A non-linear model for a sexually transmitted disease with contact tracing

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A non-linear model is developed for an epidemic with contact tracing, and its dynamic is studied. We present the data for the Cuban HIV/AIDS epidemic and fit the non-linear model, we obtain estimates for the size of the Cuban HIV epidemic, and for the mean time for detecting a person that is infected with HIV.

Keywords: HIV/AIDS epidemic; contact tracing; epidemic control; deterministic model.

1. Introduction

The first AIDS case was diagnosed in Cuba in April of 1986. This signalled the start of the AIDS epidemic in the country. Some HIV seropositives had been detected at the end of 1985. Earlier the Cuban Government had started taking preventive measures to try to contain the possible outbreak of the epidemic. Among these measures was a total ban on the import of blood, and blood byproducts. Once the first cases were confirmed, a programme based on the experience with other sexually transmitted diseases was started. This programme had among other measures, the tracing of sexual contacts of known HIV seropositives (HIV+), to prevent the spreading of the virus. When a person is detected as living with HIV, an epidemiological interview is carried out by the Epidemiology Department of his municipality or by his family doctor (partner notification). After this interview the Epidemiology Department tries to locate the sexual partners of the person through the network of the Health System. The person living with HIV usually does not participate in this process, though they normally help in notifying their present partners. Trying to locate the sexual partners is a very complex job and one that in some cases takes a lot of time. This task is one of high level of priority for the Health System, and it is something that is in constant supervision to try to determine how effective it is in the prevention of the spread of HIV. All data used is for the period 1986-2000.

The number of AIDS cases in Cuba is 1284 with 318 females and 966 males. Of the males 79.3% are homo–bisexuals (we consider the group of homo–bisexuals to be formed by homosexuals and bisexuals). There have been 874 deaths due to AIDS. Through the

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Year	HIV+	AIDS	Death due to aids
1986	99	5	2
1987	75	11	4
1988	93	14	6
1989	121	13	5
1990	140	28	23
1991	183	37	17
1992	175	71	32
1993	102	82	59
1994	122	102	62
1995	124	116	80
1996	234	99	92
1997	363	129	99
1998	362	150	98
1999	493	176	122
2000	545	251	142
Total	3231	1284	874

TABLE 1 HIV+ and AIDS cases and deathsdue to aids by year Cuba; 1986–2000

programme a total of 3231 HIV+ individuals have been found, 730 females and 2501 males. Of the males 84.4% are homo–bisexuals. Table 1 gives the new cases detected by year.

As we can see, the epidemic is a small one. With a population of around 11 millions we have a cumulative incidence rate for AIDS of 116.7 per million (7.7 per million per year). One of the characteristics of the Cuban programme for the HIV/AIDS epidemic is that there is an active search of seropositives through the sexual contacts of known HIV-infected persons: 30% of the seropositives have been found through contact tracing. The rest of the infected persons are found through a 'blind' search of blood donors, pregnant women, persons with other sexually transmitted diseases, etc. Non-parametric estimation of the mean time it takes to find a sexual partner notified by a seropositive through contact tracing has been found to be 54.3 months, with a standard deviation of 0.631 (Fig. 1).

Contact tracing has been used as a method to control endemic contagious diseases (Hethcote *et al.*, 1982; Hethcote & Yorke, 1984). While there is still a debate about contact tracing for the HIV infection (April & Thévoz, 1995; Rutherford & Woo, 1988) the resurgence of infectious tuberculosis and outbreaks of drug-resistant tuberculosis secondary to HIV induced inmunodepression is forcing many public health departments to re-examine this policy (Altman, 1997; CDC, 1991). A model of the HIV epidemic allowing for contact tracing would help evaluate the effect of this method of control on the size of the HIV epidemic, and give some idea as to the effectiveness of the Health System in finding them.

Our objective is to model the contact tracing aspect of the HIV detection system, to try to obtain some information that could be useful to the Health System in Cuba in evaluating the way the programme is working. The authors have studied other models with this objective in mind in Lounes & de Arazoza (1999), Arazoza *et al.* (2000). These were essentially linear models. We will now introduce non linearity to model contact tracing.



FIG. 1. Kaplan-Meier for contact tracing.

2. The model

As we noted, the Cuban programme to control the HIV/AIDS epidemic is based in the active search of persons infected with HIV long before they show any signs of AIDS. Our objective is not to model how new infections by HIV are generated, but how the HIV-infected persons are detected. We will consider the following variables:

- (1) X(t), the number of HIV infected persons that do not know they are infected at time t,
- (2) Y(t), the number of HIV infected persons that know they are infected at time t,
- (3) Z(t), the number of persons with AIDS at time t,

with the following constant coefficients:

- (1) N, sexually active population,
- (2) α , the rate of recruitment of new HIV infected persons, infected by X,
- (3) α' , the rate of recruitment of new HIV infected persons, infected by *Y*,
- (4) k_1 , the rate at which the unknown HIV infected persons are detected by the system, independently of other seropositives ('random' search),
- (5) k_2 , the rate at which unknown HIV infected persons are detected by the system, through contact tracing,
- (6) β , the rate at which the HIV positives develop AIDS,
- (7) μ , the mortality rate of the sexually active population,
- (8) μ' , the mortality rate of the population with AIDS.

The dynamics are described by the following system:

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \alpha N X + \alpha' N Y - (k_1 + \mu + \beta) X - k_2 X Y,$$

$$\frac{\mathrm{d}Y}{\mathrm{d}t} = k_1 X - (\mu + \beta) Y + k_2 X Y,$$

$$\frac{\mathrm{d}Z}{\mathrm{d}t} = \beta X + \beta Y - \mu' Z,$$
(1)

and we consider the system only in the region $\mathcal{D} = \{X \ge 0, Y \ge 0, Z \ge 0\}$. It is clear that \mathcal{D} is positively invariant under the flow induced by (1). We make four comments about (1):

(1) In (1) there are two ways individuals go from 'unknown HIV infected' (X) to

- (i) in (i) infected two ways individuals go from unknown HiV infected (X) to 'known HIV infected' (Y). One is through the non linear term k_2XY : this is the part we consider to model contact tracing; the individual is found through his contacts with persons that are known to live with HIV. The other way they can be detected is through the term k_1X and this term models all the other 'random' ways of searching for seropositives. It is important to note that $1/k_1$ can be viewed as the mean time from infection to detection for the persons found not through contact tracing.
- (2) The term $k_2 X Y$ models contact tracing. The way it is taken indicates that the process is one that goes on for a long time and this is the case—the mean time to find a contact is 54.3 months (Fig. 1). If we consider that the mean time from detection to AIDS is 86.8 months (Fig. 2) we can see that, in the mean, contacts are found for more than half the time that a person is living with HIV, before developing AIDS. To consider one or more classes in the model, one class where contacts are found and another (before AIDS) where contacts are no longer found, would complicate the model, and it is not clear that it would give more information on the dynamics of the epidemic. Of course variations are high: some persons have very few contacts and are easy to locate, others have a large number and some are impossible to locate. Some persons have a lot of 'casual' contacts and they do not remember enough information on these contacts to make it possible to find them. Others have less contacts but with a better knowledge of their full name and some times addresses that make it possible for the Health System to find them. Some contacts, even if they are found, refuse to be tested for HIV. In general, of more than 15 000 contacts, 80% have been found and tested. In general we try, as a first approximation, to find out what is the value of k_2 , and what is the general effect of it in the time a person living with HIV is detected. The term k_2XY must be taken as an approximation of a more general term $k_2(X, Y)$ that could be studied in the future.
- (3) We assume that the known HIV infected persons are infectious, but at a much lower rate than those that do not know they are infected. In this case α' will be taken as a fraction of α.
- (4) The passage to AIDS is modelled in a linear way. This could be modelled in a more general way, but for the Cuban case the best fit to an incubation curve is still an exponential. This can be seen in Fig. 3 which gives us the cumulative hazard function for the time to AIDS as a straight line. This corresponds to an exponential model.



FIG. 2. Kaplan–Meier for incubation from detection.



FIG. 3. Cumulative hazard function for incubation period.

The system has two equilibria one at $P_0 = (0, 0, 0)$ which is the no-epidemic case, and $P^* = (X^*, Y^*, Z^*)$ the endemic equilibrium, where

$$X^* = \frac{\sigma \ \gamma + \alpha' N k_1}{k_2(\sigma + k_1)}, \quad Y^* = \frac{\sigma \ \gamma + \alpha' N k_1}{k_2(\gamma - \alpha' N)}, \quad Z^* = \frac{\beta}{\mu'} (X^* + Y^*), \tag{2}$$

with $\sigma = \alpha N - k_1 - \gamma$, $\gamma = \beta + \mu$.

The endemic equilibrium is feasible (i.e. $P^* \in D$) if and only if

$$(\sigma \gamma + \alpha' N k_1)(\sigma + k_1) > 0 \tag{3}$$

$$(\sigma \gamma + \alpha' N k_1)(\gamma - \alpha' N) > 0. \tag{4}$$

The Jacobian matrix of the linear approximation of the system in a neighbourhood of an equilibrium point P = (X, Y, Z) is given by

$$J(X, Y, Z) = \begin{pmatrix} \sigma - k_2 Y & \alpha' N - k_2 X & 0 \\ k_1 + k_2 Y & -\gamma + k_2 X & 0 \\ \beta & \beta & -\mu' \end{pmatrix}.$$

There is one eigenvalue $\lambda_3 = -\mu'$, that is always strictly negative, that is associated with the variable Z. We conclude that to study the stability of the equilibria of system (1) we can restrict ourselves to the study of the equilibria of the system

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \alpha N X + \alpha' N Y - (k_1 + \mu + \beta) X - k_2 X Y,$$

$$\frac{\mathrm{d}Y}{\mathrm{d}t} = k_1 X - (\mu + \beta) Y + k_2 X Y,$$
(5)

in the region $\mathcal{D}' = \{(X, Y) \mid X \ge 0, Y \ge 0\} \subset \mathcal{D} \cdot \mathcal{D}'$ is positively invariant under the flow induced by (5).

We denote J_1 the jacobian matrix for the system (5) that is formed by the first two rows and columns of the matrix J(X, Y, Z). We also denote $Q_0 = (0, 0)$ and $Q^* = (X^*, Y^*)$.

3. Local stability of Q_0 and Q^*

3.1 Local stability for the point Q_0

The jacobian matrix at the point Q_0 is given by

$$J_1(Q_0) = \begin{pmatrix} \sigma & \alpha' N \\ k_1 & -\gamma \end{pmatrix}.$$

 Q_0 , and therefore P_0 , is locally asymptotically stable (l.a.s. for short) if and only if the trace of J_1 is strictly negative and its determinant strictly positive, i.e.

$$Q_0$$
 l.a.s. $\iff \sigma - \gamma < 0$ and $-(\sigma \gamma + k_1 \alpha' N) > 0$.

3.2 Local stability for the point Q^*

 Q^* , and therefore P^* , is l.a.s. if and only if the trace of $J_1(Q^*)$ is strictly negative and its determinant strictly positive, i.e.

$$Q^*$$
 l.a.s. $\iff \sigma - \gamma + k_2(X^* - Y^*) < 0$ and $\sigma \gamma + k_1 \alpha' N > 0.$ (6)

REMARK: Q_0 l.a.s. implies that either Q^* does not exist in our domain, and this is the case if $(\gamma - \alpha' N)(\sigma + k_1) < 0$ or Q^* exists, and this is the case if $\gamma - \alpha' N < 0$ and $\sigma + k_1 < 0$, but Q^* is unstable.

Let us suppose that $\sigma \gamma + k_1 \alpha' N > 0$, then Q_0 is unstable and from (3) and (4), Q^* exists if and only if $\gamma - \alpha' N > 0$ and $\sigma + k_1 > 0$.

4. Global stability of Q_0 and Q^*

Let $g : (X, Y) \mapsto \frac{1}{XY}$, then $\frac{\partial}{\partial X} \{g(X, Y)X'\} + \frac{\partial}{\partial Y} \{g(X, Y)Y'\}$ keeps the same sign in \mathcal{D}' , and using the Dulac criteria we conclude that there are no periodic orbits in the set.

4.1 Global stability of Q_0

 Q_0 is l.a.s. if and only if $\sigma < \gamma$ and $\sigma \gamma + k_1 \alpha' N < 0$. We know then that if Q^* exists, Q^* is unstable.

Let *V* be the function defined on \mathcal{D}' by

$$\forall (X, Y) \in \mathcal{D}', \qquad V(X, Y) = \gamma X + \alpha' N Y.$$

Then V is a Lyapunov function for the point Q_0 on \mathcal{D}' :

$$\forall (X, Y) \in \mathcal{D}', \qquad V'(X, Y) = X(\sigma \gamma + \alpha' N k_1 + k_2(\alpha' N - \gamma) Y).$$

Let $\mathcal{N} = \{(X, Y) \in \mathcal{D}' \mid V'(X, Y) = 0\}, \mathcal{N}_1 = \{(0, Y), Y \ge 0\}$ and $\mathcal{N}_2 = \{(X, Y^*), X \ge 0\}$, when Y^* exists, for the last set.

LEMMA 1 (1) If $\alpha' N - \gamma < 0$, then $\mathcal{N} = \mathcal{N}_1$ and \mathcal{D}' contains only Q_0 , (2) If $\alpha' N - \gamma > 0$, then $\mathcal{N} = \mathcal{N}_1 \cup \mathcal{N}_2$ and \mathcal{D}' contains Q_0 and Q^* .

Proof. Look in V'.

LEMMA 2 If $\alpha' N - \gamma < 0$, then Q_0 is globally asymptotically stable (g.a.s. for short) in \mathcal{D}' .

Proof. In Lemma 1, if $\alpha'N - \gamma < 0$, then $V'(X, Y) \leq 0$ and $V'(X, Y) = 0 \iff (X, Y) \in \mathcal{N}_1$. Along the axis $\{X = 0\}, X' \geq 0$, we conclude that the largest invariant subset in \mathcal{N} is the singleton $\{Q_0\}$ and from LaSalle's invariant principle we conclude that Q_0 is g.a.s. in \mathcal{D}' .

LEMMA 3 If $\alpha' N - \gamma > 0$ then $\sigma + k_1 < 0$ and Q^* exists.

Proof. Suppose $\sigma + k_1 > 0$, then we have $-k_1 < \sigma < 0 < \gamma < \alpha' N$.

$$\sigma \gamma + \alpha' N k_1 < 0 \Longleftrightarrow \sigma < \frac{\alpha' N}{\gamma} (-k_1) < 0 \Rightarrow -k_1 < \sigma$$
$$< \frac{\alpha' N}{\gamma} (-k_1) \Rightarrow k_1 \left(1 - \frac{\alpha' N}{\gamma} \right) > 0 \Rightarrow \gamma > \alpha' N$$

and this contradicts the hypothesis.

LEMMA 4 If $\alpha' N - \gamma > 0$, then Q_0 is g.a.s. in $D_1 = [0, X^*] \times [0, Y^*] \setminus \{Q^*\}$.

Proof. $V'(X, Y) \leq 0 \iff Y \leq Y^*$. On the line $\{Y = Y^*\}, Y' < 0 \iff X < X^*$ and this limits the invariant region in which V' < 0. Then V is still a Lyapunov function on D_1 and verifies V' = 0 on $N_1 \cap D_1$. On the axis $\{X = 0\}, X' \geq 0$, and we conclude that the largest invariant subset of D_1 is the singleton $\{Q_0\}$ and again from LaSalle's invariant principle we get that Q_0 is g.a.s. in D_1 .

If $\sigma + k_1 < 0$, is D_1 the basin of Q_0 or is the basin of Q_0 larger? The answer is in the following proposition.

PROPOSITION 1 If $\alpha' N - \gamma > 0$, then the basin of attraction of Q_0 is a triangle formed by the axes and a line that goes through the point Q^* and has slope

$$\frac{\sigma+k_1}{\sigma(\alpha'N-\gamma)}\,\left\{\frac{\alpha'Nk_1}{\gamma-\alpha'N}+\lambda_1\right\}.$$

Proof. Q^* is a saddle point with a stable and an unstable manifold both of dimension one. Let λ_1 be the negative eigenvalue and E^s the eigenspace associated to λ_1 and W^s the manifold that is tangent to $Q^* + E^s$ at each point. E^s is the straight line with slope

$$\frac{\sigma + k_1}{\sigma(\alpha'N - \gamma)} \left\{ \frac{\alpha'Nk_1}{\gamma - \alpha'N} + \lambda_1 \right\}$$

and this number is negative. This means that there is a triangular region formed by the axes and E^s that forms the basin of Q_0 . A trajectory that starts in this region cannot leave the region because the vector field at the axes points inwards and it cannot cross E^s . Therefore this region is invariant and all trajectories starting inside the region must have Q_0 as it ω -limit set.

4.2 Global stability of Q^*

In the case where the point Q^* is feasible the phase portrait for the system formed by the first two equations of (1) is divided into three cases according to the value of σ . We consider the curves C_1 where X' = 0 and C_2 where Y' = 0 in the region \mathcal{D} :

- (1) σ < 0. In this case the two curves C₁ and C₂ divide the quadrant into four regions as denoted in Fig. 4. The union of regions II, III and IV (denoted by S) forms a positively invariant domain. Every trajectory starting in this region stays in the region and every trajectory starting at a point (x₀, y₀) ∈ S is contained in a compact set determined by the boundary of S and the line y = y₀. Therefore, any trajectory that enters S stays in it, and the ω-limit set of any trajectory in S is the point Q* (there are no other stable critical points and there are no limit cycles). A trajectory that starts in I must enter S, as X' is negative in I and the vector field is transversal in the boundary of S. In this case then all trajectories enter S and they have as ω-limit set the point Q*. Then the point Q* is globally stable.
- (2) $\sigma = 0$ (Fig. 5). In this case the curve C_1 is a vertical line and is a trajectory of the system (in reality, three trajectories, two half-lines and the point Q^*). This divides the phase plane into two parts that are positively invariant. If a trajectory starts in I, then X is decreasing and Y is increasing; this implies that the trajectory enters region II at a point (x_1, y_1) , as the flow is transversal along C_2 . We can then build a rectangle by taking the rectangle formed by the points $\{(x_0, y_0), (x_1, y_1), (x_2, y_1), (x_2, y_0)\}$, where x_2 is any value $0 < x_2 < X^*$. The trajectory stays inside this rectangle, therefore its ω -limit set is the point Q^* . A similar analysis can be done if the trajectory starts at a point in III or IV.



(3) σ > 0. As the phase portrait indicates (Fig. 6), if a trajectory starts at a point (x₀, y₀) in I then it must cross into region II at a point (x₁, y₁); then it goes into III at a point (x₂, y₂) from there it goes into region IV at (x₃, y₃) and from there back to region I. This establishes a circular flow. The trajectory stays inside a rectangle of vertices {(x₁, y₀), (x₁, y₂), (x₃, y₂), (x₃, y₀)} and as there are no closed orbits the ω-limit set is the point Q*.

From the analysis of these three cases we obtain the following theorem.

THEOREM 1 In the system (5), if the equilibrium point Q^* exists in the positive quadrant and it is locally stable then it is globally asymptotically stable.

From this result we can now look into the global stability of the point P^* in system (1).

THEOREM 2 In system (1), if the equilibrium point P^* exists in the region \mathcal{D} and it is locally stable then it is globally asymptotically stable.

Proof. The proof rests on the global stability of the point Q^* for the system (1). Integrating the last equation in (1) we obtain

$$Z(t) = \left(\beta \int_{t_0}^t e^{\mu' s} (X(s) + Y(s)) \, ds + Z(t_0)\right) e^{-\mu' t} \ge 0 \quad ; \quad t \ge t_0$$



FIG. 5. The case $\sigma = 0$.

By L'Hôpital's rule, if $\lim_{t \to \infty} X(t) = X^0$ and $\lim_{t \to \infty} Y(t) = Y^0$ then

$$\lim_{t \to \infty} Z(t) = \lim_{t \to \infty} \frac{\beta \left(X(t) + Y(t) \right)}{\mu'} = \frac{\beta \left(X^0 + Y^0 \right)}{\mu'}$$

As the point Q^* is globally asymptotically stable in the system (5), then $X^0 = X^*$ and $Y^0 = Y^*$ and we have

$$\lim_{t \to \infty} Z(t) = \frac{\beta \left(X^* + Y^* \right)}{\mu'} = Z^*.$$

Now let (X(t), Y(t), Z(t)) be a solution of (1). Then

$$||(X(t), Y(t), Z(t)) - (X^*, Y^*, Z^*)||_{\infty} \leq ||X(t) - X^*||_{\infty} + ||Y(t) - Y^*||_{\infty} + ||Z(t) - Z^*||_{\infty}$$

and this tends to 0.

5. Application to the Cuban HIV/AIDS data

We will use the model (1) to fit the data for the known HIV positives and AIDS cases in Cuba. We use the following values for the parameters:



FIG. 6. The case $\sigma > 0$.

X(0) = 230, estimated from the number of HIV positives that were found after 1986 and were already infected at that time,

Y(0) = 94, number of HIV positives that were alive at the end of 1986,

Z(0) = 3, number of AIDS cases that were alive at the end of 1986,

 $\mu = 0.0053$, yearly mortality rate for the HIV+ cases for 1991–1997, (S.D. = 0.00254), computed from the number of death for HIV infected persons not related to AIDS,

 $\mu' \in [0.66, 0.85]$, obtained from the 95% confidence interval for the median of the survival time to AIDS,

 $\alpha N = 0.5594$ is obtained from parameter λ in Arazoza *et al.* (2000).

We fit the model to the data to obtain values for α' , k_1 , k_2 and β by minimizing a relative error function. As traditional optimization methods failed to work properly we used a genetic algorithm approach. To compute standard errors for the parameters, 300 fitting runs were made using different values for μ and μ' taken randomly from their confidence interval.

We obtain the values shown in Table 2 ($\alpha' = r \alpha$).

TABLE 2 Parameters

Parameter	Mean	Standard deviation
r	0.0579	0.035 5
k_1	0.3743	0.03979
k_2	0.0000227	0.000 034 67
в	0.10788	0.001 67



FIG. 7. Cuban HIV epidemic.

In Fig. 7 (known HIV) and Fig. 8 (AIDS) we can see the data and the curve given by the model using the mean value for the parameters.

6. Discussion

From the values we have obtained, we can see that k_2 is the parameter that varies the most (relatively). The value of the equilibrium point P^* is very sensitive to these variations, as k_2 appears in the denominator of (2). If we take a value for k_2 in the upper half of the confidence interval, for example $k_2 = 0.000085$, we get the endemic point $P_1^* = (487, 2665, 420)$, but if we take the mean value we get $P_2^* = (1996, 11050, 1872)$. This gives us an epidemic that can go from a rate of 324 per million to one of 1356 per million. Points P_1^* are in the region \mathcal{D} , and as conditions given in (6) are satisfied they are globally asymptotically stable following Theorem 2.

Point P_1^* gives us a total of 324 per million as the rate of persons living with HIV/AIDS at the equilibrium, and 38 per million living with AIDS. The level found for the number of unknown HIV infected persons (487) is consistent with the one found in Arazoza *et al.* (2000) where it was found that the number of unknown HIV infected persons for the year 1997 was in the interval [342, 486].



FIG. 8. Cuban AIDS epidemic.

 k_1 may be considered as the inverse of the average time from infection to detection for a person that is detected not through contact tracing but through one of the 'blind' or 'random' types of search for seropositives. In this case, for an optimal value of k_1 , the value we get is 34 months or 2.85 years. In Arazoza *et al.* (2000) a similar coefficient was found but including also contact tracing, and at that time we obtained a value of 26 months or 2.2 years for the average time between infection and detection. We can see here that contact tracing is an important element in the control of the epidemic—without contact tracing a person infected with HIV is not aware of his infection for almost three years; contact tracing helps reduce this time by 23%.

Parameter k_2 is crucial for the size of the epidemic; the Health System should continue to improve the efficiency of contact tracing.

The results obtained from the model give indications that any search method that is based on targeting a group that has been in contact with persons that carry the human immunodeficiency virus is far more important in the control of the epidemic than a method that is directed to the general population. Special emphasis should be devoted to instrument such search methods if the HIV/AIDS is to be controlled.

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REFERENCES

ALTMAN, L. (1997) Sex, Privacy and Tracking the HIV Infection, New York Times.

- APRIL, K. & THÉVOZ, F. (1995) Le Contrôle de l'entourage ('Contact Tracing') a été négligé dans le cas des infections par le VIH. *Revue Médicale de la Suisse Romande*, **115**, 337–340.
- ARAZOZA, H., LOUNES, R., HOANG, T. & INTERIAN, Y. (2000) Modeling HIV epidemic under contact tracing—the Cuban case. J. Theor. Med., 2, 267–274.
- CDC (1991) Transmission of multidrug resistant tuberculosis from an HIV positive client in a residential substance-abuse treatment facility—Michigan. *MMWR*, **40**, 129.
- HETHCOTE, H. W., YORKE, J. A. & NOLD, A. (1982) Gonorrhea modeling: comparison of control methods. *Math. Biosci.*, **58**, 93–109.
- HETHCOTE, H. W. & YORKE, J. A. (1984) Gonorrhea Transmission Dynamics and Control, Lecture Notes in Biomathematics, vol. 56. Berlin: Springer.
- LOUNES, R. & DE ARAZOZA, H. (1999) A two-type model for the Cuban national programme on HIV/AIDS. *IMA. J. Math. Appl. Med. Biol.*, **16**, 143–154.
- PÉREZ, J., TORRES, R., JOANES, J., LANTERO, M. & DE ARAZOZA, H. (1996) HIV control in Cuba. *Biomed. Pharmacother.*, **50**, 216–219.
- RUTHERFORD, G. & WOO, J. (1988) Contact tracing and the control of human inmunodeficiency virus. J. Amer. Med. Assoc., 259, 3609–3610.