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AIDS: SOME ASPECTS OF MODELING THE INSURANCE RISK

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ABSTRACT

This paper is an attempt to examine transmission and incubation period models. The number of HIV+ persons in the population is estimated. This estimate is applied to insurance company data in order to estimate the risk exposure of an individual company and the resulting reserve needs based on prior infections only.

1. INTRODUCTION

Recently a large number of papers and reports dealing with the potential impact of the AIDS epidemic on the insurance (especially life) industry have been published in various counties. These include Cowell and Hoskins [9] and Reese [36] in the U.S.A., Kolbye [24] in Denmark, The AIDS Working Party [41, 42] in the United Kingdom, Mann et al. [28] in Australia, and Castellino and Bridel [8] in Canada. These papers and reports generally provide information on the current state of knowledge of scientific work in the area of AIDS, forecast the number of cases over some future period, report on current and proposed legislation affecting the insurance industry, and recommend appropriate action by insurers for pricing insurance products and underwriting new insurance applicants. The report of the Society of Actuaries Task Force on AIDS [40] includes a number of papers of interest to insurance applications.

The scope of this paper is more limited than that of the papers cited above. In this paper we are primarily concerned with the risk of human immunodeficiency virus (HIV) infection in the population of persons who are currently insured. Whereas previous papers were concerned with adjustments to premium levels and changes to underwriting standards for future insureds, we focus on the additional risk due to infection that already exists amongst policyholders and that has not yet emerged in the form of death (or health) claims. This is a risk that was unanticipated at the time of issuance of insurance policies, at least until recently.

In order to recognize the emergence of future AIDS claims arising from the current book of business, it may be necessary to set aside a portion of a company's surplus to systematically fund this additional liability. An analogy may be drawn between this liability and an Incurred But Not Reported

Liability (IBNR). If the claim is deemed to have been "incurred" at the time of infection rather than the actual time of death (or disablement), all "incurred" claims require appropriate funding. Although an infected insured does not become an actual death claim until death occurs, a recognition of increased risk suggests that an increase in reserves (or some appropriation of surplus) is warranted. The additional liability may be an increase in existing policy reserves or a separate allocation of surplus. Nation-specific or state-specific insurance regulations will dictate the form of the increase in reserves.

A number of factors need to be considered in estimating the increased risk to a given insurer. These include the rate of HIV infection in the population; the lengths of the latency period, the incubation period and infective period; the level and rate of growth of reported AIDS cases in the general population; the level and rate of growth of AIDS-related death claims; the relative size of AIDS-related death claims as compared to other death claims; and the changes in the above factors that have occurred as a result of increased awareness, education and underwriting practices.

The following sections of this paper address these factors and develop a general methodology as well as give some numerical results. It is anticipated that the actuary will be able to use and adapt the techniques given in this paper to his or her own situation.

2. TRANSMISSION MODELS FOR HIV INFECTION

After a susceptible individual becomes infected with the HIV, the individual will become infectious (able to pass the infection to others) after some latency period. This latency period is believed to be very short (less than one month), because antibodies are detectable in the blood a few weeks after infection [2, 25]. The possibility of longer latency periods has been suggested by Ranki et al. [34] on the basis of observed seroconversion in stored serum samples obtained from individuals in high-risk groups. When antibodies are detectable in the blood, the individual is deemed to be "seropositive."

The incubation period is the period from infection (or perhaps seropositivity) until overt symptoms appear and a diagnosis of AIDS is made. Medical studies indicate that the incubation period has a mean length of several years, rendering the latency period more or less insignificant insofar as modeling is concerned. In Section 3, models for the incubation period are discussed in more detail.

In comprehensive survey papers, Isham [17] and Anderson [1] discuss mathematical models of the spread of HIV infection. In this section, we

depend heavily on these outstanding papers, which contain many references to other work that may be of interest to the reader.

In the analysis of the increase of the number of infectives, the latency period is usually ignored for convenience. Following the notation of Isham [17], we let a fixed population (or subpopulation) of n individuals at time t consist of $X(t)$ susceptibles and $Y(t)$ infectives, so that $X(t) + Y(t) = n$ at all times t. If the population mixes homogeneously, then the number of contacts between an infective and a susceptible must be proportional to both $X(t)$ and $Y(t)$ and the number of infectives is governed by the differential equation

$$\frac{dY(t)}{dt} = \alpha X(t)Y(t), \tag{2.1}$$

which has the simple solution

$$Y(t) = \frac{nY(0)}{Y(0) + [n - Y(0)]e^{-n\alpha t}}. \tag{2.2}$$

Equation (2.2) describes an S-shaped logistic curve and is essentially equivalent to that described by Cowell and Hoskins [9]. If the initial number of infectives is assumed to be small in relation to the population size, then for small values of t, the number of infectives can be approximated as

$$Y(t) \approx Y(0)e^{n\alpha t}, \tag{2.3}$$

which shows that in the early stages of an epidemic, the number of infectives grows exponentially.

A convenient statistic often quoted in connection with HIV infection is the "doubling time" of the epidemic, the number of years required for the number of infecteds or the number of observed AIDS cases to double. For the early stages of an epidemic satisfying (2.1), the doubling time of infecteds is constant. The doubling time of HIV infection in the U.S.A. and Europe has been estimated at approximately one year [29,30]. De Gruttola and Lagakos [10] discuss the interpretation of varying doubling times of observed AIDS cases with particular reference to the effects of changing models of the incubation period and of observed changes in the doubling time.

In the above development, no account was taken of the possibility that infectives will leave the infective class because of death, because of diagnosis and subsequent changes in behavior, or possibly because infectivity

decreases over time. If it is assumed that individuals leave the infective class at some rate ν, then the differential equation governing the number of infectives becomes

$$\frac{dY(t)}{dt} = \alpha X(t)Y(t) - \nu Y(t). \tag{2.4}$$

This equation is discussed extensively in Bailey [3] and was solved approximately by Kermack and McKendrick [22]. If we again assume that the number of infectives is small initially, the number of infectives is still approximately exponential in the early stages of the epidemic.

$$Y(t) \approx Y(0)e^{(n\alpha - \nu)t} \tag{2.5}$$

The beauty of this result becomes apparent in later sections, in which we estimate the rate of exponential growth and not the individual parameters n, α, and ν separately.

Isham [17] generalizes this model to one more appropriate to a closed group of male homosexuals. If each susceptible acquires new sexual partners at rate κ and becomes infected by any given infective partner with probability β, and the population mixes homogeneously, then the number of infectives is governed by the differential equation

$$\frac{dY(t)}{dt} = \frac{\beta\kappa}{n} X(t)Y(t), \tag{2.6}$$

which is approximately exponential for small values of t

$$Y(t) \approx Y(0)e^{\beta\kappa t}. \tag{2.7}$$

Furthermore, if infecteds are removed (voluntarily or otherwise) from the class of infectives in accordance with an exponential distribution of time in the infective class and play no further part in the spread of the disease, then the differential equation governing the number of infectives is

$$\frac{dY(t)}{dt} = \frac{\beta\kappa}{N(t)} X(t)Y(t) - \nu Y(t), \tag{2.8}$$

where $N(t) = X(t) + Y(t)$. When $Y(t)$ is small in the initial stages of the epidemic, the behavior of the epidemic is given by

$$Y(t) \approx Y(0)e^{(\beta\kappa - \nu)t}. \tag{2.9}$$

Anderson et al. [2] and van Druten et al. [43] generalize the models to allow each withdrawal to be either in the class of AIDS patients or remaining seropositive but not infectious. Bailey and Estriecher [3] allow for differences between infectives who will ultimately develop AIDS and the remainder who will remain seropositive indefinitely. Anderson et al. [2] also allow for migration to the infective class and death due to AIDS and other causes by using a system of five differential equations involving eight parameters. Anderson [1] also considers models in which an infected may move to a noninfectious state and return to being infectious but at a new rate. In the early stages of the epidemic an exponential model is still justified.

Isham [17] also considers heterogeneity amongst homosexual males by assigning a distribution to the rate of sexual activity, which may be expressed as a rate per unit time or a rate per sexual partner, with a further distributional assumption made about the length of sexual partnerships. Anderson et al. [2] explore the numerical ramifications of these more complex models. Fortunately, the behavior of the epidemic in the early stages is still exponential.

With respect to heterosexual transmission, Knox [23] considers transmission rates between twelve "behavior classes." Keissling et al. [21] and Stannat et al. [38] report on simulation studies based on assumed transmission between six groups, including bisexuals, homosexuals, heterosexuals, and prostitutes. Gonzalez [14] et al. divide individuals by sexual preference and consider intravenous drug use.

Finally, the most comprehensive models appear to be those of Dietz [11], which use 29 variables involving 42 parameters!

By now the reader will be aware that all the models presented up to this point are deterministic and not stochastic. Isham [17] shows that because the rate of spread of infection is nonlinear in the number of infectives, the deterministic model does not give exactly the behavior of a corresponding stochastic model. However, if the number of initial infectives is sufficiently small, the deterministic model gives a good approximation to a stochastic model [3, ch. 5]. Isham points out that different stochastic models may have the same deterministic approximation and that knowledge of the behavior of the mean of the process gives little guidance as to the stochastic nature of the process. Nevertheless, in this kind of study we are forced to make some not-yet-verifiable assumptions about the stochastic nature of the epidemic. This is done in later sections.

In the remainder of this paper, we assume exponential growth of the number of infectives. This implies that with respect to infection in the population, no changes in the rate of transmission due to changes in sexual and

drug use habits can be reflected. In the early stages of the epidemic, this is probably not an unreasonable first approximation. Because of the long incubation period, changes in transmission rates would not be apparent in the number of reported AIDS cases for some years.

3. INCUBATION AND INFECTION PERIOD MODELS

The time from infection to the development of AIDS is generally referred to as the incubation period. A variety of models have been developed recently based on medical studies. Lui et al. [27] used a Weibull distribution for transfusion-related infections and obtained a mean incubation period of 4.5 years. This study was criticized because of length-biased sampling, which was not recognized in the estimation procedure. Rees [35] used fit normal distributions heuristically, obtaining a mean of fifteen years. Blythe and Anderson [4] compare the properties of the Weibull and gamma distributions and conclude that using these distribution in this context will lead to similar results. Consequently, the choice between these distributions is not critical. Medley et al. [30] fit Weibull and gamma distributions to transfusion-related infections and recognize length-biased sampling. Using the Weibull distribution, they found a mean incubation period of 8.8 years for females and 5.6 years for males in the 5–59 age group. The mean of the combined group is 6.4 years. Kalbfleisch and Lawless [19, 20] reexamine the same data and conclude that the estimates of Medley et al. [30] for the incubation period are unreliable when the incubation period and the infection rate are estimated simultaneously.

The above studies are generally based on infections caused by blood transfusions. It has been argued that the incubation period for this group should be shorter than for other types of transmission because of the large amount of initial contamination associated with the transfusion of a large quantity of blood. On the other hand, it also has been argued that intravenous drug users should have a shorter incubation period due to impairment of the immune system as a result of drug use.

Based on a German study [6] of a mixture of drug users, homosexual males, female prostitutes and bisexuals, Panjer [33] obtains a mean incubation period of 6.4 years. This is consistent with Medley et al. [30], who obtained the same mean for blood transfusees. The model developed by Panjer [33] uses an incubation time that is the sum of three exponential random variables. A random variable that is the sum of exponential random variables has a generalized Erlang distribution. The three exponential random variables represent the time between diagnoses of intermediate stages: (1)

from seropositivity to lymphadenopathy syndrome (LAS), (2) from LAS to AIDS-Related Complex (ARC), and (3) from ARC to AIDS. Panjer [33] found that exponential distributions with means 1.2 years, 1.9 years and 3.3 years, respectively, adequately describe the observed data in the three stages of infection.

In the remainder of this paper, we use the model of Panjer [33] and the gamma model of Medley et al. [30] for estimating the number of infecteds in the population. More details about the distributions are given in the next section.

4. ESTIMATION OF THE NUMBER OF HIV INFECTIONS

4.1 Development of Formulas

In this section, we use the exponential growth model for the number of infectives, the generalized Erlang incubation period model of Panjer [33], and the gamma incubation period model of Medley et al. [30].

Let $M(t)$ be a Poisson process representing the random number of persons becoming seropositive before time t. We denote its mean by $\Gamma(t)$ and its intensity function by

$$\gamma(t) = \frac{d}{dt}\Gamma(t).$$

The Poisson assumption is a particularly convenient assumption. It means that the numbers of infections in disjoint intervals are stochastically independent and Poisson distributed. The Poisson assumption is not verifiable because we have only one sample path of the observed process.

Let $G(t)$ be the probability distribution function of the "incubation period." It denotes the probability that a person who has just become seropositive will have developed AIDS within t years.

Let $N(t)$ be a Poisson process denoting the random number of persons developing AIDS before time t. We denote its mean by $\Lambda(t)$. The Poisson process is also used by Medley et al. [30] and other previous authors. Then it can be shown that $N(t)$ is also a Poisson process with mean function

$$\Lambda(t) = \int_{-\infty}^{t} \gamma(s)G(t-s)ds \tag{4.1}$$

(see, for example, Ross [37]). If we assume an exponential growth model for the number of infectives, that is,

$$\gamma(t) = \beta e^{\alpha+\beta t}, \tag{4.2}$$

then the expected numbers of persons developing AIDS before time t becomes

$$\Lambda(t) = \int_{-\infty}^{t} \beta e^{\alpha+\beta s}\, G(t-s)ds = \beta e^{\alpha} \int_{-\infty}^{t} e^{\beta s}\, G(t-s)ds. \tag{4.3}$$

Let $-\infty = t_0 < t_1 < t_2 < \ldots < t_m$ denote the end points of time intervals and let n_i denote the number of reported AIDS cases in the time interval (t_{i-1}, t_i); $i = 1, 2, \ldots, m$ with $n = n_1 + n_2 + \ldots + n_m$.

We use the maximum likelihood method to obtain estimates $\hat{\alpha}$ and $\hat{\beta}$ of the parameters α and β, respectively. Because of the independence of the numbers of AIDS cases in disjoint time intervals, the likelihood function is written as

$$L = \prod_{i=1}^{m} \frac{\Delta_i^{n_i}}{n_i!} e^{-\Delta_i}, \tag{4.4}$$

where $\Delta_i = \Lambda(t_i) - \Lambda(t_{i-1})$ is the expected number of cases in time interval (t_{i-1}, t_i). Then the log-likelihood $\mathcal{L}$ may be written as

$$\mathcal{L} \propto \sum_{i=1}^{m} n_i \log \Delta_i - \Lambda(t_m). \tag{4.5}$$

The maximum likelihood estimates (MLEs) $\hat{\alpha}$ and $\hat{\beta}$ are the values of α and β that maximize $\mathcal{L}$.

Using (4.3), the following derivates may be obtained after some simplification:

$$\frac{\partial \mathcal{L}}{\partial \alpha} = n - \Lambda(t_m) \tag{4.6}$$

$$\frac{\partial \mathcal{L}}{\partial \beta} = \sum_{i=1}^{m} \frac{n_i}{\Delta_i} \frac{\partial \Delta_i}{\partial \beta} - \frac{\partial \Lambda(t_m)}{\partial \beta} \tag{4.7}$$

$$\frac{\partial^2 \mathcal{L}}{\partial \alpha^2} = -\Lambda(t_m) \tag{4.8}$$

$$\frac{\partial^2 \ell}{\partial \beta^2} = \sum_{i=1}^{m} \left\{ -\frac{n_i}{\Delta_i^2} \left(\frac{\partial \Delta_i}{\partial \beta} \right)^2 + \frac{n_i}{\Delta_i} \frac{\partial^2 \Delta_i}{\partial \beta^2} \right\} - \frac{\partial^2 \Lambda(t_m)}{\partial \beta^2} \tag{4.9}$$

$$\frac{\partial^2 \ell}{\partial B \partial \alpha} = -\frac{\partial}{\partial \beta} \Lambda(t_m). \tag{4.10}$$

Using the convenient substitutions $u(\beta\ ;\ t) = \Lambda(t)e^{-\alpha}$ and $\nu(\beta\ ;\ t) = \frac{\partial}{\partial \beta} u(\beta\ ;\ t)$, we obtain

$$\frac{\partial \ell}{\partial \beta} = \sum_{n=1}^{m} n_i \frac{\nu(\beta\ ;\ t_i) - \nu(\beta\ ;\ t_{i-1})}{u(\beta\ ;\ t_i) - u(\beta\ ;\ t_{i-1})} - e^{\alpha} \nu(\beta\ ;\ t_m) \tag{4.11}$$

Setting (4.6) and (4.11) to zero yields the likelihood equations

$$\hat{\alpha} = \log n - \log u(\hat{\beta}\ ;\ t_m) \tag{4.12}$$

and

$$\sum_{i=1}^{m} n_i \frac{\nu(\hat{\beta}\ ;\ t_i) - \nu(\hat{\beta}_{i-1})}{u(\hat{\beta}\ ;\ t_i) - u(\hat{\beta}\ ;\ t_{i-1})} = n \frac{\nu(\hat{\beta}\ ;\ t_m)}{u(\hat{\beta}\ ;\ t_m)}. \tag{4.13}$$

Equation (4.13) can be solved numerically for $\hat{\beta}$ and the result substituted into (4.12) to obtain $\hat{\alpha}$.

The asymptotic variance-covariance matrix of $(\hat{\alpha}, \hat{\beta})$ can be approximated as the matrix

$$\begin{bmatrix} \dfrac{\partial^2 \ell}{\partial \alpha^2} & \dfrac{\partial^2 \ell}{\partial \beta \partial \alpha} \\ \dfrac{\partial^2 \ell}{\partial \beta \partial \alpha} & \dfrac{\partial^2 \ell}{\partial \beta^2} \end{bmatrix}^{-1}$$

evaluation at $\alpha = \hat{\alpha}$ and $\beta = \hat{\beta}$. Because

$$\frac{\partial^2 \ell}{\partial \alpha^2} = -\frac{\partial}{\partial \alpha} \Lambda(t_m), \tag{4.14}$$

the first entry into the matrix is

$$\left. \frac{\partial^2 \ell}{\partial \alpha^2} \right|_{\alpha = \hat{\alpha}} = n.$$

Similarly, the second and third entries are

$$\left.\frac{\partial^2 \ell}{\partial\beta\partial\alpha}\right|_{\substack{\alpha=\hat{\alpha}\\ \beta=\hat{\beta}}} = e^{\hat{\alpha}\nu(\hat{\beta};t_m)}.$$

The fourth entry is more complex and can be evaluated numerically by using (4.9).

The MLEs and covariance-covariance matrix can be used to determine the MLEs and standard errors of all desired quantities.

A. *Generalized Erlang Model*

The generalized Erlang distribution of incubation times developed by Panjer [33] has the distribution function

$$G(t) = 1 - \sum_{r=1}^{r} \delta_j e^{-\mu_j t} \tag{4.16}$$

where $\delta_j = \prod_{k\neq j} \frac{\mu_k}{\mu_k - \mu_j}$. Substituting (4.16) into (4.3) yields

$$\Lambda(t) = e^{\alpha} u(\beta\,;\,t) \tag{4.17}$$

where

$$u(\beta\,;\,t) = e^{\beta t}\left\{1 - \beta\sum_{j=1}^{r} \frac{\delta_j}{\beta + \mu_j}\right\}1 \tag{4.18}$$

Finally

$$\nu(\beta\,;\,t) = tu(\beta\,;\,t) = e^{\beta t}\sum_{j=1}^{r} \frac{\mu_j\delta_j}{(\beta + \mu_j)^2} \tag{4.19}$$

and

$$\begin{aligned} w(\beta\,;\,t) &= \frac{\partial \nu(\beta\,;\,t)}{\partial\beta} \\ &= t\nu\,(\beta\,;\,t) - te^{\beta t}\sum_{j=1}^{r} \frac{\mu_j\delta_j}{(\beta + \mu_j)^2} + 2e^{\beta t}\sum_{j=1}^{r} \frac{\mu_j\delta_j}{(\beta + \mu_j)^3}. \end{aligned} \tag{4.20}$$

B. *Gamma Model*

One of the models considered by Medley et al. [30] is a gamma distribution with the distribution function

$$G(t) = \int_0^t q^p \frac{x^{p-1}e^{-qx}}{\Gamma(p)}\, dx \tag{4.21}$$

for the incubation period. From (4.1), it follows that

$$\Lambda(t) = \left(\frac{q}{q+\beta}\right)^p e^{\alpha+\beta t} = e^{\alpha}u(\beta\,;\,t) \tag{4.22}$$

where

$$u(\beta\,;\,t) = e^{\beta t}\left(\frac{q}{q+\beta}\right)^p. \tag{4.23}$$

Hence, it follows that

$$\nu(\beta\,;\,t) = u(\beta\,;\,t)\left(t - \frac{p}{q+\beta}\right) \tag{4.24}$$

and that

$$w(\beta\,;\,t) = u(\beta\,;\,t)\left[\left(t - \frac{p}{q+\beta}\right)^2 + \frac{p}{(q+\beta)^2}\right] \tag{4.25}$$

Although we do not give numerical values of asymptotic covariances in the next section, the above formulas may be of interest to the reader.

4.2 Numerical Results

In this section we experiment with numerical values for the parameters of the generalized Erlang and gamma distributions suggested by previous research. This is done in order to study the sensitivity of the results to varying distributional assumptions. The generalized Erlang model of Panjer [33] with parameters $\mu_1 = 0.86359$, $\mu_2 = 0.53478$ and $\mu_3 = 0.30000$ has a mean of 6.3612 years and a standard deviation of 3.9936 years. The Weibull model of Medley et al. [30] for all ages combined has a mean of 6.4059 years and a standard deviation of 2.8294 years and for adults (5–59 years) has a mean of 8.2307 years and a standard deviation of 3.6585 years.

In Table 1, results are given for the above generalized Erlang distribution and for the gamma distribution with means and variances based on the values of Medley [30]. For comparative purposes, we also use the generalized Erlang model with a mean of 8.2307 years by adjusting all parameters proportionally. Table 1 gives the maximum likelihood estimates of the number of HIV infecteds (including diagnosed AIDS cases) up to mid-1987 based on data available in early 1988. Due to delays in reporting times, values for the last half of 1987 were still unreliable at the time of the analysis.

TABLE 1

ESTIMATES OF NUMBER OF HIV INFECTEDS (1 JULY 1987)

Incubation Period Model	Gamma		Generalized Erlang	
Mean	6.4059	8.2307	6.3612	8.2307
Standard Deviation	2.8294	3.6585	3.9936	5.1672
U.S.A.				
No. of AIDS Cases	44,714	44,714	44,714	44,714
No. of HIV Infections	858,013	1,548,164	513,750	801,026
Infection Growth Rate	0.62379	0.62379	0.62379	0.62379
Canada				
No. of AIDS Cases	1,285	1,285	1,285	1,285
No. of HIV Infections	28,260	51,921	16,297	25,684
Infection Growth Rate	0.66216	0.66216	0.66216	0.66216
Australia				
No. of AIDS Cases	558	558	558	558
No. of HIV Infections	26,164	52,614	12,099	20,093
Infection Growth Rate	0.89493	0.89493	0.89492	0.89492

A number of observations can be made from Table 1. First, the predicted number of HIV infecteds varies dramatically between the four models. The generalized Erlang model produces estimates that are roughly half of those of the gamma model with roughly the same mean, primarily because the generalized Erlang distribution has a larger standard deviation, resulting in a flatter distribution. A larger proportion of the distribution is in the region being fitted, resulting in a smaller proportion in the region being forecast. Second, the choice of the mean has a significant influence in the forecast of the number of infecteds. The standard errors associated with the estimates of Medley et al. [30] are quite large, making each of the gamma model estimates quite plausible. Similar comments hold for the generalized Erlang model and the estimates of Panjer [33]. An increase in the mean, of about 30 percent, while holding the coefficient of variation constant, causes estimates of the number of infecteds to increase by about 80 percent in the case

of the gamma distribution and about 60 percent in the case of the generalized Erlang distribution.

The corresponding estimates coming from other sources are of the order of 1,000,000 for the U.S.A., 50,000 for Canada, and 50,000 for Australia. For Canada and Australia, these would appear to be at the upper end of our four estimates but cannot be rejected. For the U.S.A. the estimate of one million falls roughly in the middle of the four estimates.

The variation in these estimates indicates that any single estimate should be viewed with considerable suspicion. Sensitivity of the estimate to changes in model assumptions should accompany any estimates. Furthermore, confidence intervals of any estimates should be given because they provide some indication of the reliability of the estimates.

An additional point of interest and cause for concern about the data is the observed infection growth rate (β of Equation 4.2). Although it is stable across models for Canada and the U.S.A., it is very different (much higher) for Australia. A possible explanation is that in the early years (up to mid-1984) the level of unreported cases was significantly higher than that in later years when the general level of knowledge of AIDS increased. This hypothesis is supported by the data for Australia, in which a major jump in reported AIDS cases occurs between the first half and second half of 1984.

When the fitted AIDS cases are compared with the observed AIDS cases in Tables 2, 3 and 4, it can be seen that the number of fitted AIDS cases exceeds the number of observed AIDS cases in the early and late years, and is less than the number of observed cases in the middle years, indicating that the actual incidence of AIDS grows at a rate that is slower than would be inferred by the models used. A variety of plausible explanations could account for this poor fit; they include the following:

(i) *Reporting Variations.* If the proportion of unreported AIDS cases varies over the period of observation, the shape of the reported cases is distorted. Because the number of unreported cases is unobservable, it would be difficult to pursue this further except to note that it is entirely plausible and consistent with an increasing knowledge of AIDS and its diagnosis over time. External studies may be useful in evaluating the level of underreporting.

(ii) *Alternate Incubation Period Models.* Models for the incubation period used in this paper and other models cited in Section 3 are chosen for mathematical convenience and because they fit available data well. Further research may reveal that some characteristics of these distributions are inappropriate.

(iii) *Variable Infectivity.* Hyman and Stanley [16] discuss the hypothesis that the infectivity of an infected individual varies over the course of the disease. The amount

of free virus increases dramatically in the first few weeks following infection (Francis et al. [12], Sulahuddin et al. [39]) and then drops off sharply as the production of antibodies is adequate to reduce the number of viruses to a very low level. Over the next few years the number of free viruses may increase slowly for some time and then more rapidly as the immune system collapses (Lange et al. [26], Goedert et al. [13]) when the level of infectivity is again very high. Grant et al. [15], Padian et al. [31], and Hyman and Stanley [16] suggest that the chances of infection from a single sexual contact are very small (less than one percent) for most of the infection but may be dramatically higher in the first few weeks and after fully developed AIDS begins. Such levels of variability of infectivity can have a dramatic effect on the number of observed AIDS cases in the early part of the epidemic.

(iv) *Variable Transmissions Rates.* The majority of observed AIDS cases arise from homosexual men, drug abusers, female prostitutes, and blood transfusees. It is generally believed that the progress of the disease will be determined by contacts amongst the first three groups and between these groups and the balance of the susceptible population. The risk of transmission depends upon the nature of the sexual contact. Receptive anal intercourse has been recognized as the sexual act with the highest risk. Johnston [18] includes many references to studies of homosexual men and the associated risks of various sexual acts. Intraveneous drug users play a significant role in the U.S.A. and some European countries. Clearly, the risk of infection from a single occurrence of drug infection with a needle shared by another drug user is different than that from a sexual act. The annual transmission rate depends upon the number of contacts as well as the risk per single contact and the density of the infection in the particular subpopulation under consideration. Johnston [18] provides an outstanding overview of the social aspects of AIDS.

Futher refinements to the models may be justified as more data become available.

5. APPLICATION TO INDIVIDUAL COMPANIES

In general, the primary risk of AIDS-related claims in the future is in the homosexual male population and in the heterosexual population as spread from the homosexual, drug-using and female prostitute populations. In order to apply a population-based model to an insured population, it is useful to assume that key characteristics of the two populations are the same. Because drug users are less likely to be insured than male homosexuals, this assumption is violated by at least one subpopulation. However, in the simple models of Section 4, only two population characteristics were significant, namely, α and β, the growth parameters. The parameter α is associated with the level of HIV infections, while β is associated with the annual rate of growth of HIV infections. As a first approximation, it is probably reasonable to assume that the growth rate β in an insured population is the same as that

TABLE 2

RESULTS FOR U.S.A.*

Year	Half-Year	AIDS Cases Observed	AIDS Cases Fitted	HIV Cases Predicted	AIDS Cases Fitted	HIV Cases Predicted
			Gamma Incubation Period Model			
			Mean = 6.4059, S.D. = 2.8294		Mean = 8.2307, S.D. = 3.6585	
to 1981	1	88	1,059	20,327	1,059	36,676
	2	183	388	7,440	388	13,424
1982	1	365	530	10,163	530	18,337
	2	650	723	13,882	723	25,048
1983	1	1,229	988	18,963	988	34,216
	2	1,600	1,350	25,904	1,350	46,739
1984	1	2,478	1,844	35,385	1,844	63,845
	2	3,234	2,519	48,336	2,519	87,213
1985	1	4,456	3,441	66,027	3,441	119,133
	2	5,701	4,700	90,193	4,700	162,736
1986	1	7,063	6,421	123,204	6,421	222,298
	2	8,234	8,771	168,297	8,771	303,660
1987	1	9,433	11,981	229,894	11,981	414,801
Total		44,714	44,714	858,013	44,714	1,548,125
			Generalized Erlang Incubation Period Model			
			Mean = 6.3612, S.D. = 3.9936		Mean = 8.2307, S.D. = 5.1672	
to 1981	1	88	1,059	12,171	1,059	18,976
	2	183	388	4,455	388	6,945
1982	1	365	530	6,085	530	9,487
	2	650	723	8,312	723	12,960
1983	1	1,229	988	11,354	988	17,703
	2	1,600	1,350	15,510	1,350	24,183
1984	1	2,478	1,844	21,187	1,844	33,034
	2	3,234	2,519	28,942	2,519	45,125
1985	1	4,456	3,441	39,535	3,441	61,641
	2	5,701	4,700	54,004	4,700	84,202
1986	1	7,063	6,421	73,771	6,421	115,021
	2	8,234	8,771	100,771	8,771	157,120
1987	1	9,433	11,981	137,654	11,981	214,627
Total		44,714	44,714	513,750	44,714	801,026

*Source: "AIDS Weekly Surveillance Report," CDC, February 1, 1988.

TABLE 3

RESULTS FOR CANADA*

Year	Quarter	AIDS Cases Observed	AIDS Cases Fitted	HIV Cases Predicted	AIDS Cases Fitted	HIV Cases Predicted
Gamma Incubation Period Model						
			Mean = 6.4059, S.D. = 2.8294		Mean = 8.2307, S.D. = 3.6585	
to 1979	4	1	9	197	9	362
1980	1-4	3	8	185	8	340
1981	1-4	6	16	359	16	659
1982	1-4	22	32	695	32	1,277
1983	1	14	12	258	12	475
	2	15	14	305	14	560
	3	11	16	360	16	661
	4	14	19	425	19	780
1984	1	28	23	501	13	921
	2	33	27	591	27	1,087
	3	40	32	698	32	1,282
	4	42	37	824	37	1,513
1985	1	59	44	972	44	1,785
	2	74	52	1,147	52	2,107
	3	91	62	1,353	62	2,486
	4	94	73	1,597	73	2,934
1986	1	90	86	1,884	86	3,462
	2	119	101	2,224	101	4,085
	3	118	119	2,624	119	4,821
	4	133	141	3,096	141	5,689
1987	1	127	166	3,654	166	6,713
	2	151	196	4,311	196	7,921
Total		1,285	1,285	28,260	1,285	51,921
Generalized Erlang Incubation Period Model						
			Mean = 6.3612, S.D. = 3.9936		Mean = 8.2307, S.D. = 5.1672	
to 1979	4	1	9	114	9	179
1980	1-4	3	8	107	8	168
1981	1-4	6	16	207	16	326
1982	1-4	22	32	401	32	632
1983	1	14	12	149	12	235
	2	15	14	176	14	277
	3	11	16	208	16	327
	4	14	19	245	19	386
1984	1	28	23	289	23	456
	2	33	27	341	27	538
	3	40	32	402	32	634
	4	42	37	475	37	748
1985	1	59	44	560	44	883
	2	74	52	661	52	1,042
	3	91	62	780	62	1,230
	4	94	73	921	73	1,451
1986	1	90	86	1,087	86	1,713
	2	119	101	1,282	101	2,021
	3	118	119	1,513	119	2,385
	4	133	141	1,786	141	2,814
1987	1	127	166	2,107	166	3,321
	2	151	196	2,486	196	3,918
Total		1,285	1,285	16,297	1,285	25,684

*Source: "Update: AIDS in Canada," LCDC, Ottawa, February, 1988.

TABLE 4

RESULTS FOR AUSTRALIA*

Year	Half-Year	AIDS Cases Observed	AIDS Cases Fitted	HIV Cases Predicted	AIDS Cases Fitted	HIV Cases Predicted
			Gamma Incubation Period Model			
			Mean = 6.4059, S.D. = 2.8295		Mean = 8.2307, S.D. = 3.6585	
to 1982		1	10	466	10	938
1983	1	1	6	263	6	529
	2	5	9	412	9	828
1984	1	6	14	644	14	1,295
	2	36	21	1,008	21	2,026
1985	1	62	34	1,576	34	3,169
	2	51	53	2,466	53	4,958
1986	1	95	82	3,857	82	7,756
	2	128	129	6,034	129	12,133
1987	1	173	201	9,439	201	18,981
Total		558	558	26,164	558	52,614
			Generalized Erlang Incubation Period Model			
			Mean = 6.3612, S.D. = 3.9936		Mean = 8.2307, S.D. = 5.1672	
to 1982		1	10	216	10	358
1983	1	1	6	122	6	202
	2	5	9	190	9	316
1984	1	6	14	298	14	495
	2	36	21	466	21	774
1985	1	62	34	729	34	1,210
	2	51	53	1,140	53	1,893
1986	1	95	82	1,784	82	2,962
	2	128	129	2,790	129	4,634
1987	1	173	201	4,365	201	7,248
Total		558	558	12,099	558	20,093

*Source: NH & MRC Special Unit in AIDS Epidemiology and Clinical Research, February 11, 1988.

in the population as a whole. This growth rate should be generally independent of geographical or other subdivisions. If the above arguments are accepted, then it is only necessary to estimate the value of α for the insured population. This can be done for life insurance by comparing the number of AIDS-related death claims to the number of AIDS deaths in the population.

Underreporting of AIDS-related claims occurs in the general population as well as in the insured population. If the level of underreporting in the two populations is the same, the estimates produced will be of reported cases only. Although increased awareness of AIDS should increase the reporting level, an increased stigma associated with AIDS may actually decrease the tendency to report a claim as AIDS-related. This may be especially true in health insurance. In the ACLI/HIAA report on claims paid in 1986, Carroll

[7] lists the following reasons for understatements of the number of AIDS-related life and health claims:

1. "Death certificate may not show cause of death when death has occurred due to disease. With relatively few exceptions this is true of New York City. This is extremely critical since currently about 25 percent of all AIDS cases reported to the CDC in the United States have occurred among New York City residents."
2. "Disease may not be recognized as AIDS-related."
3. "Diagnosis may not yet have been made at time of claim (primarily health insurance)."
4. "Opportunistic disease may be shown as diagnosis or cause of death and not picked up by insurer."
5. "Diagnosis or cause of death may not be precisely stated. For example, pneumocystis carinii pneumonia (PCP) may be given only as pneumonia, Kaposi's sarcoma (KS) as cancer."
6. "Diagnosis may be intentionally misstated (especially health insurance)."
7. "Companies have had little financial incentive to determine cause of death for deaths beyond the contestable period."
8. "Companies have had little financial incentive to determine precise diagnosis for health insurance benefits which are payable on the basis of a general diagnosis or are payable regardless of diagnosis."
9. "Claim administration systems involving third party administrators, self-administered cases, and other decentralized systems may be such that the company is unable to identify AIDS-related claims (primarily group health insurance)."
10. "Companies may not have established systematic tracking of AIDS-related claims prior to 1986. In this regard, some companies indicated that their data covered only part of the year; other companies may be retroactively attempting to identify AIDS-related claims."
11. "Company efforts to identify AIDS-related claims may have been inadequate."

6. RELATIVE SIZES OF AIDS-RELATED CLAIMS

The risk of antiselection by infected persons or persons in high-risk groups is apparent. Evidence of significant antiselection prompted insurers to dramatically reduce nonmedical limits on life insurance in 1986 and 1987.

Reese [36] reports that in a survey of life insurance company AIDS claims, average sizes of AIDS claims were larger than the averages for all claims by a factor of seven in 1985 and a factor of five in 1986. In the ACLI/HIAA survey of claims paid in 1986, Carroll [7] notes that the average individual life AIDS-related death claim was about four times larger than the average size death claim. For group life insurance, the AIDS-related death claims were about twice as large on average as the average size death claims.

Note that these numbers may overestimate the amount of antiselection because death claims on older policies are smaller. Policies issued as a result

of antiselection should be newer policies. Because more recent issues are generally larger, death claims of recently issued polices would be expected to be larger than average. For a more accurate comparison, policies should be identified by year of issue. Furthermore, at least theoretically, the amount of antiselection should be based on the net amount at risk, not the full face amount. For recent issues this will not be a major problem, because reserves are relatively small at early observations.

To develop a model for reserving purposes, it is necessary to study the distribution of insured amounts by HIV-infection persons. This will vary by company because it depends upon many variables affecting the distribution of sizes of the insured amounts for non-HIV-infected persons. No attempt is made in this paper to develop any specific claim amount model.

7. ADDITIONAL RESERVE REQUIREMENTS

The papers discussed in Section 1 of this paper generally approach the question of reserving by using forecasts of the excess deaths for each year in the future and developing appropriate adjustments to mortality assumptions. In this section we attempt to complement these papers by developing a macro model for the aggregate excess AIDS costs that is consistent with the models used in Section 4.

Suppose that the number of infections and the number of AIDS cases are Poisson processes as described in Section 4 up to Formula (4.3). Then the number of new AIDS cases between any times t_1 and t_2 is Poisson distributed with mean $\Lambda(t_2) - \Lambda(t_1)$ by using the notation of Section 4. Furthermore, if the time from developing AIDS to death is described by a distribution function $H(t)$, then the number of AIDS-related deaths between times t_1 and t_2 is also Poisson distributed with mean

$$\int_{t_1}^{t_2} \int_{-\infty}^{t} \lambda(s)H(t - s)dsdt$$

where $\lambda(t) = \dfrac{d}{dt}\Lambda(t)$.

Because of the introduction of AIDS testing for larger policy amounts in 1986 and 1987, the incidence as well as the amounts will be dramatically different for policies issued before and after. Consequently, it is important to recognize these time periods separately. The number of AIDS-related

death claims between t_1 and t_2 arising from infections before t_0, $t_0 < t_1 < t_2$ is Poisson distributed with mean

$$\int_{t_1}^{t_2} \int_{-\infty}^{t_0} \lambda(s)H(t - s)dsdt.$$

Similarly, the number of AIDS-related death claims between t_1 and t_2 arising from infection after t_0 is Poisson distributed with mean

$$\int_{t_1}^{t_2} \int_{t_0}^{t} \lambda(s)H(t - s)dsdt.$$

Let $f_B(x)$ and $f_A(x)$ denote the distribution of claim sizes in the insured population before and after the introduction of HIV testing. Then the AIDS-related losses (ignoring reserves) for the time period t_1 to t_2 for issues before and after testing have compound Poisson distributions (compare Bowers et al. [5]) with appropriate Poisson frequencies and claim amount distributions. Furthermore, because of the additivity of the compound Poisson distribution, the distribution for several time periods combined, for before and after testing combined, or for several subportfolios combined will remain compound Poisson. Panjer [32] discusses these concepts in a different context and provides a simple algorithm for evaluating these distributions.

The use of these distributions allows the actuary to compute both the mean and quantiles of the distribution of the ultimate losses due to past infections (that is, analogous to an IBNR) to establish a reserve with an appropriate safety margin.

The reserve from past infections can be run off annually by adjusting for the difference between actual and expected. If a past infection is viewed as an IBNR claim, then a reserve must be established immediately out of surplus to fund future claims arising from past infections.

For future infections, mortality and morbidity tables for new issues need to be adjusted appropriately to reflect the revised underwriting practices.

If the approach of extrapolating excess claims in the future (both past and future infections) and adjusting reserve factors for all policies is used, an inequity will arise as new issues will fund past infections on old policies. An applicant could, at least theoretically, obtain a cheaper policy from a new company or a new independent block of business than from an existing

block of business that includes policies issued prior to the introduction of testing.

For these reasons, the author believes that it is important to separate past infections from future infections and that full funding of past infections is required immediately, whereas reserving for future issues will be taken care of by adjustments to the underlying mortality or morbidity tables.

Past infections can be funded in full in advance in aggregate or by adjusting mortality factors for old policies. The aggregate approach described in this section provides a guidelines as to how much the company's reserve should increase in total.

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DISCUSSION OF PRECEDING PAPER

FUNG-YEE CHAN:

It is interesting to see the modeling of AIDS as a rate of HIV infection followed by an incubation period. Although the infection rate may be environmental, subject to societal influence, education, and risk-taking attitudes, the incubation is biological and should be common to all countries. In this paper, Professor Panjer assumes an exponential infection rate. His incubation distributions (gamma and Erlang) use the same distribution parameters for all three countries: U.S.A., Canada, and Australia.

I would like to suggest the following reasoning: If the AIDS cases are described by the product of an infection factor and an incubation factor that is common to different countries, the ratios of observed AIDS cases are the ratios of the infection rates of the different countries. These ratios are important because they grow exponentially if the observed AIDS cases, or the HIV cases, grow exponentially. On the other hand, if the observed AIDS cases or the HIV cases grow polynomially with time, say, cubically, then the ratios should stay quite flat with time. Given that the infection rates are environmental and that environments change only rather slowly, I would suspect that these ratios did not change drastically during the past few years.

We construct in Table 1 three time series of observed AIDS cases using the data extracted from Panjer's paper. We can smooth the time series using the mean of three adjacent values. The resulting series and their ratios (starting one observation later and terminating one observation sooner) are given in Table 2 and Table 3, respectively.

TABLE 1

OBSERVED AIDS CASES IN U.S., CANADA AND AUSTRALIA

	U.S.		Canada		Australia	
	Half-Year		Half-Year		Half-Year	
Year	1	2	1	2	1	2
1983	1,299	1,600	29	25	1	5
1984	2,478	3,234	61	82	6	36
1985	4,456	5,701	133	185	62	51
1986	7,063	8,234	290	251	95	128
1987	9,433		278		173	

TABLE 2

AIDS CASES SMOOTHED BY [lag(y, −1) + y + lag(y, 1)]/3

	U.S.		Canada		Australia	
	Half-Year		Half-Year		Half-Year	
Year	1	2	1	2	1	2
1983		1792.33		38.34		4.00
1984	2437.33	3389.33	56.00	92.00	15.67	34.67
1985	4463.67	5740.00	133.34	175.67	49.67	69.33
1986	6999.33	8243.33	215.00	246.00	91.33	132.00

TABLE 3

RATIOS OF TIME SERIES

	U.S./Canada		Canada/Australia	
	Half-Year		Half-Year	
Year	1	2	1	2
1983		46.76		9.58
1984	45.32	36.84	3.57	*2.65*
1985	*33.48*	*32.68*	*2.68*	*2.53*
1986	*32.56*	*33.51*	*2.35*	1.86

The U.S.A./Canada ratio shows a slow decline and a stabilized ratio around 33 in 1985 and 1986. The Canada/Australia ratio shows a rapid decrease and a stabilized ratio around 2.5 about the same time.

To determine whether the fitted data of Panjer's would show the same kind of features, we also compute the means of three adjacent values of Panjer's fitted values even though they are already smoothed by formulas. They are given in Table 4. We see that the features as noted in Table 3 are lost in Panjer's model, which instead shows an overall gentle decline with time.

TABLE 4

RATIOS OF PANJER'S FITTED SERIES

	U.S.A./Canada		Canada/Australia	
	Half-Year		Half-Year	
Year	1	2	1	2
1983		37.68		3.83
1984	37.10	38.26	3.50	2.96
1985	36.89	35.78	2.68	2.41
1986	34.18	33.59	2.20	1.96

MICHAEL J. COWELL:

Professor Panjer's paper is a welcome and significant addition to the literature on modeling the spread of the human immunodeficiency virus (HIV) in North America. This discussion addresses the topics covered in Sections 2, 3 and 4 of the paper.

The summer of 1989 marked the 100,000th case of AIDS in the U.S. reported to the Centers for Disease Control (CDC). The 10,000 mark was reached in early 1985, and the 1,000th case was reported in 1982. The CDC estimates that "Currently about 1 million persons in the United States are infected with HIV" [1]. If this estimate is reasonably accurate, at current rates of progression from initial infection to the full-fledged clinical symptoms of AIDS and in the absence of a major medical breakthrough, the number of AIDS cases will reach one million sometime in the late 1990s.

In developing our model of HIV infection and AIDS mortality in 1987 [2], Walter Hoskins and I concluded that predicting the course of the epidemic depended on knowing the dynamics of the spread of HIV and the rate of progression of the disease in a cohort of newly infected individuals.

In Section 2 of his paper, Professor Panjer explores, with more mathematical elegance than Walter Hoskins and I, the notion that the spread of HIV infection in a homogeneously mixing population may be modeled by an S-shaped logistic curve. This assumption is often made in the study of "classical" epidemics such as influenza. When we published our report, we had no empirical evidence that this assumption would necessarily be valid for HIV infection with its long incubation period.

Information published over the past two years on the changing prevalence of HIV infection over time among subjects in the San Francisco Men's Health Study and in the New York State Drug Abuse Study lends strong evidence to the logistic assumption, at least among these two samples from the highest risk groups. Considerably more time will be required to determine whether the assumption will continue to be valid as HIV infection spreads more widely, and increasingly, into the non-drug-abusing heterosexual population.

In Section 3, Professor Panjer explores the fit of both Weibull and gamma distributions to a number of clinical studies of progression from initial HIV infection to the development of AIDS. He cites studies with mean progression times ranging from 6 to 9 years, seemingly a narrow range, although the implications of the difference can be substantial.

At the time we were studying this progression, Walter Hoskins and I depended largely on the University of Frankfurt data and information newly emerging from the San Francisco City Clinic Study. More recent information from the latter study, which traces HIV infection through frozen blood samples all the way back to 1978, tends to support a mean progression time of 9 or 10 years. Some lengthening of the incubation period in the past year or two has been attributed to the more widespread application of retroviral drugs such as AZT. Increasing numbers of HIV-infected individuals are being administered such drugs in the earlier stages of infection, before reaching full-fledged AIDS. This trend, and other advances in treatment, will likely lengthen the mean progression time further.

Also, as more data become available on other than the principal risk groups, there is evidence that progression rates may vary considerably by such factors as sex, number of exposures to the virus, and health at time of initial infection.

In Section 4, Professor Panjer presents a major breakthrough in the technique of "back-estimating" the number of HIV infections from the number of AIDS cases and knowledge of the distribution of the progression, a process that has been compared to measuring an iceberg by analyzing its tip.

Walter Hoskins and I constructed a crude "back estimation" method by (i) assuming a series of new annual HIV infections, Hz, for the years through 1986 that followed the logistic assumption of prevalence; (ii) calculating the number of new AIDS cases that would follow from the distribution of progression; and (iii) solving the resulting system of simultaneous equations to produce the number of AIDS cases reported to the CDC. The problem with our approach, especially in the early years of the epidemic, was that the largest number of new infections is matched to the smallest component of progression. The result is thus extremely unstable, with seemingly minor changes in the assumed distribution of progression producing widely different estimates of total HIV infections. We concluded that the number of HIV infections at year-end 1987 was in the order of magnitude of a million, but we could not say with certainty whether it was 600,000 or 1.5 million.

Using far more robust statistical approaches, Professor Panjer estimates as of mid-1987, under the assumption of 8.2 years mean progression, that the number of HIV infections in the U.S. may be as low as 800,000 and as high as 1.5 million. The wide variations that can result from seemingly minor changes in assumptions about the distribution of progression from initial infection to AIDS is well illustrated in his Table 1. An increase of 28 percent in mean progression time from 6.4 to 8.2 years results in an 80

percent increase in the estimate of HIV infections under the gamma distribution, and a 56 percent increase under the generalized Erlang distribution. One point here is that as the mean progression time increases with the more widespread application of retroviral drugs, the number of reported AIDS cases portends a correspondingly larger number of HIV infections.

Education continues to be the primary method for controlling the spread of HIV infection, although medical intervention is beginning to slow the progression of the disease among many who are already infected. Recent progress in the perceived effectiveness of retroviral drugs also seems to have had a favorable effect on the willingness of groups at high risk of infection to have their HIV status tested. The CDC has indicated its intention to conduct nationwide sampling of HIV prevalence in the early 1990s. In the meantime, those of us responsible for projecting the impact of the epidemic on our life and health insurance institutions will have to continue to rely on the modeling work available. We are indebted to Professor Panjer for his significant contributions to this research.

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ELIAS S. W. SHIU:

I wish to comment on Section 2 of this interesting paper.

The classical principle of mass action, formulated by Guldberg and Waage more than 120 years ago, is that the rate of a chemical change is at any instant proportional to the product of the effective concentrations of the reactants at that instant. Equation (2.1), with $X(t)$ defined as $n - Y(t)$,

$$\frac{dY(t)}{dt} = \alpha[n - Y(t)]Y(t)$$

may be viewed as a consequence of this principle. This differential equation is now part of the course of study for the Course 161 examination ([7], [1, p. 531, Exercise 18.18]), and in each of the Course 161 examinations so

far, there has been at least one question on the logistic curve. To solve the differential equation, observe that

$$\frac{1}{(n - Y)Y} = \frac{1}{n}\left(\frac{1}{n - Y} + \frac{1}{Y}\right).$$

For small $Y(t)$, Equation (2.1) is approximately

$$\frac{dY(t)}{dt} \approx \alpha n Y(t),$$

from which Formula (2.3)

$$Y(t) \approx Y(0)\, e^{\alpha n t}$$

follows. This formula implies that "in the early stages of an epidemic, the number of infectives grows exponentially." I now quote from [2]:

"The most reliable data on the course of the AIDS epidemic—the total number of cases in the U.S. as a function of time—have been compiled by the Centers for Disease Control. Analysis of these data has revealed several surprising facts.

"First, unlike most epidemics, the number of cases of AIDS has not grown exponentially with time, but rather as the cubic power of time, t^3. Virtually all epidemiological models of infectious diseases, including sexually transmitted diseases, predict exponential growth for the early phases of the epidemics, and most of the epidemics that have been studied appear to have followed this pattern. Second, when the data for the AIDS epidemic are broken down into subgroups by race and sex, the number of cases in each subgroup is seen to have grown at t^3, and the growth in all groups appears to have begun at nearly the same time."

A team of mathematical scientists at the Los Alamos National Laboratory has developed a mathematical model explaining the observed cubic growth of the AIDS epidemic in the homosexual population ([2], [3], [4], [5]). "The Los Alamos model has been very accurate, with predictions that have matched within 2–3%" [6, p. 983].

Why does AIDS, unlike most epidemics, not exhibit exponential growth? Why is Equation (2.1) not applicable to the AIDS epidemic? The main reason seems to be the social factors that play a large role in the spread of HIV. The behaviors that put people at risk of exposure to HIV are not randomly distributed, but tend to be confined to certain segments of the population.

It is reported in [6] that the Los Alamos group is involved in a project to develop user-friendly software that will allow public health workers to use the model to better understand the future of the epidemic. For this project,

researchers at the University of Illinois are working on the software, the Census Bureau is providing data, and the Air Force Academy will be testing the package.

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(AUTHOR'S REVIEW OF DISCUSSION)

HARRY H. PANJER:

I thank Professor Chan, Mr. Cowell and Professor Shiu for their thoughtful discussions. This paper was prepared originally for the symposium "Insurance and the AIDS Epidemic" held in May 1988 [1]. The purpose of the paper was to provide an overview of epidemiological considerations in modeling the growth of the epidemic as well as to test the appropriateness of the epidemiological models by fitting them to the observed data.

Professor Chan and Professor Shiu point out the poor fit exhibited by assuming exponential growth in the early part of the epidemic. As a consequence of the poor fit of the exponential growth model in this paper, I prepared a subsequent paper [2] in which the growth rate is modeled as a power function, referred to as a polynomial growth model. In that paper the polynomial growth model exhibited a dramatically better fit than the exponential growth model. This poor fit has been pointed out by other authors, some of which are referenced by Professor Shiu. In [2], it is shown that for Canada, U.S.A. and Australia quadratic or cubic growth models for the

epidemic fit significantly better than exponential growth models. This observation is consistent with the observations made by the authors referenced by Professor Shiu.

Mr. Cowell points out the differences between his earlier paper, the use of the discrete version of the back calculation technique, as opposed to the continuous version used in this paper. The continuous version in this paper can be approximated by a discrete version with short time intervals. One further purpose of this paper was to point out the sensitivity of the results to the various assumptions involved. This is evidenced by the wide differences in the estimates of the number of infections resulting from relatively small changes in the incubation model. Consequently, minor differences in the method of calculation resulting from the difference in calculation used in Mr. Cowell's paper and this paper are insignificant relative to the differences resulting from small variations in assumptions.

It might be of interest to the reader that the methodologies used in this paper were also used in the Canadian Institute of Actuaries reports on AIDS [3, 4] with a Weibull incubation model. This incubation model was chosen because most biomedical researchers have selected it as an appropirate model. However, a significant difference between the Weibull model and the models of this paper are in the right-hand tail of the distribution where we have very little information.

I am indebted to all three reviewers for their constructive comments on this survey paper.

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