

**AIDS: THE EPIDEMIOLOGICAL SIGNIFICANCE
OF TWO DIFFERENT MEAN RATES OF
PARTNER CHANGE**

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1. Introduction

In their work on AIDS, Anderson, Medley, May et al. (1986) and May and Anderson (1987) have stressed the importance of a mean contact rate, c , which is the ratio of the population-weighted second (non-central) moment to the first moment (the mean \bar{c}) of the contact rates, for the initial growth of an epidemic. For a discrete heterogeneous population, let N_i be the number in population subgroup i , X_i the number of susceptibles and Y_i the number of infectives, $N_i = X_i + Y_i$. Let c_i be the contact rate per person in subgroup i . Then,

$$\bar{c} = \frac{\sum_i c_i N_i}{\sum_i N_i} = \frac{\sum_i c_i N_i}{N}, \quad (1)$$

$$c = \frac{\sum_i c_i^2 N_i}{\sum_i c_i N_i} = \bar{c} + \frac{\sigma^2}{\bar{c}}, \quad (2)$$

where σ^2 is the variance of the c_i 's about \bar{c} . Jacquez, Simon, Koopman et al. (1988) have stressed the importance of another mean contact rate, the mean infective weighted contact rate, \bar{c}_Y :

$$\bar{c}_Y = \frac{\sum_i c_i Y_i}{\sum_i Y_i} = \frac{\sum_i c_i Y_i}{Y}. \quad (3)$$

Note that both of these means change with time as the epidemic progresses and as subgroups change in size; we use c^o and \bar{c}_Y^o to indicate their initial values.

It is the purpose of this note to provide simple derivations for each that make clear the differences between the two.

We assume a population heterogeneous in contact rates with proportional mixing between discrete subgroups and with only one stage to the infectious period. Assume β is the probability of transmission per contact between an infective and a susceptible, μ is the rate constant for removal of individuals by all competing causes and k is the fractional rate (rate constant) at which infectives become sick enough with AIDS to be removed from the transmission process. Let U_i be the constant rate of recruitment of susceptibles into subgroup i . Then, the equations for the process become:

$$\dot{X}_i = -c_i \beta \frac{\sum_j c_j Y_j}{\sum_j c_j N_j} X_i - \mu X_i + U_i \quad (4a)$$

$$\dot{Y}_i = c_i \beta \frac{\sum_j c_j Y_j}{\sum_j c_j N_j} X_i - (\mu + k) Y_i. \quad (4b)$$

Equations (4) are the same as equations (5.1) of Anderson, Medley, May et al. (1986), except for the addition of a non-zero competing mortality rate constant, μ , and a constant recruitment rate, and are the same as equations (1) and (2) of Jacquez, Simon, Koopman et al. (1988) for proportional mixing and with one instead of m infective stages.

2. The Initial Growth of the Number of Infectives

We are concerned with the early phases of the epidemic after the introduction of a few infectives into a large population, so that one can make the assumption $X_i \approx N_i$.

Substitute N_i for X_i on the right side of equation (4b) and sum over i to obtain equation (5).

$$\dot{Y} = \beta \sum_j c_j Y_j - (\mu + k)Y \quad (5)$$

Using equation (3), substitute $\bar{c}_Y^o Y$ for $\sum_j c_j Y_j$ at the start of the process.

$$\dot{Y} = [\beta \bar{c}_Y^o - (\mu + k)]Y \quad (6)$$

Thus, at the start of the process, the total number of infectives grows exponentially with growth rate,

$$\frac{\dot{Y}}{Y} = \beta \bar{c}_Y^o - (\mu + k). \quad (7)$$

The initial growth rate of infectives depends on the initial distribution of infectives, which makes sense intuitively.

3. The Initial Growth of the Number of Contacts of Infectives

Let $V = \sum_i c_i Y_i$ be the total number of *contacts* of the infectives. Multiply equation (4b) by c_i and sum over i to obtain equation (8).

$$\dot{V} = \left(\beta \frac{\sum_j c_j^2 X_j}{\sum_j c_j N_j} - (\mu + k) \right) V \quad (8)$$

At the start of the process, (8) can be rewritten as,

$$\dot{V} = [\beta c^o - (\mu + k)]V. \quad (9)$$

Initially, the total number of contacts of infectives grows exponentially with growth rate,

$$\frac{\dot{V}}{V} = \beta c^o - (\mu + k). \quad (10)$$

Comparing (7) and (10), we have a simple result. The initial growth rates for number of infectives and for number of contacts of infectives are given by the same expression; they differ in the mean contact rate that appears in the expression.

- a. The infective weighted mean contact rate, \bar{c}_Y^o , is appropriate for *the growth of the number of infectives* at the start of the process.
- b. The ratio of the population-weighted second moment of the contact rate to the first moment is appropriate for *the growth of the number of contacts of the infectives*, at the start of the process.

At the onset of the epidemic, when $X_i \approx N_i$ and $\dot{X}_i \approx 0$ for all i , the above $V = \sum c_i Y_i$ is just a constant multiple of the $\lambda(t) = \beta \sum_i i Y_i(t) / \sum_i i N_i(t)$, which Anderson et al. (1986) work with in their Section 5.1. Anderson et al. (1986) use a different method to derive their version of relationship (10). However, they do misstate their result by writing that

the “number of infected” (instead of the number of infective contacts) has growth rate $\beta c^o - (\mu + k)$.

Anderson et al. (1986) emphasize the importance of the initial growth rate in an epidemiological model. First, $\ln 2$ divided by the initial growth rate is the doubling time, a number that can often be estimated directly from data. Furthermore, the sign of the initial growth rate tells whether the epidemic is increasing or decreasing. This is especially important for the growth rate of infective contacts, $\beta c^o - (\mu + k)$, because, as we will see, the sign of $\beta c^o - (\mu + k)$ determines the stability of the no-disease equilibrium. The condition $\beta c^o - (\mu + k) < 0$ can be restated as $R \equiv \beta c^o / (\mu + k) < 1$. The expression R is the important epidemiological threshold called the **reproductive number**. Since it equals the average number of contacts times the average infectivity per contact times the average incubation period $D = 1/(\mu + k)$, it measures the number of secondary infections produced, on the average, by one primary infection in a population of susceptibles.

4. An Apparent Paradox and its Resolution

On further examination the simple result of the previous Section seems to lead to an ambiguity. We can choose a distribution of contact rates and initial sizes of population subgroups so that the number of contacts of infectives has a positive growth rate (equation (9)), but introduce the infectives only into low contact rate groups so that the number of infectives has an initial negative growth rate (equation (6))! In this case, the number of infectives will be decreasing while the number of their contacts will be increasing. What will happen to the disease?

To resolve this apparent ambiguity and to better understand the relationship between these two means c and \bar{c}_Y , we examine more closely the behavior of system (4) in a neighborhood of the no-disease equilibrium: $X_i^o = U_i/\mu$, $Y_i^o = 0$, for $i = 1, \dots, n$. To accomplish this task, we will compute and study the solution of the linearized system at the no-disease equilibrium, since such a solution is *the* first order approximation to the solution of the non-linear system (4) in a neighborhood of the no-disease equilibrium.

First, note that if we start at the disease-free equilibrium, $N_i^o = X_i^o = U_i/\mu$, then $c^o = \sum_i c_i^2 U_i / \sum_i c_i U_i$. The Jacobian of system (4) with respect to $X_1, \dots, X_n, Y_1, \dots, Y_n$ at the no-disease equilibrium is:

$$\mathbf{D} = \left(\begin{array}{ccc|ccc} -\mu \mathbf{I} & & & & & -\mathbf{B} \\ \hline & & & & & \\ \mathbf{0} & & & & & \mathbf{B} - (\mu + k)\mathbf{I} \end{array} \right),$$

where \mathbf{B} is the rank-one matrix

$$\mathbf{B} = \frac{\beta}{\sum_j c_j U_j} \begin{pmatrix} U_1 c_1^2 & \cdots & U_1 c_1 c_n \\ \vdots & \ddots & \vdots \\ U_n c_n c_1 & \cdots & U_n c_n^2 \end{pmatrix}.$$

The $2n$ simple eigenvalues of \mathbf{D} are:

- 1) $-\mu$ with multiplicity n and
- 2) the n eigenvalues of $\mathbf{B} - (\mu + k)\mathbf{I}$, namely:

- 2a) $\beta (\sum_i c_i^2 U_i / \sum c_i U_i) - (\mu + k) = \beta c^o - (\mu + k)$ with multiplicity one and
 2b) $-(\mu + k)$ with multiplicity $n - 1$.

The linearization of system (4) at the no-disease equilibrium is

$$\begin{pmatrix} \Delta \mathbf{X} \\ \Delta \mathbf{Y} \end{pmatrix}' = \mathbf{D} \begin{pmatrix} \Delta \mathbf{X} \\ \Delta \mathbf{Y} \end{pmatrix}.$$

To simplify notation, write $x_i(t)$ for $\Delta X_i(t)$ and $y_i(t)$ for $\Delta Y_i(t)$. The solution of the linear initial value problem:

$$\begin{pmatrix} \mathbf{x} \\ \mathbf{y} \end{pmatrix}' = \mathbf{D} \begin{pmatrix} \mathbf{x} \\ \mathbf{y} \end{pmatrix}, \quad \mathbf{x}(0) = \mathbf{x}^o, \mathbf{y}(0) = \mathbf{y}^o,$$

is

$$\begin{pmatrix} \mathbf{x}(t) \\ \mathbf{y}(t) \end{pmatrix} = e^{\mathbf{D}t} \begin{pmatrix} \mathbf{x}^o \\ \mathbf{y}^o \end{pmatrix} = \begin{pmatrix} e^{-\mu t} \mathbf{x}^o + F(t) \mathbf{y}^o \\ e^{(\mathbf{B} - (\mu + k)\mathbf{I})t} \mathbf{y}^o \end{pmatrix},$$

for some matrix $F(t)$. We focus on the last n equations:

$$\begin{aligned} \begin{pmatrix} y_1(t) \\ \vdots \\ y_n(t) \end{pmatrix} &= e^{(\mathbf{B} - (\mu + k)\mathbf{I})t} \begin{pmatrix} y_1^o \\ \vdots \\ y_n^o \end{pmatrix} \\ &= \mathbf{P} \begin{pmatrix} e^{(\beta c^o - (\mu + k))t} & 0 & \cdots & 0 \\ 0 & e^{-(\mu + k)t} & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & e^{-(\mu + k)t} \end{pmatrix} \mathbf{P}^{-1} \begin{pmatrix} y_1^o \\ \vdots \\ y_n^o \end{pmatrix} \\ &= e^{-(\mu + k)t} \begin{pmatrix} y_1^o \\ \vdots \\ y_n^o \end{pmatrix} + \mathbf{P} \begin{pmatrix} e^{(\beta c^o - (\mu + k))t} - e^{-(\mu + k)t} & 0 & \cdots & 0 \\ 0 & 0 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & 0 \end{pmatrix} \mathbf{P}^{-1} \begin{pmatrix} y_1^o \\ \vdots \\ y_n^o \end{pmatrix}, \end{aligned} \tag{11}$$

where \mathbf{P} is the matrix of the eigenvectors of $\mathbf{B} - (\mu + k)\mathbf{I}$:

$$\mathbf{P} = \begin{pmatrix} c_1 U_1 & c_2 & \cdots & c_n \\ c_2 U_2 & -c_1 & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ c_n U_n & 0 & \cdots & -c_1 \end{pmatrix}, \tag{12}$$

and \mathbf{P}^{-1} is its inverse:

$$\mathbf{P}^{-1} = \frac{1}{c_1 \sum_i c_i^2 U_i} \begin{pmatrix} c_1 c_1 & c_2 c_1 & \cdots & c_n c_1 \\ c_1 c_2 U_2 & -\sum_{i \neq 2} c_i^2 U_i & \cdots & c_n c_2 U_2 \\ \vdots & \vdots & \ddots & \vdots \\ c_1 c_n U_n & c_2 c_n U_n & \cdots & -\sum_{i \neq n} c_i^2 U_i \end{pmatrix}. \tag{13}$$

One computes from (11), (12) and (13) that

$$\sum_i y_i(t) = \sum_i y_i^o \left[e^{-(\mu+k)t} + \frac{\bar{c}_Y^o}{c^o} \left(e^{(\beta c^o - (\mu+k))t} - e^{-(\mu+k)t} \right) \right]. \quad (14)$$

By (14), the instantaneous growth rate of $\sum y_i(t)$ at $t = 0$ is

$$\frac{\sum_i y_i'(0)}{\sum_i y_i^o} = \beta \bar{c}_Y^o - (\mu + k),$$

as we found in (7). Thus, immediately after the introduction of infected individuals into a disease-free system, the total number of infectives grows like $e^{(\beta \bar{c}_Y^o - (\mu+k))t}$. This initial growth rate may be positive or negative, but soon the growth of $\sum y_i(t)$ will be dominated by the term in $e^{(\beta c^o - (\mu+k))t}$ in (14).

If one looks at the initial growth rate of the number of *contacts* of the infected individuals, one finds from (11) that

$$\sum_i c_i y_i(t) = \left(\sum_i c_i y_i^o \right) e^{(\beta c^o - (\mu+k))t}, \quad (15)$$

confirming (10).

To summarize the results of these computations, if a few infectives are introduced into a disease-free population which satisfies proportional mixing, the total number of infected individuals has *initial* growth rate $\beta \bar{c}_Y^o - (\mu + k)$, while the total number of their contacts has *initial* growth rate $\beta c^o - (\mu + k)$. As time passes, the growth rate of the number of infectives tends to $\beta c^o - (\mu + k)$, at least for the linear approximation of the solution of (4).

We note that Jacquez, Simon, Koopman et al. (1988) show that the sign of the threshold $\beta c^o - (\mu + k)$ determines whether or not an endemic equilibrium exists. The calculation of the eigenvalues of the Jacobian D shows that the sign of $\beta c^o - (\mu + k)$ also determines the *local* stability of the disease-free equilibrium. See Lin (1989) for generalizations of this local stability result to more complex HIV systems.

Some final comments are in order on the applicability of these results. First, they depend on the assumption of proportional mixing. Furthermore, the basic processes at work are really stochastic, and deterministic models of epidemic processes can be good approximations to reality only when there are large numbers in all groups. At the start of an epidemic when there are few infectives, the basic stochastic nature should predominate; and even if equation (10) is greater than zero, there must be a non-zero probability of extinction of the epidemic.

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