A B S T R A C T

This paper is an introduction to the mathematical epidemiology of sexually transmitted diseases (STDs) and its application to public health. After a brief introduction to transmission dynamics models, the construction of a deterministic compartmental mathematical model of HIV transmission in a population is described. As a background to STD transmission dynamics, basic reproductive rate, intergroup mixing, rate of partner change, and duration of infectivity are discussed. Use of the models illustrates the effect of sexual mixing (proportionate to highly assortative), of preventive intervention campaigns, and of HIV-chlamydia interaction on HIV prevalence in the different population groups. In particular, planned prevention campaigns can benefit the targeted intervention group but surprisingly can be disadvantageous for the general population. Through examples, mathematical models are shown to be helpful in our understanding of disease transmission, in interpretation of observed trends, in planning of prevention strategies, and in guiding data collection.

A B R É G É

Cet article est une introduction à l'épidémiologie mathématique des maladies transmises sexuellement (MTS) et de ses applications à la santé publique. Les modèles de dynamique de transmission des MTS sont d'abord introduits de façon concise. À la section méthode, l'élaboration d'un modèle déterministe compartimental de transmission du VIH dans une population est illustrée. Certains concepts de base des modèles dynamiques tels le taux de reproduction de base, l'interaction intra groupes, le taux de changement de partenaires sexuels et la durée d'infectiosité sont discutés. L'impact des interactions sexuelles entre individus, des mesures préventives visant à modifier les comportements sexuels et de l'interaction entre les MTS classiques et le VIH est illustré. Plus particulièrement, il est démontré que les campagnes de prévention peuvent bénéficier au groupe directement ciblé par la campagne au détriment de la population générale. En résumé, cet article illustre, à l'aide d'exemples, l'utilité des modèles mathématiques permettant d'améliorer notre compréhension de la dynamique de transmission des MTS, notre interprétation des tendances temporelles observées et notre évaluation des stratégies de prévention; ceci permettant également de mieux cibler les données les plus utiles à recueillir lors d'études futures.

Mathematical Models of Disease Transmission: A Precious Tool for the Study of Sexually Transmitted Diseases

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Mathematical models of disease transmission that are used to study the within or between host dynamics of microparasite or macroparasite infections[1] have a long history and numerous applications in different areas of natural sciences, including epidemiology, ecology, evolution, public health, and genetics.[1-6] Within and between host dynamics describe the course of infection in a population of cells within an individual and in a population of individuals within a community respectively. This paper focuses on models of between host dynamics.

Dynamic models are an analytical tool used primarily to gain a better understanding of the evolution and dynamics of the infection with time, space or age. Such models can help identify the major epidemiological forces acting on infections, draw attention to important gaps in key parameter values, act as a guide for future epidemiological studies and permit the formulation and validation of new hypotheses. The approach can also strengthen arguments in favour of or against public health programs for control and prevention, can identify the most cost-effective programs, and can anticipate inadvertent effects of disease control.[1-7]

The general aim of this paper is to introduce readers to mathematical models of disease transmission and control, and to illustrate how they can improve our understanding of sexually transmitted diseases (STDs) and elucidate counter-intuitive results of disease propagation.

The paper is divided into three sections. The Methods section includes a short description of the structure of mathematical models of STD transmission. In the Results section some important findings derived from mathematical models are given as well as a few examples of counter-intuitive results pertinent to public health for the prevention of classical STDs or HIV/AIDS. Finally, a discussion of the benefits of such models, their use in public health, the theoretical study of STDs, and further research areas is presented.

METHODS

Transmission dynamics models incorporate in their framework the appropriate epidemiological, biological and demographic characteristics of the disease and the population. The structure of such models is typically shown as compartments representing the flow of individuals from one disease state to another.

Figure 1 represents the compartmental structure of a very simple model of HIV transmission in a homogeneous population. Everybody (males and females) has the same behavioural and demographic characteristics. The model represents the passage of individuals to and from various stages of HIV infection. The three compartments represent the three disease states: susceptible, HIV infected, and full-blown AIDS. The symbols $X(t)$, $Y(t)$, and $A(t)$ represent at time t the number of individuals in each disease state ($N(t) = X(t) + Y(t) + A(t)$). The transition from the susceptible to infected states occurs at a rate $\lambda(t)$, also called the force of infection (similar to the incidence rate in person years), at which

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new HIV infections occur. It is a function of time given by

$$\lambda(t)= m\,\beta\, Y(t)/N(t) \qquad (0.1)$$

where m represents the mean rate of partner change per unit time, β the HIV transmission probability per partnership, and $Y(t)/N(t)$ the prevalence of HIV infection in the population at time t, which also represents the risk of exposure to the virus.[8,9] Individuals leave the infectious state at a rate γ ($1/\gamma$ = HIV incubation period duration) as full-blown AIDS develops.This compartmental model can be analysed by at least two types of models, deterministic or stochastic, which are based respectively on a set of differential or stochastic equations that can be solved analytically in simple cases, or numerically or by Monte-Carlo simulations in more complex ones.

With deterministic models, the course of infection, determined by the initial conditions (population size, number of infected, etc.) will always take the same course.[10] Thus, they are used when the spread of infection is relatively insensitive to chance fluctuations and can therefore be approximated by average values.[11] Stochastic models are generally used when chance fluctuations or predefined heterogeneities are important in the system (e.g. small sub-populations, small attack rates).[10] Each event, represented by an arrow in Figure 1, has a given probability of occurrence in a given time interval.[12-13] For fixed initial conditions, the course of infection is not always the same. These models are useful because they provide a natural framework for incorporating methods of statistical inference into the analysis.[6,14] They are therefore suitable for situations in which variances and covariances are as important as average behaviour in the interpretation of results.[11]

The resolution of the set of mathematical equations from either deterministic or stochastic models enables one to follow, through the relevant quantities of interest (e.g. prevalence, incidence), the course of infection with time. For simplicity, our discussion will be restricted to deterministic models.

The model in Figure 1 can be made more realistic by adding relevant compartments and heterogeneities.[15-20] For example, biological heterogeneity can be added by dividing the HIV incubation period into three compartments corresponding to the three infectious stages of HIV.[15-21] Correspondingly, the average time spent and the probability of HIV transmission in each stage would have to be defined. Spatial heterogeneity can be incorporated to reflect differences in the population characteristics between regions. Assuming that individuals have different levels of sexual activity, then variability in sexual behaviour can be introduced. The refinements and the degree of complexity introduced into the model depend on the objectives of the study.

Different types of analyses can be performed on such systems. With simple models it is usually possible to study the system analytically, that is, to study its stability at equilibrium, to derive explicit solutions and threshold conditions for the establishment and persistence of the infection in the population.[2,22-24] Equilibrium solutions correspond to the level of infection (prevalence or net numbers) as t tends to infinity, when the rate of infection remains constant with time. Thus, despite their simplicity, basic models prove to be very useful.[1,5,25] When the model is more complex, numerical simulations are required to solve the system's equations and to reproduce the course of infection with time. By varying the parameter values one at a time in the simulations, as in a controlled experiment, the impact of one parameter on the course of the infection can be assessed. When there are uncertainties in parameter estimates, a sensitivity analysis (parameter values are varied over a reasonable range) can be performed to assess the sensitivity of the results to parameter assumptions and to highlight the most important parameters to estimate. These models are mostly used to gain a better understanding of the dynamics of

TABLE I
Examples of Median Based Estimates of the HIV Incubation Period

Study Population	Median Incubation Period (yr)
Homosexual men in Vancouver[26]	9.5*
Homo/bisexual men in Amsterdam[27]	7.3 to 9.2
Homo/bisexual men in Canada[28]	7.6
Homosexual men in multi-centre study	9.3
From prevalence and diagnosis patterns[30]	9.8
Varied risk factors in San Francisco[31]	8.3
Homo/bisexual men in San Francisco[32]	7.8
Female prostitutes in Nairobi, Kenya[33]	3.5

* Mean incubation period calculated from a Markov process

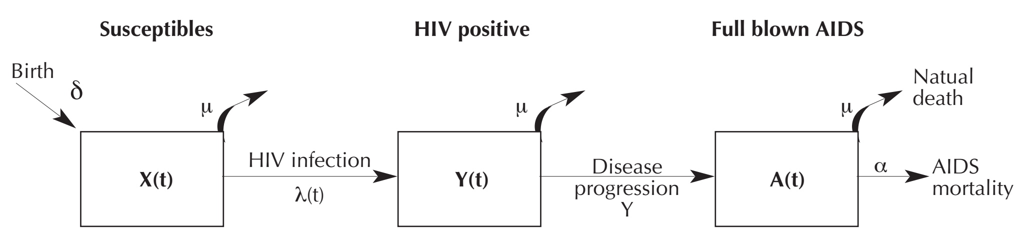

Figure 1. Compartmental model representing passage of individuals to and from various stages of HIV infection (μ = natural mortality rate, α = AIDS associated mortality rate [$1/\alpha$ = life expectancy of an AIDS patient], and δ = birth rate).

the infection in the population. They are seldom used to make medium or long term projections. Instead they are used to identify the conditions that favour establishment and persistence of infection, to approximate the time scale of the epidemic and its maximum size reached under certain conditions or to study how prevention strategies or vaccination campaigns influence the course of the disease. The parameters of the models are usually obtained from field data (see Table I). Once a range of acceptable parameter values is chosen, the predictions and assumptions of the model can, when appropriate, be validated by comparison with the observed data. However, the lack of observed data with time due to the relative apparent novelty of an infection like HIV can render validation of models difficult and, in conjunction with parameter uncertainties, can make long term predictions hazardous.

RESULTS

The successful pathogen invasion into a host community involves three major phases: initial establishment, persistence within the invaded host population, and spread to other host subpopulations. The initial establishment of an infection in a population is determined by the basic reproductive rate, R_o, of a pathogen, which measures the average number of secondary infections generated by a single index case in a fully susceptible host population.[1,3,5,25,34-37] The value of R_o is crucial for determining the reproductive success of the infection. To invade and establish within a population an R_o of greater than 1 is required. However, establishment does not imply that the pathogen will be able to persist in the host population in the long term. If the infection persists at endemic equilibrium, the reproductive rate of the parasite, R ($R = R_o x$, where x is the fraction of susceptibles at equilibrium), must equal 1.[36] After establishment and persistence, the spread of an infection to other communities depends on a variety of demographic and social factors such as spatial distribution and migration.[37] For STDs,[1,3,8,9,35-37] R_o is given by

$$R_o = m\beta D \qquad (0.2)$$

and unlike directly transmitted infections[34,36,37] is independent of host density. D represents the average duration of infection, and m and β are defined as in (0.1). D and β capture the biological components of the STD while m reflects the behavioural component of the population.[7] Therefore, for an STD to be able to establish itself and persist in a population, the sexual activity should be above a critical threshold, namely $m \geq 1/\beta D$.

This expression of R_o is valid for any STD in a homogeneous population. Everybody has the same demographic, biological and behavioural characteristics. However, in reality, sexual behaviour is greatly variable between individuals (see Figure 2).[38-42] In the presence of heterogeneity in sexual activity R_o becomes

$$R_o = c\beta D \qquad (0.3)$$

where c represents the effective mean rate of sexual partner change per unit time[1,8,9] and

$$c = m + \sigma^2/m \qquad (0.4)$$

where σ^2 is the variance in sexual activity. This is valid under the assumption of proportionate mixing in a population. Individuals randomly choose their sexual partners without any preference in partner selection. Their choice depends only on the quantity of a certain type of partner in the population and the number of partners that person has. Heterogeneity in sexual activity favours the establishment and persistence of STDs, as do other types of heterogeneities.[1,37,43,44] The relationship in (0.4) reflects the disproportionate role played by the minority of very sexually active individuals, who are more likely to both acquire and transmit infection by virtue of their sexual activity. These individuals who contribute to the increase of σ^2 and therefore of R_o are sometimes referred to as "core group transmitters" (CGT).[25,45] The importance of the CGT has been recognised with the use of mathematical models.[25] The exact definition of the CGT is more restrictive than that commonly used in practice, when "core groups" often refers to individuals at higher risk of infection.[46] Explicitly, it implies that the size of

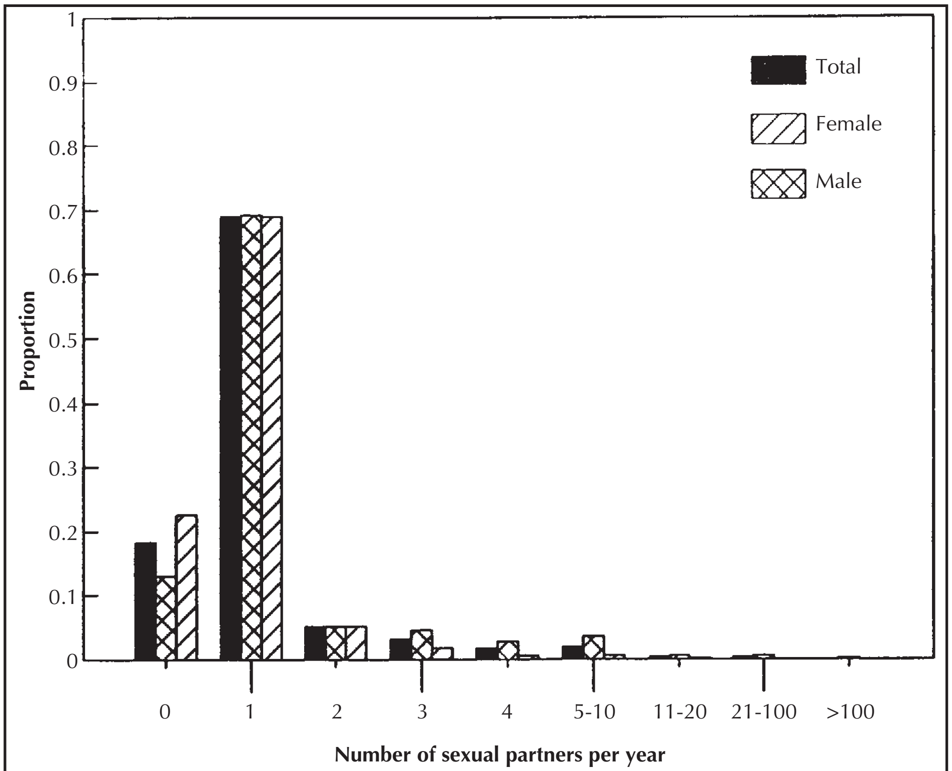

Figure 2. Distribution of sexual activity in a random sample of the general population of the U.S.[39]

the core group will vary for different STDs, since the threshold level of sexual activity (i.e., $c \geq 1/\beta D$) necessary to maintain R_o at greater than 1 varies among STDs.[7,9,36,45] This is because D and β differ between STDs and even between countries. In developed countries the average duration of infection with gonorrhoea is estimated at two months compared with six months in developing countries.[7,45] Thus, in developed countries, the effective mean annual rate of partner change (*c*) must be ≥ 13 (D=1/6 years, β = 0.5) in order for gonorrhoea to become established, compared with ≥ 4 (D=1/2 years, β=0.5) in developing countries[7,45] (see Table II). Some thresholds in Table II might appear high, but they are mainly due to the core group contribution. Because core groups play an important role in the persistence of infection, they provide a sensible target for control interventions through diagnosis and treatment.[7,25]

Heterogeneity influences not only R_o but it largely determines the prevalence of infection within a community. Surprisingly, perhaps, greater heterogeneity in sexual activity, while favouring establishment, does not necessarily imply that the prevalence of infection will be higher in the total population.[9,47] Paradoxically, greater heterogeneity will result in a smaller proportion of infected individuals for a given value of *c* than would the situation with a smaller σ^2 but the same *c*.

The establishment and rate of spread of infection also depend greatly on who is forming a sexual partnership with whom (i.e., the degree of mixing between the different sexual risk groups in the population).[48-56] In simple terms, who has sex with whom is an important determinant of incidence and prevalence of infection in the general population and in the different risk groups. Preference in partner acquisition defines a network of sexual contacts between individuals within different sexual activity classes. Conceptually, mixing between sexual activity classes can be seen as a continuous spectrum from assortative, in which individuals choose sexual partners only from their sexual activity level, to proportionate, in which there is a lack of preference (partnerships are formed according to the number of relationships available at any time), to disassortative, in which there is a choice of partners only from other sexual activity classes (see Figure 3).[55]

The impact of the mixing pattern on the spread of STDs is complex and not obvious. Figures 4a and 4b show its predicted impact on HIV prevalence in the general population with time, for a population with a high and low mean rate of partner change respectively.[56,57] In Figure 4a, extremely assortative mixing really slows the spread of infection by limiting its introduction from the high activity classes (first peak) to the low activity classes (second peak). In Figure 4b, the prevalence attained with assortative mixing is higher than that with proportionate mixing, from which HIV cannot even become established. In that case, by restricting contact within members of the core group, assortative mixing favours the establishment of the infection. This occurs when the average level of sexual activity is below a cer-

TABLE II
Estimates of Key Epidemiological Parameters Necessary to Sustain Transmission of Five Different STDs (c = 1βD)

Agent	Duration of Infectious Period (D) in years	Transmission Probability per Partnerships (β)	Effective Mean Rate of Partner Change (c) per year
Neisseria gonorrhoeae			
No control	0.5	0.5	4
Control	0.15	0.5	13
Chlamydia trachomatis	1.25	0.2	4
Treponema pallidum			
No control	0.5	0.3	7
Control	0.25	0.3	13
HIV			
African parameters	2	0.1	5
American parameters	8	0.01	13
Haemophilus ducreyi	0.08	0.8	15

Table was produced from data colllected by Brunham & Plummer (1990).[45]

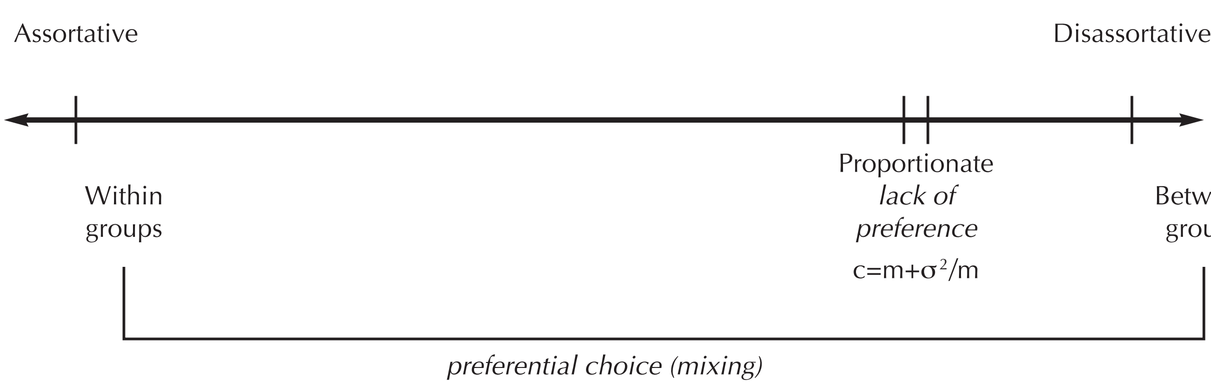

Figure 3. Schematic representation of sexual mixing.

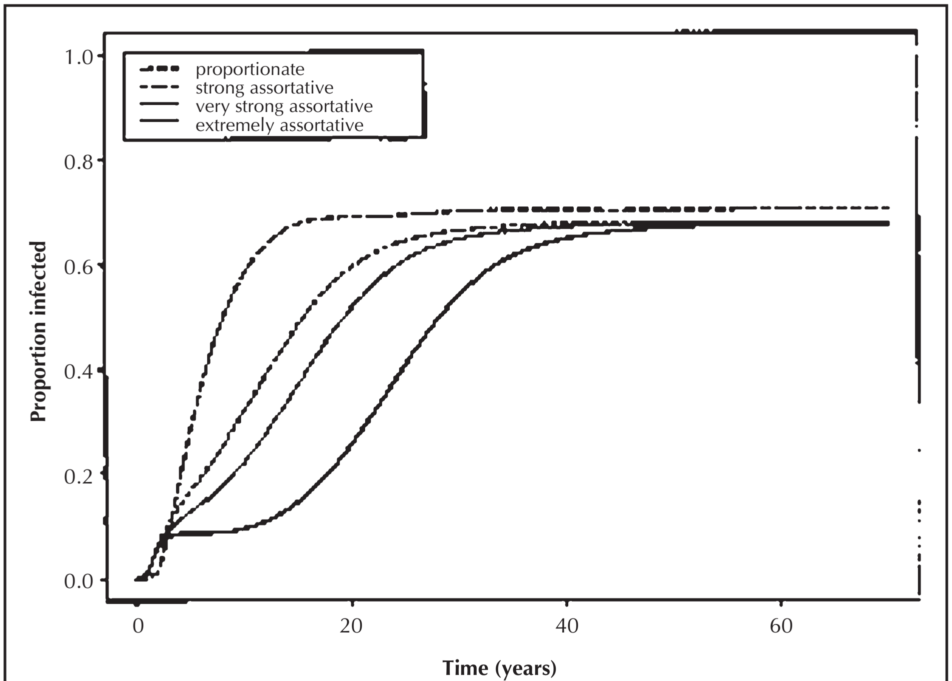

Figure 4a. Predicted prevalence of HIV in the general population according to degree of mixing in a population with two sexual activity classes (high and low) and a high overall mean rate of partner change (*m* = 8 new partners per year).[56]

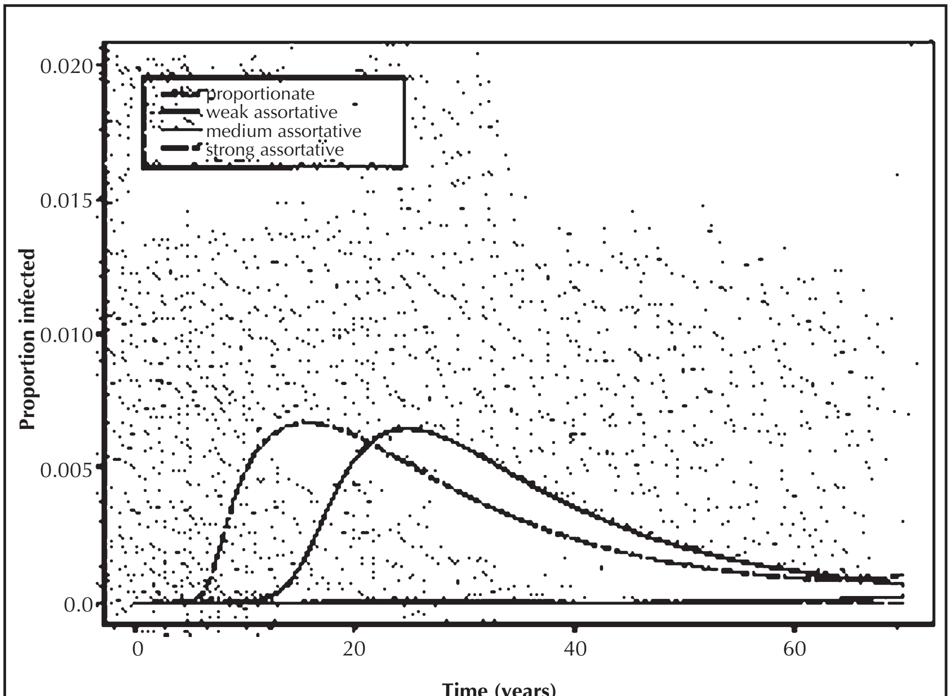

Figure 4b. Predicted prevalence of HIV in the general population according to degree of mixing in a population with four sexual activity classes (from low to high) and a low overall mean rate of partner change (*m* = 0.48 new partners per year).[57]

tain threshold.[46] Under a low average level of sexual activity, assortative mixing favours the establishment of the STD because contacts between infected and susceptible individuals are concentrated within the high risk population. This results in more effective transmissions than under proportionate mixing, in which a larger proportion of contacts occur with non-core transmitters, who are unable to sustain the infection.[56-58] Thus, with a low average level of sexual activity, a certain degree of assortativeness is necessary for an STD to become established. For mixing types other than proportionate, the expression of R_o is more complex.[3,9,46]

Interestingly, an increase in the average level of sexual activity of the general population achieved through an increase in the rate of partner change of the lowest activity classes[57,59] does not always result in an increase in STD transmission. This is because, when the non-core population has a very low level of sexual activity, very few partnerships are permitted between the core and non-core population, forcing members of the core group to form partnerships among themselves; this results in an assortative mixing in which most transmissions are concentrated in the core group, ensuring persistence of the infection.

Targeted interventions aimed at reducing risky sexual practices can be very hazardous because such interventions are likely to lead to changes in the mixing pattern,[60-63] the consequences of which are far from trivial. Figures 5a-g illustrate the predicted results of a hypothetical campaign targeted at male clients of prostitutes at time 20. The initial mixing pattern is proportionate. In Figure 5a,[62] male clients reduce their rate of partner change by 20%. In order to minimize their chance of getting infected, they also choose a partner from the non-core population. Thus, the mixing pattern becomes more disassortative. Consequently, the prostitute sexual activity drops significantly (Figure 5b). Despite these changes in sexual activity, the prevalence of infection in the high risk groups is only slightly decreased, by 3% (Figure 5c), because before prevention the population was already saturated with HIV. The impact of the campaign from

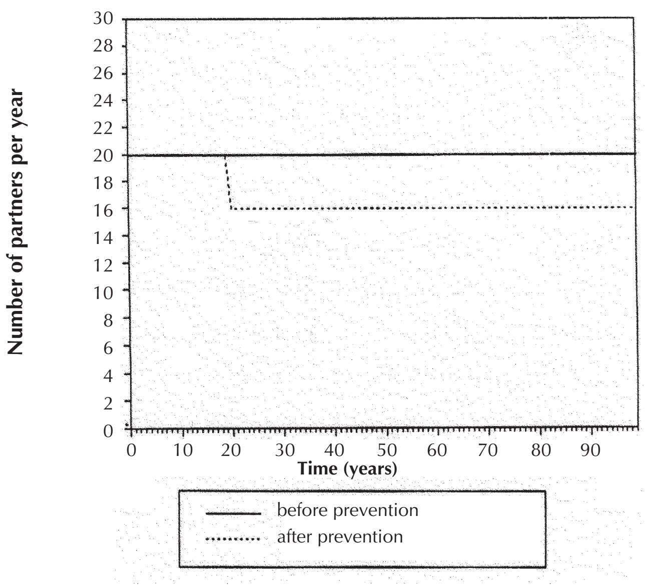

Figure 5a. Predicted rate of partner change among males of high sexual activity before and after a prevention campaign.

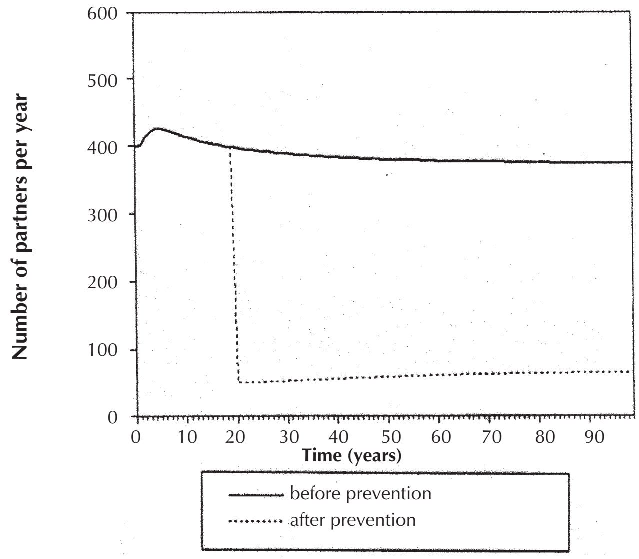

Figure 5b. Predicted rate of partner change among females of high sexual activity before and after a prevention campaign.

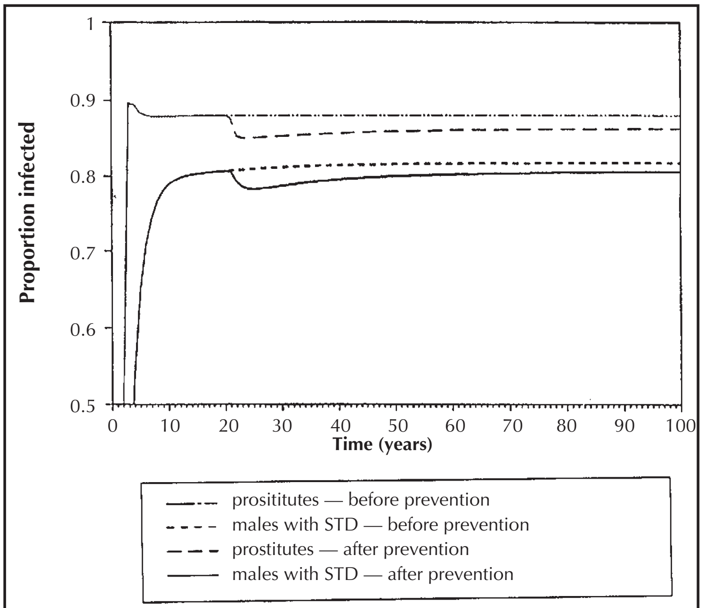

Figure 5c. Predicted HIV seroprevalence changes in high activity males and females before and after a prevention campaign.

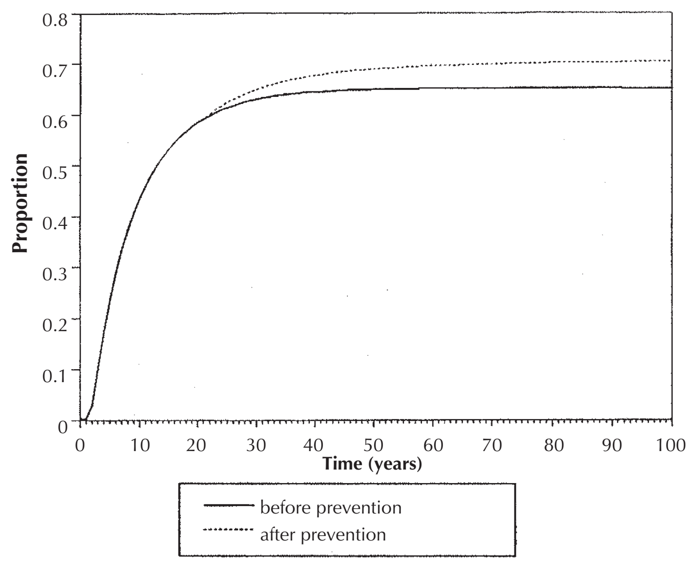

Figure 5d. Predicted HIV seroprevalence changes in total male and female population.

the point of view of changes both in sexual activity and in seroprevalence levels in the targeted group would erroneously suggest to public health officials that the prevention program was successful. However, the impact on the general population is different: the targeted prevention results in a slight increase in seroprevalence in the general population (Figure 5d). Even if the male clients of prostitutes reduce their sexual activity, they increase their demand for low risk female partners, who in turn increase their activity from four to six partners per year (Figure 5e). The latter case assumes that these partners do not feel concerned about the HIV threat and are relatively liberal in their sexual behaviour. The level of sexual activity of the male from the low activity class remains unchanged (Figure 5f) whereas that of the general population eventually increases slightly (Figure 5g). Therefore, the prevention program has induced a change in the mixing pattern, resulting in a negative effect on the general population.

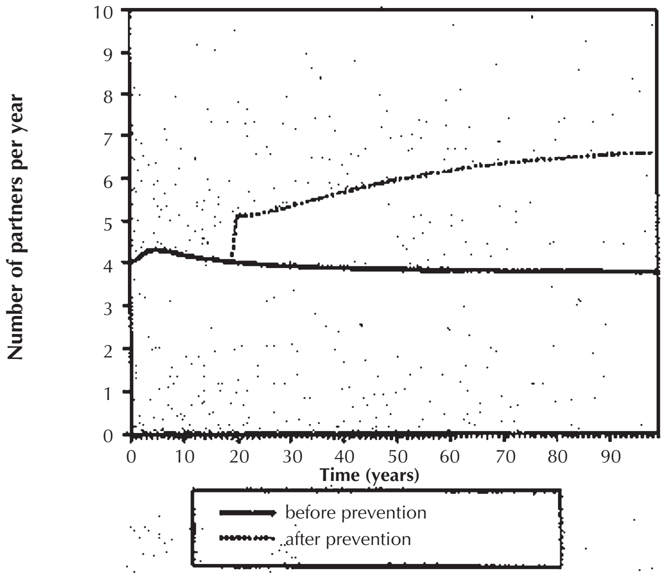

Figure 5e. Predicted rate of partner change among females of low sexual activity before and after a prevention campaign.

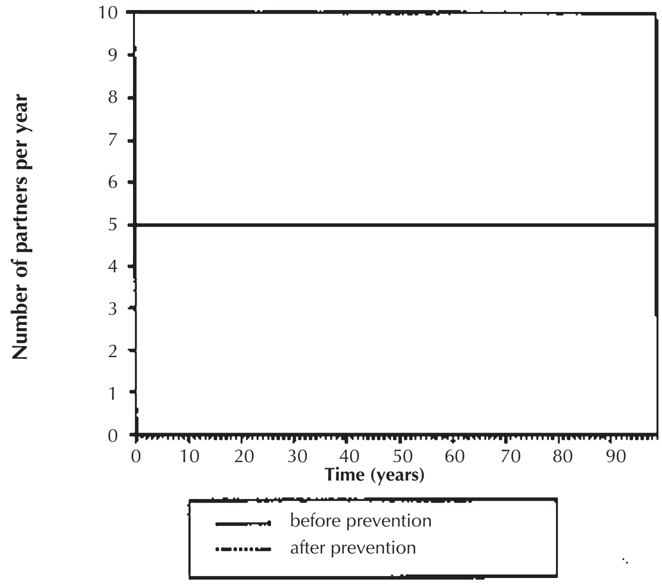

Figure 5f. Predicted rate of partner change among males of low sexual activity before and after a prevention campaign.

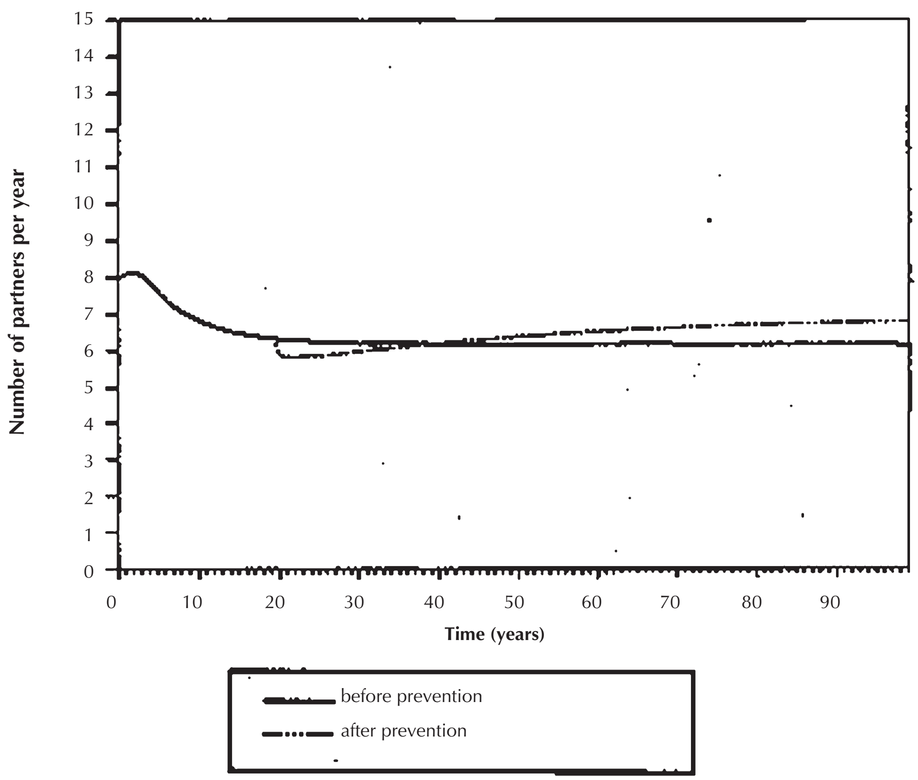

Figure 5g. Predicted overall rate of partner change in the total male and female population.

Another interesting point emerges from models of co-existing STD and HIV infections. Effectively, as HIV spreads in the population, the prevalence of STD decreases over time (Figure 6).[14,64-66] As reported by others,[47,56,64-66] this effect is the result of AIDS-induced mortality taking a greater toll of the most sexually active, who contribute disproportionately to the transmission success of the STD cofactor.[7,14,57] This results in a change in overall behaviour that is induced solely by the mortality of the most sexually active individuals. For this reason, trends in classical STDs can be poorer markers of sexual behaviour than expected when the efficacy of prevention strategies is evaluated.[14,57,69] If a cohort of individuals were followed up for an extended risk period, serial observations of the sexual behaviour of each individual with time would not show any individual trends, but a trend in average behaviour following AIDS-induced mortality in the most sexually active individuals. Blower et al[66] estimate that, after seven years' follow-up, AIDS-induced mortality could be responsible for 21% and 33% of the observed reduction in the mean and the variance respectively of risk behaviour in a cohort of gay men in Amsterdam. Such observations (from both models and field studies) may explain partly, at least, the decrease in gonorrhoea prevalence among homosexuals in different countries in the 1980s,[67,68] contrary to the sole explanation of the adoption of safer sexual practices.[59,65,67-70]

In a situation in which STD enhances HIV transmission,[71-76] the decrease in STD prevalence, coupled with the depletion of the most sexually active population, contributes to limiting the spread of HIV. The consequences on HIV spread would be otherwise more devastating if STD prevalence remained the same throughout the duration of the epidemic. The extent to which differential mortality will modify STD trends in an open population is very difficult to predict. For example, if replenishment of the high risk population occurs rapidly, if high risk behaviour is a very transient process, or if HIV also enhances STD transmission, then STD rates could be maintained or even increased above initial levels for an extended period of time, with a potentially greater effect on HIV transmission than that predicted by the current model. Figure 7 shows the predicted HIV and chlamydia trends in the total female population. The reference lines refer to spread under HIV-enhanced transmission due to the presence of chlamydia (5-fold increase in HIV transmission probability due to chlamydia) and slow replenishment of high risk population at time t = 20. Spread under HIV-chlamydia mutual enhancement or fast replenishment is also shown. For further model and parameter details see Boily, Desai and Garnett.[57]

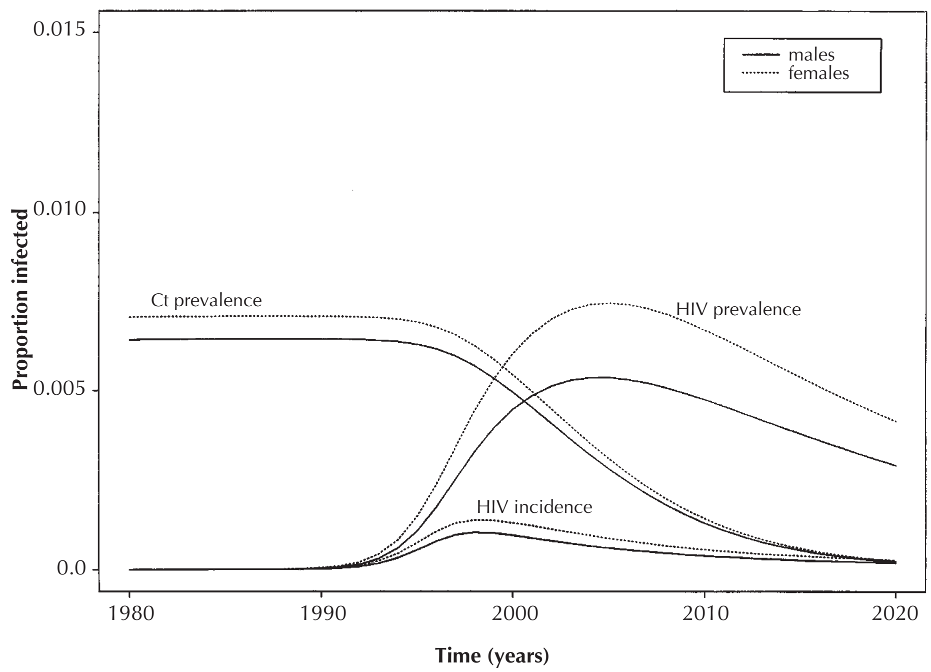

Figure 6. Predicted HIV and Chlamydia (Ct) prevalence in the general population, males and females (see Boily, Desai, and Garnett[57] for model details and parameters).

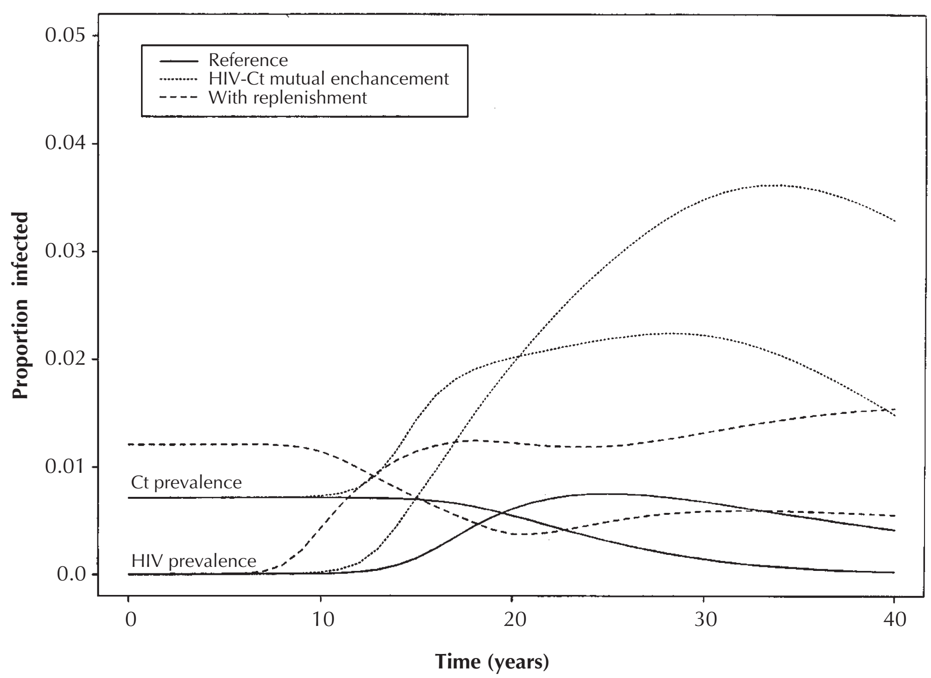

Figure 7. Predicted HIV and Chlamydia (Ct) in the general female population.

DISCUSSION

The objective of this paper was to introduce and describe the use of mathematical modelling to study the transmission dynamics of STDs. Basic theoretical concepts, their applications and implications for public health were introduced. The results and examples presented constitute only a sample of what has been done in the field of mathematical epidemiology, and many more are described elsewhere.[1,3,10,77-81]

The examples demonstrate how mathematical models can identify and explain counter-intuitive results, which in turn can be useful in interpreting observed trends. Such results occur because infectious diseases are very dynamic and nonlinear in nature. The dynamic aspect implies that changes occurring in one stratum of the population can influence transmission and induce unexpected changes in other parts of the population. Thus, reductions in individual risks of HIV infection will not necessarily imply a diminution in population risks.

The system is qualified as being nonlinear because the occurrence of new HIV infections depends on the number of individuals already infected (dependent happening).[82-83] In other words, the incidence of HIV infection depends on its prevalence in the population. A change in the characteristics of the population that influence the incidence of infections will also modify the prevalence; the change in prevalence will then further influence the incidence of infection, and so on, until a new equilibrium is reached.

Because of this dependence between the occurrence of past and present events, prevention strategies against infectious diseases have not only a direct protective effect for the person receiving the intervention but also indirect effects resulting from changes in the intensity of transmission in the population.[82-83] This feature, common to most infectious diseases, is the basic underlying concept of herd immunity, in which a certain fraction of the unvaccinated population is protected from infection just because other individuals have been immunized.[1,5,25,36,57,83] This can also be observed in the etiologic fraction of a certain cofactor of transmission or the preventable fraction following prevention.[57] For example, the etiologic fraction, being time dependent, is greater if obtained over longer time periods.[84] As with the effect of herd immunity, the elimination of the cofactor from the population also protects susceptible individuals never exposed to the cofactor. This is a result of the reduced risk of infection due to the overall decrease in the force (incidence) of infection incurred by reduction of the infection in the total population. The protected fraction, therefore, consists of a direct effect (reduction in infection among those recently protected from the cofactor) and an indirect effect (reduction in infection among those never exposed to the cofactor).

Theoretical analyses have helped identify the importance of different types of heterogeneities in sexual behaviour. Given the important effect of the sexual network and the heterogeneity in sexual activity on the dynamics of spread of STDs, many countries have undertaken studies to estimate the distribution of sexual activity.[38-42,85] However, few data exist on the estimates of the mixing pattern in different communities. Most estimates tend to indicate that the mixing pattern is located in the assortative range of the spectrum.[46,52-55,86] In this paper, the impact of social networks has been discussed only in the context of sexual behaviour. However, the structure of social networks is also important when relating different heterogeneous communities such as intravenous drug users (IDUs), heterosexual, homosexual and bisexual communities. It has been suggested that changes in STD transmission rates in some US communities could be attributed to changes in the social network within the drug users community following the crack epidemic in such a manner that increases in prostitution practices occurred in order to finance drug habits.[61]

Since the different aspects of STDs can be very complex, mathematical models are powerful and indispensable tools. Not only do such studies and their results have theoretical interest, but they have numerous applications in the field too. They can guide future data collection in order to refine our comprehension and improve prevention strategies. Models indicate that data are greatly needed on the mixing pattern, on the number of sexual acts per partnership as a function of the duration of the relationship, on HIV transmisson probability in an STD-free population, and on the STD-HIV association. In the Canadian community, data on the sexual behaviour of the different risk communities including homosexual, heterosexual, bisexual, IDUs and large ethnic communities as well as the general population are definitely required.

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In Memoriam

RONALD ADRIAN DRAPER

1935–1997

Canadians lost a long-time champion of public health this past week with the death of Ron Draper, who passed away peacefully at his cottage on Friday, August 22. He is survived by his wife and best friend, Marion, their children Michael and Lisa, and his sister Ruth Jackson. He will be remembered by all who knew him for his warmth, his humour, and his absolute love of life.

Over his long career, Ron proved that a bureaucrat with a highly developed social conscience and an ability to make complex concepts practical can be in the forefront of progressive social policy development. In 1972, as Director General of the Non-Medical Use of Drugs Directorate, he developed a program to address the findings of the LeDain Commission. He was advisor to the Canadian delegation to the United Nations Commission on Narcotic Drugs, carried out an organizational study for the International Council on Alcohol and Addictions, and chaired a group studying adaptation in programs designed to reduce the demand for drugs.

It is in the field of health promotion, however, that Ron made a particularly distinguished contribution, nationally and internationally. In 1985, Ron chaired a group developing a global health promotion strategy; he was a major organizer of the First International Conference on Health Promotion held in Canada in 1986, which resulted in the Ottawa Charter; and he was a faculty member of the European Summer School on Health Promotion Policy. He can be credited with introducing the visions of health and health promotion so well expressed in Achieving Health for All: A Framework for Health Promotion. In 1987 he became a full-time consultant on health promotion to the World Health Organization.

Ron's life and work have been an inspiration to those working in the health field. In 1988, he was awarded CPHA's highest honour, the R.D. Defries Medal, and an honorary life membership for his outstanding contribution in the field of public health. His openness to new ideas, his commitment to community-based approaches, and his willingness to represent disadvantaged groups made him a worthy recipient.

This past July, Ron, Marion and Michael participated in the presentation of the very first CPHA Ron Draper Health Promotion Award to the North End Community Health Centre Association of Halifax, at the CPHA Annual Conference. The award is given in recognition of an individual, group, or organization that has made a significant contribution to health promotion by working in the community. Memorial donations to CPHA on behalf of the Ron Draper Health Promotion Award fund, c/o CPHA's National Office in Ottawa, would be appreciated by the family.

JEAN GOODWILL

1928–1997

Jean Goodwill, one of Canada's leading Aboriginal health care advocates, died of cancer on Monday, August 25, at the age of 69. Jean, who was born and raised on the Little Pine reserve near North Battleford, Saskatchewan, was awarded the Order of Canada in 1992 in recognition of a career that spanned more than three decades.

She was one of the first native women appointed to the federal civil service as a special advisor for the departments of Indian and Northern Affairs and Health Canada. During her 20-year career with the federal government, Jean was instrumental in developing health and social policies for Aboriginal people.

For the past two years, Jean was Co-chair of the Working Group for CPHA's Aboriginal Program, and Chair of the Aboriginal Youth Committee—exploring ways in which Aboriginal youth could be encouraged to choose careers in the health field. She was also a former member of the CPHA Board of Directors.

Jean was a founding member of the Native Women's Association of Canada, a founding member and Past-President of the Indian and Inuit Nurses of Canada, a Past-President of the Canadian Society for Circumpolar Health, and the author of four books.

Jean Goodwill was dedicated to improving the public health of all Canadians and especially that of Aboriginal people. Her strength, leadership, and personal commitment contributed significantly to the many initiatives in which she was involved. Her wisdom and vision for the future will be greatly missed.