therefore, by developing such mathematical models, we can to some extent anticipate its spread in different populations and evaluate the potential effectiveness of different approaches for bringing the epidemic under control, and thus help to devise effective strategies to minimize the destruction caused by this epidemic.

2. Description of Stochastic Model

Let us consider a susceptible population whose major mode of transmission is through is heterosexual activity. Assume that at time $t=0$, a new member of tested HIV negative enters the population and makes sexual contacts with member of susceptible. Let the sexual contacts occur at random time points which is assumed to follow the Alpha Poisson distribution⁶ with parameters 'a' and ' α ' which is given as

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$$P_{a,\alpha}(n, t) = \sum_0^{\infty} (-1)^k \binom{k+n}{k} \frac{(at)^{\alpha(k+n)}}{\Gamma(\alpha(k+n)+1)}, a > 0, 0 < \alpha < 1, n = 0, 1, 2 \dots$$

$$= \frac{e^{-(at)^\alpha} (at^\alpha)^k}{\Gamma(\alpha k + 1)}, a > 0, 0 < \alpha < 1$$

Let G(t) be the distribution function of the interarrival between the contacts which follows Mittag-Leffler distribution. The distribution function of Mittag-Leffler distribution⁷ is given by

$$G_{a,\alpha}(t) = \sum_0^{\infty} \frac{(-1)^k (at)^\alpha}{\Gamma(\alpha k + 1)}, t \geq 0, a > 0, 0 < \alpha \leq 1$$

Let the seroconversion time of the HIV of the individual the represented by the random variable T, we obtain the seroconversion distribution of HIV by a stochastic model based on the following assumptions.

1. Sexual contact is the only sources of HIV transmission.
2. An uninfected individual has sexual contacts with a HIV infected partner.
3. Damages to individuals are caused by transmission of HIV at each contact and the interarrivals between the contacts are independent, identically distributed random variables.
4. The damage process acting on the immune system of an infected individual is non-linear and cumulative.
5. The total damage caused exceeds a threshold level Y which itself is a random variable, the seroconversion and the person is recognised as infected.
6. The process that generates the contacts, the sequence of damages and threshold are mutually independent.

Let X_i be the antigenic diversity arising to due to HIV transmission during the i^{th} Contact and X_i s are i.i.d for $i=1,2,\dots,k$

$G(\cdot)$ = The c.d.f of X by taking $X_i = X$ for $i=1,2,\dots,k$

$g(\cdot)$ = The probability densit function of X_i

$V_k(t)$ = The probability of exactly k contacts in (0,t]

$g^*(s)$ = Laplace transform of $g(x)$

$g_k(\cdot)$ = The probability density function of random variable $\sum_0^{\infty} X_i$,

$$= \sum_0^{\infty} \frac{e^{-(at)^\alpha} (at^\alpha)^k}{\Gamma(\alpha k + 1)}, a > 0, 0 < \alpha \leq 1 P\{X_1 + X_2 + \dots + X_k < (Y_1, Y_2)\} = \int_0^{\infty} g_k(X) e^{(\mu_1 + \mu_2)x} P\{\sum_{i=1}^{\infty} X_i < (Y_1, Y_2)\} =$$

$$\{g^*(\mu_1 + \mu_2)\}^k$$

where $g_k(X) = P.d.f$ of $\sum_{i=1}^{\infty} X_i$ and $g^*(\mu_1 + \mu_2)$ Laplace is the Transformation of $g(x)S(t) = \sum_0^{\infty} V_k(t)\{g^*(\mu_1 + \mu_2)\}^k =$

$$\sum_0^{\infty} \frac{e^{-(at)^\alpha} (at^\alpha)^k}{\alpha k + 1} \{g^*(\mu_1 + \mu_2)\}^k = e^{-(at)^\alpha} \sum_0^{\infty} \frac{[(at^\alpha)^k g^*(\mu_1 + \mu_2)]^k}{\alpha k + 1}$$

$$= e^{-(at)^\alpha} e^{-[(at)^\alpha g^*(\mu_1 + \mu_2)]}$$

$$= e^{-(at)^\alpha} [1 - g^*(\mu_1 + \mu_2)]$$

$$L(t) = 1 - S(t) = 1 - e^{-\{a^\alpha [1 - g^*(\mu_1 + \mu_2)] t^\alpha\}}$$

Since the probability density function X_i follows Mittag-Leffler, then $g^*(\mu_1 + \mu_2) = \frac{a^\alpha}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} \Rightarrow 1 - g^*(\mu_2) =$

$$\frac{\mu_1^\alpha + \mu_2^\alpha}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}$$

$$\text{Thus } L(t) = 1 - e^{-\left\{\frac{a^\alpha (\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} t^\alpha\right\}}$$

The probability density function of seroconversion time T is

$$\Psi\Psi(t) = \left\{\frac{a^\alpha (\mu_1^\alpha + \mu_2^\alpha) \alpha}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}\right\} t^{\alpha-1} e^{-\left\{\frac{a^\alpha (\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} t^\alpha\right\}}$$

$t \geq 0, a > 0, 0 < \alpha \leq 1$ and $\mu_1 > 0, \mu_2 > 0$

Which is the form of three parameter weibull distribution.

Which is the kth convolution of $g(\cdot)$

Y = Random variable denoting the antigenic diversity threshold, which follows exponential distribution with parameter

$f(\cdot)$ = The probability density function of antigenic diversity threshold levels for the two sources, and $Y_1 \sim \exp(\mu_1), Y_2 \sim \exp(\mu_2)$ has exponential distribution with parameters μ_1, μ_2 so that

$$P[Y < y] = 1 - e^{-(\mu_1 + \mu_2)y}, y > 0, \mu_2 > 0, \mu_1 > 0$$

$P[X < Y]$ = The probability that damaged caused in a single contact is less than the threshold Y

$$S(t) = P[\text{no infection in } (0, t]] = P[T > t]$$

The model parameters are

a- Contact of the infected partner.

α - Intensity of the HIV of the infected partner.

μ - Antigenic diversity threshold

under the above assumptions with non linear damage process acting on the immune system, we have the following theorem.

3. Theorem

If the number of contacts is an Alpha poisson process with Parameters 'a' and ' α ' and inter contact time is a Mittag-Leffler distribution while the threshold level is an Competing exponential distribution with parameter ' μ_1, μ_2 ', then the probability density function of seroconversion time is a three parameter Weibull distribution

3a. Proof

$S(t) = P\{\text{no infection } (0, t]\} = P\{T > t\} = \sum_0^{\infty} P[\text{exactly k contact in } (0, t] \text{ with intensity } \alpha] \times p[\text{exactly k contact in } (0, t] \text{ with intensity } \alpha]$

$= \sum_0^{\infty} V_k(t) P\{\sum_0^{\infty} X_i < (Y_1, Y_2)\}$ Where $V_k(t)$ = probability of exactly k contacts in (0,t] with intensity 'a' which is the Alpha poisson distribution with parameter 'a' and ' α '

4. Probability of Seroconversion Time

The probability of seroconversion time is calculated for the various intervals by defining

$$p_i = \int_{t_i}^{t_{i+1}} \Psi\Psi(t)dt \text{ for } i = 1,2,3 \dots$$

$$p_i = \int_{t_i}^{t_{i+1}} \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)\alpha}{a^\alpha + \mu^\alpha} t^{\alpha-1} e^{-\left\{\frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}\right\}t^\alpha} dt \text{ for } i = 1,2,3 \dots$$

$t \geq 0, a > 0 >, 0 < \alpha \leq 0$ and $\mu_1 > 0, \mu_2 > 0$

$$p_i = \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)\alpha}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} \int_{t_i}^{t_{i+1}} t^{\alpha-1} e^{-\left\{\frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}\right\}t^\alpha} dt \text{ for } i = 1,2,3 \dots$$

$t \geq 0, a > 0 >, 0 < \alpha \leq 0$ and $\mu_1 > 0, \mu_2 > 0$

$$\text{Let } z = \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} t^\alpha \Rightarrow \alpha t^{\alpha-1} dt = \frac{dz}{\frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}} = \frac{dz}{c}, \text{ where } c = \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}$$

5. Performance Measures

The expected time to seroconversion time is

$$E(T) = \int_0^\infty \Psi(t) dt = \int_0^\infty \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)\alpha}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} t^{\alpha-1} e^{-\left\{\frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}\right\}t^\alpha} dt$$

$$= \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)\alpha}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} \int_0^\infty t^{\alpha-1} e^{-\left\{\frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}\right\}t^\alpha} dt$$

$$\text{Let } c = \frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)} \text{ and } \left(\frac{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}\right) t^\alpha = y \text{ then}$$

$$t^\alpha = \frac{y}{c} \Rightarrow \alpha t^{\alpha-1} dt = \frac{dy}{c} \text{ anyd } \alpha t^\alpha dt = \left(\frac{y}{c}\right)^{\frac{1}{\alpha}} \frac{dy}{c}$$

$$\therefore E(T) = \int_0^\infty e^{-y} \left(\frac{y}{c}\right)^{1/\alpha} \frac{dy}{c}$$

$$= \frac{1}{c^{1/\alpha}} \int_0^\infty e^{-y} y^{1/\alpha} dy = \frac{1}{c^{1/\alpha}} \Gamma\left(\frac{1}{\alpha} + 1\right)$$

$$E(T) = \left(\frac{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}\right)^{1/\alpha} \Gamma\left(\frac{1}{\alpha} + 1\right)$$

Similarly

$$E(T^2) = \left(\frac{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}\right)^{2/\alpha} \Gamma\left(\frac{2}{\alpha} + 1\right)$$

The variance of seroconversion Time is

$$V(T) = E(T^2) - E(T)^2$$

$$V(T) = \left[\frac{a^\alpha + (\mu_1^\alpha + \mu_2^\alpha)}{a^\alpha(\mu_1^\alpha + \mu_2^\alpha)}\right]^{2/\alpha} \left[\Gamma\left(\frac{2}{\alpha} + 1\right) - \Gamma\left(\frac{1}{\alpha} + 1\right)^2\right]$$

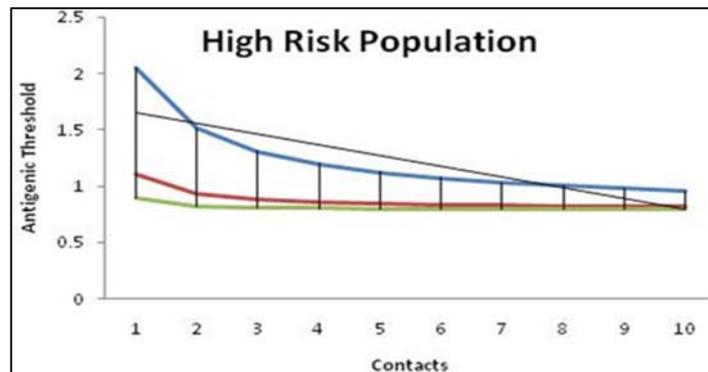


Fig 1

6. Conclusion

From the Figure (1), we observed that for fixed μ_2', μ_2 when 'a' (Contact rate) increases, the mean of time decreases. Also if 'a' is fixed and μ_2', μ_2 (Antigenic Diversity threshold) is allowed to increase then the mean time to seroconversion decreases. Also intensity of the human immuno Deficiency virus transmission of the infected partner increases, the mean time to seroconversion time decreases. The practical implication of the result is that the spread of HIV is faster as the intensity of the immune system is lower. It shows that from the above model to cross antigenic diversity threshold level of the fected high risk population attains the intensity of susceptible level in a quick period of time when the sexual contact increases. The Antigenic threshold level falls below the trend line when the population is in high sexual contact. This is observed for all the parameters of the high risk group population. Selection of individuals to be included in mass screening form entire population in future study models and programs

7. Acknowledgement

First Author like to thank heart fully to Prof. Dr. M.R. SRINIVASAN, Head, Department of Statistics, University of Madras: big thank you for your unflinching patient support, invaluable advice and continue supervision throughout the paper of this work. You indeed made me a continual thirst for research.

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