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METHODOLOGY TO DETERMINE THE EVOLUTION OF ASYMPTOMATIC HIV POPULATION USING FUZZY SET THEORY

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The aim of this paper is to o study the evolution of positive HIV population for manifestation of AIDS, the Acquired Immunodeficiency Syndrome.

For this purpose, we suggest a methodology to combine a macroscopic HIV positive population model with an individual microscopic model. The first describes the evolution of the population whereas the second the evolution of HIV in each individual of the population. This methodology is suggested by the way that experts use to conduct public policies, namely, to act at the individual level to observe and verify the manifest population.

The population model we address is a differential equation system whose transference rate from asymptomatic to symptomatic population is found through a fuzzy rule-based system. The transference rate depends on the $CD4+$ level, the main T lymphocyte attacked by the HIV retrovirus when it reaches the bloodstream. The microscopic model for a characteristic individual in a population is used to obtain the $CD4+$ level at each time instant. From the $CD4+$ level, its fuzzy initial value, and the macroscopic population model, we compute the fuzzy values of the proportion of asymptomatic population

at each time instant t using the extension principle. Next, centroid defuzzification is used to obtain a solution that represents the number of infected individuals. This approach provides a method to find a solution of a non-autonomous differential equation from an autonomous equation, a fuzzy initial value, the extension principle, and center of gravity defuzzification. Simulation experiments show that the solution given by the method suggested in this paper fits well to AIDS population data reported in the literature.

Keywords: Epidemiological modeling; HIV population model; dynamic fuzzy modeling; fuzzy set theory.

1. Introduction

Traditionally, engineering and applied mathematics have endeavored to model and solve technological problems. Nowadays, they are becoming increasingly important to the non technological world as well, especially for bioengineering, medicine and epidemiology to mention a few.

Despite considerable advances in its biological foundations, epidemiological modeling still needs appropriate mathematical and computational structures to deal with imprecision and uncertainty, apart from those treated by stochastic models. The theory of the fuzzy sets,[1] and systems[2–4] provide key notions to model epidemiological phenomena.

The first application of fuzzy set theory in biomathematics dates medical diagnosis area,[5,6] where most of its use were concentrated. More recently, fuzzy set theory has shown to be useful in a variety of other areas, epidemiology being one of the most fruitful.[7–12] In epidemiology, the same disease can be displayed in different ways and with different degrees of severity in different patients. Often, diseases characteristics and symptoms are qualified linguistically and are intrinsically imprecise because they usually refer to biological variables. In medical sciences we frequently encounter difficulties when using conventional quantitative approaches and methods. There is also a close relationship between microscopic and macroscopic phenomenon, which means that the models are of difficult analysis to comprehend the phenomenon as a whole. A common approach in this case is to develop a broader model and use different scales.

In this paper, we first generalize the classical Anderson population model[13] assuming that the transference rate from asymptomatic to symptomatic population, λ, is a parameter whose values depend on the $CD4+$ level. The values of the transference rate are found via a fuzzy rule base derived from expert medical knowledge. This brings the transference rate closer to its intended biological meaning. Second, from the solution of a non-linear system of differential equations,[16] viewed as a microscopic model for HIV infection dynamics, we obtain $CD4+$ levels as function of time. Next, given a fuzzy initial condition, we find a fuzzy solution for the microscopic model. From the fuzzy values of $CD4+$ obtained from the microscopic model and from the extension principle, whose transformation function is the solution of the generalized population model, we find a fuzzy solution that models the evolution of the proportion of the infected asymptomatic population. The fuzzy

population solution is defuzzified using the center of gravity. We show that the defuzzified solution actually is a solution of a non-autonomous differential equation, the one that emerges if we consider the transference rate a function of the $CD4+$ and time. Finally, we verify that the result provided by the methodology addressed here fits well known population data reported in the literature. The paper concludes suggesting issues that need further developments.

2. Classic AIDS Models

The classical Anderson's model[13] is a macroscopic model for AIDS given by:

$$\begin{aligned} \frac{dx}{dt} &= -\lambda(t)x & x(0) &= 1 \\ \frac{dy}{dt} &= \lambda(t)x = \lambda(t)(1-y) & y(0) &= 0 \end{aligned} \tag{1}$$

where $\lambda(t)$ is the transference rate between infected individuals and infected individuals that develop AIDS, x is the proportion of infected population that does not have AIDS symptoms yet (asymptomatic), and y is the proportion of the population that has developed AIDS symptoms (symptomatic). Anderson assumes[13] $\lambda(t) = at$, $a > 0$. Thus the solution of (1) is

$$x(t) = e^{-\frac{at^2}{2}} \qquad y(t) = 1 - e^{-\frac{at^2}{2}} \tag{2}$$

Peterman and co-workers[17] report data related to 194 cases of blood transfusion-associated AIDS. From Peterman[17] data, Murray[13–15] shows that Anderson's model (1) can be adjusted through a best-fit procedure to find the value for the parameter a. The rate of increase $\frac{dy}{dt}$ of AIDS patients as a function of time, provided by the Anderson's model (1), is shown by the continuous curve of Figure 1. Notice that this scheme provides a best fit to data solution, with no clear biological explanation for the transference rate origin.

Novak and Bangham (1996) introduced a microscopic model for HIV infection dynamics in the individuals organism with no anti-retroviral therapy. In particular, it models HIV positive individuals during the asymptomatic phase. Therefore, we adopt Novak and Bangham model once it is closely related with the purpose of this paper.

Four variables are considered: uninfected cells n, infected cells i, free virus particles v and z which denotes the magnitude of the CTL (cytotoxic T lymphocyte), that is, the abundance of virus-specific CTLs. Infected cells are produced from uninfected cells and free virus at rate βnv and die at rate bi. Free virus is produced from infected cells at rate ki and declines at rate sv. Uninfected cells are produced at a constant rate, r, from a pool of precursor cells and die at rate an.The rate of CTL proliferation in response to antigen is given by ciz. In the absence of stimulation, CTLs decay at rate dz. Infected cells are killed by CTLs at rate piz (see Ref. 16 for further details). These assumptions lead to the following system of differential

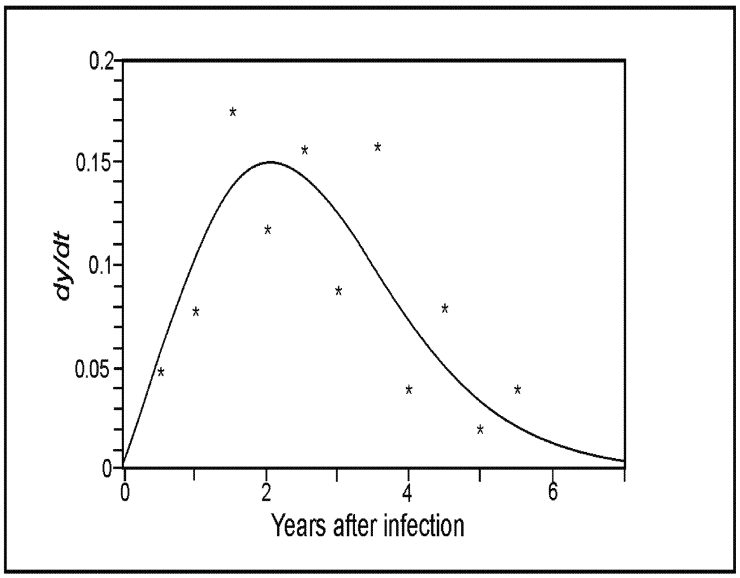

Figure 1. The rate of change in the proportion of the population who develop AIDS who were infected with HIV (through blood transfusion) at time $t = 0$. The data, from Ref. 17, provide a best-fit value of $a = 0.237yr^{-1}$ for model solution (2).

equations:

$$\begin{aligned} \frac{dn}{dt} &= r - an - \beta nv \\ \frac{di}{dt} &= \beta nv - bi - piz \\ \frac{dv}{dt} &= ki - sv \\ \frac{dz}{dt} &= liz - dz \end{aligned} \tag{3}$$

Figure 2 shows the solution of the microscopic HIV model for parameters and initial conditions given in Tables 1 and 2, respectively, obtained from Ref. 18.

Table 1. Parameters of the microscopic model used in simulations.

$r = 0.3$	$a = 0.3$	$\beta = 0.6$
$b = 0.01$	$p = 0.03$	$k = 0.5$
$s = 0.01$	$l = 0.01$	$d = 0.01$

The uninfected cells of $CD4+$ show a rapid decline in the first weeks with a slow recovery when the number of lymphocytes is close to the maximum (depicted

Table 2. Initial conditions used in simulations of the microscopic model.

$n(0)$	0.99
$i(0)$	0.01
$v(0)$	0.1
$z(0)$	0.01
t initial	0 time units
t final	500 time units

in logarithmic scale in Figure 2(a)). The increase in the number of lymphocytes is related to the presence of infected cells and the virus replication mediated by them.

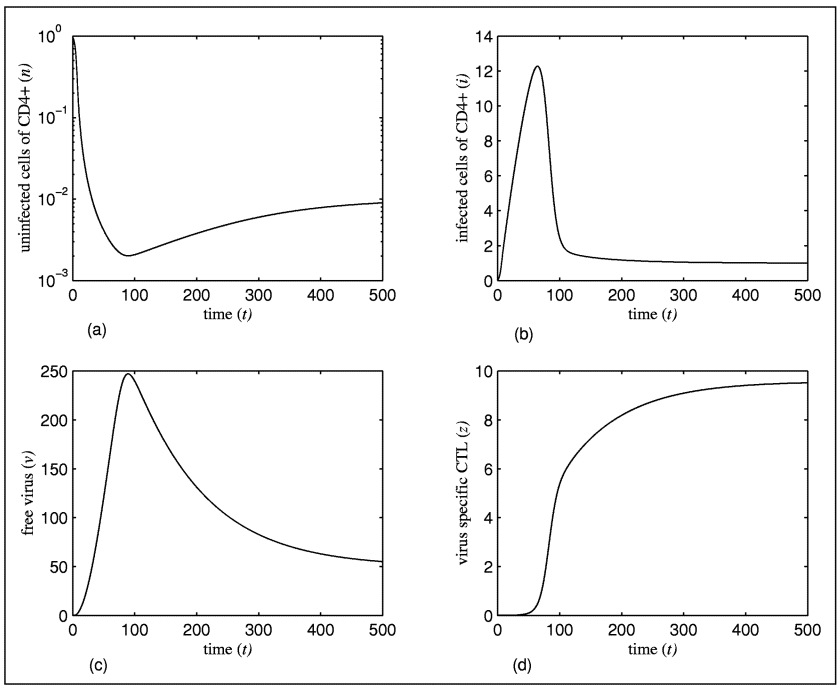

Figure 2. Numerical solutions of system (3).

Figure 3 is a schematic view of the currently accepted natural history of HIV infection in medical sciences. Comparing the solution of system (3) shown in Figure 2 with Figure 3, we notice that the uninfected cells of $CD4+$ identifies with the $CD4+$ level, the free virus with the HIV virus, and the virus-specific CTLs with the HIV antibodies. These correspondences will be important to derive a macroscopic, HIV asymptomatic population model using fuzzy set-based modeling.

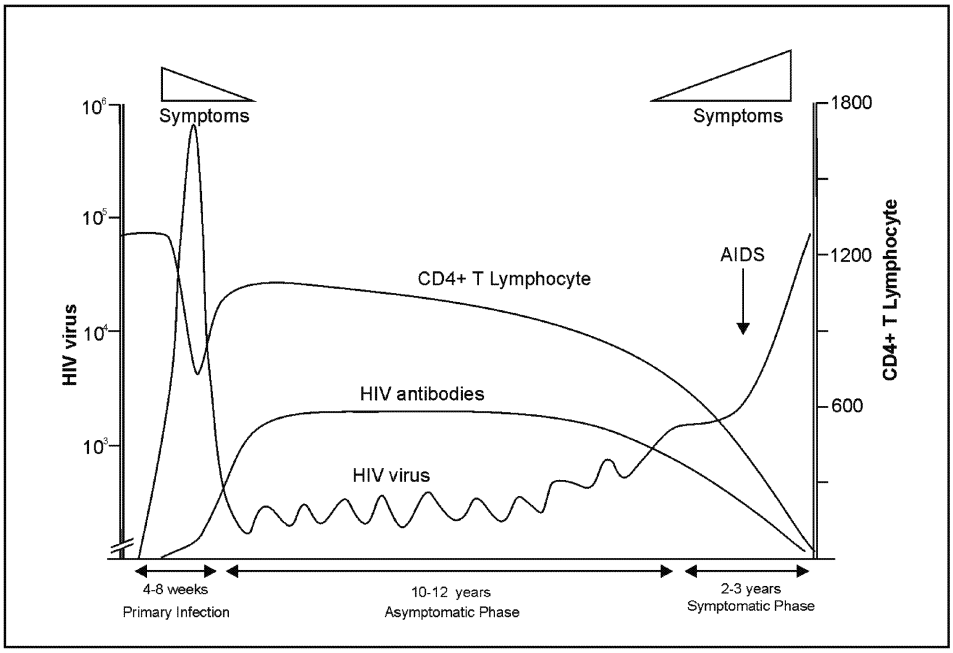

Figure 3. Schematic representation of the currently accepted natural history of HIV infection.[19–21]

3. Modeling HIV Population Evolution

When HIV reaches the bloodstream it attacks mainly the lymphocyte T of the $CD4+$ type. The amount of cells $CD4+$ in peripheral blood has prognostic implications in infection evolution by HIV. Nowadays, the amount of immune competence cells is the most clinically useful and acceptable measurement to follow the evolution of infected individuals by HIV, although it is not the only one. The identification of the disease's stages and its respective treatment is based on the relationship between viral load and $CD4+$ level. The viral load and $CD4+$ cells level interfere in the transference rate λ . Thus, the conversion from an asymptomatic individual to a symptomatic individual depends on the individual characteristics, as measured by the viral load v and level of $CD4+$ (c). Therefore, we assume the following, as a generalization of model (1):

$$\begin{aligned} \frac{dx}{dt} &= -\lambda(v,c)x & x(0) &= 1 \\ \frac{dy}{dt} &= \lambda(v,c)x = \lambda(v,c)(1-y) & y(0) &= 0 \end{aligned} \tag{4}$$

The difference between the model suggested in (4) and the classic model (1) is that in (4) the parameter $\lambda = \lambda(v,c)$. This assumption comes from its biological meaning and is a more faithful characterization of the transference rate because it

Table 3. Fuzzy rules.

$CD4+$ \ V	*low*	*medium*	*high*
very low	*strong*	*strong*	*strong*
low	*medium*	*strong*	*strong*
medium	*medium*	*medium*	*medium*
high medium	*weak medium*	*weak medium*	*medium*
high	*weak*	*weak*	*weak*

depends of the viral load and the of the $CD4+$ level.

To get the relationship $\lambda = \lambda(v, c)$ we adopt expert knowledge encoded in the form of a fuzzy rule base. This approach seems to be appropriate since medical experts use viral load and $CD4+$ level values to infer the infection phase and to decide the proper treatment. The rule base that encodes the relationship between c, v, and λ, as suggested by expert medical knowledge, is summarized in Table 3. Viral load (v) and the level of $CD4+$ (c), and the transference rate (λ) are linguistic variables denoted by V, $CD4+$ and λ , respectively. Viral load V has its values in {*low*, *medium*, *high*}, $CD4+$ in {*very low*, *low*, *medium*, *high medium*, *high*}, and transference rate in the term set {*weak*, *medium weak*, *medium*, *strong*}. The membership functions that specify the meaning of the linguistic variables are shown in Fig. 4, 5 and 6 for viral load, $CD4+$ level, and transference rate, respectively.

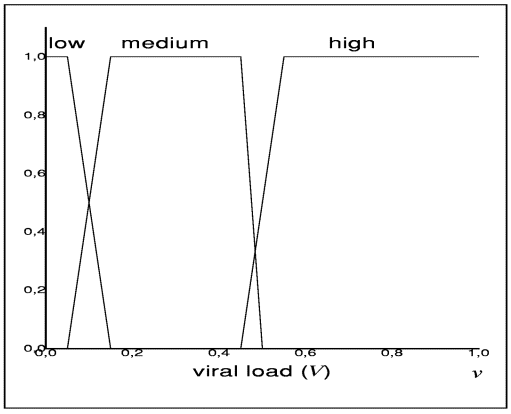

Figure 4. Membership functions for viral load (V).

From the mathematical point of view (4) can be seen as a parametric family of systems. It seems reasonable that λ, and consequently the population of infected individuals y, varies with v and c. From (4) we have

$$\begin{aligned} x(t) &= e^{-\lambda(v,c)t} \\ y(t) &= 1 - e^{-\lambda(v,c)t}, \qquad\qquad t > 0\,. \end{aligned} \tag{5}$$

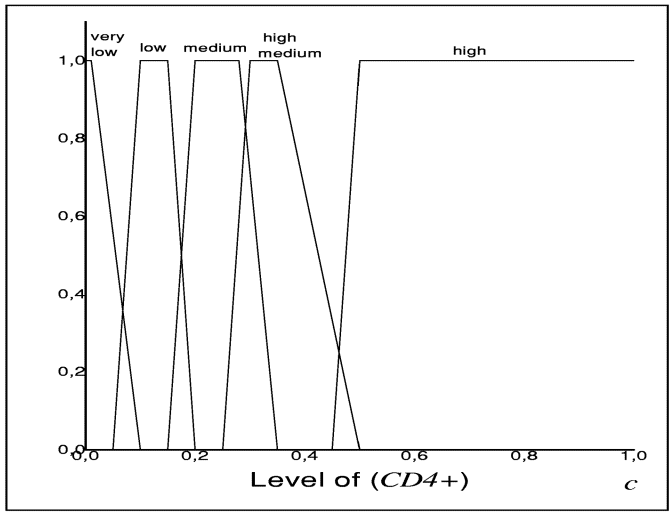

Figure 5. Membership functions for $CD4+$ level.

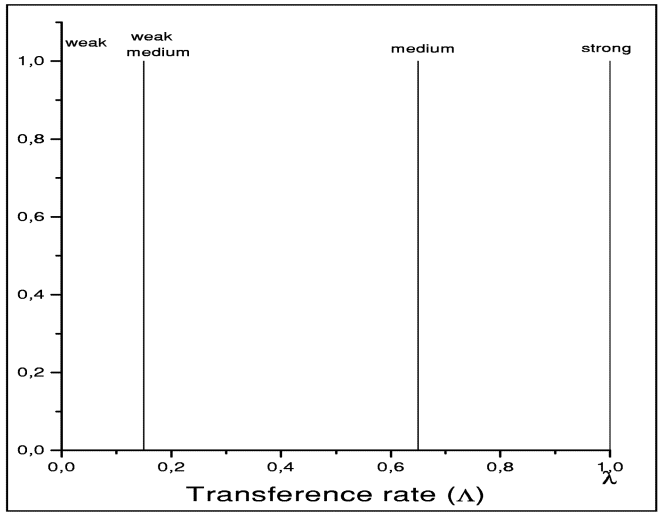

Figure 6. Membership functions transference rate (λ).

See Refs. 8 and 9 for further details the fuzzy rule-based model developed to find the transference rate, given the viral load and the $CD4+$ level. The rule base is processed using the Mamdani inference method with center of gravity defuzzification.[9]

We note that, according to experts, there is an inverse relationship between viral load and $CD4+$ level during the asymptomatic phase, when the individuals are not HIV manifest. It is also interesting to note that the microscopic model reveals the following relationship between viral load and $CD4+$ level.

$$c(v) = \frac{r}{a + \beta v} \tag{6}$$

This relationship between $CD4+$ level (c) and viral load (v) is justified when we compare the solution of system (3) of Figure 2 with the history of HIV infection of Figure 3, where the uninfected cells of $CD4+$ identifies with the $CD4+$ level, the free virus with the HIV virus, and the virus-specific CTLs with the HIV antibodies. This is because, during the asymptomatic phase, the variation of uninfected cells of $CD4+$ is small. Therefore, we may assume $\frac{dn}{dt} \cong 0$ which means that $n(v) \cong \frac{r}{a + \beta v}$. Since blood test does not differentiate uninfected cells n from infected cells i (current blood test identifies $CD4+$ level only) we may also assume $CD4+$ proportional to n. Thus, (6) provides an approximation of the relationship between $CD4+$ level (c) and viral load (v).

When the surface of the transference rate produced by fuzzy inference and defuzzification is intersected with the graph of (6), the result is the piecewise linear curve shown in Figure 7. When we 1) approximate the defuzzified transference rate curve by the smooth curve $\lambda(v, c)$, and 2) project the smooth $\lambda(v, c)$ curve in the transference rate versus $CD4+$ level plane, the projection becomes (7), illustrated in Figure 8.

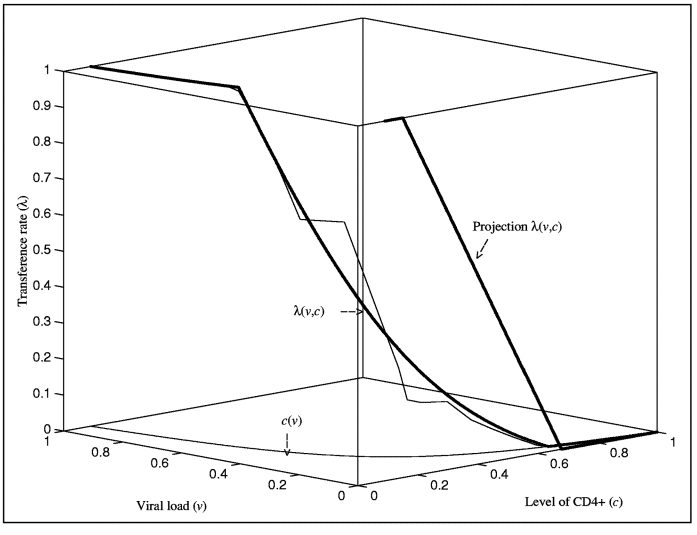

Figure 7. Approximation of the transference rate for values of $c(v)$ and its projection in the $CD4+$ plane.

$$\lambda(c) = \begin{cases} 1 & \text{if } 0 < c < c_{min} \\ \dfrac{c_M - c}{c_M - c_{min}} & \text{if } c_{min} \leq c \leq c_M \\ 0 & \text{if } c > c_M \end{cases} \tag{7}$$

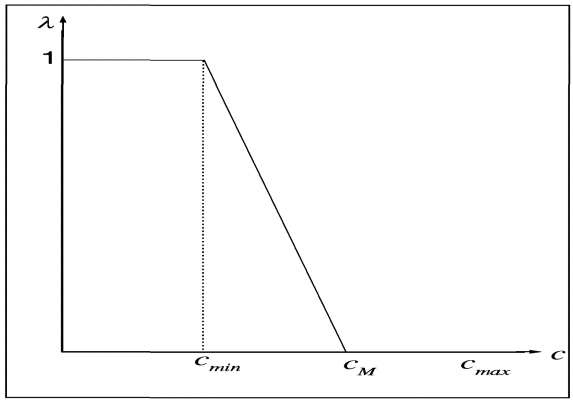

Figure 8. Transference rate λ as a function of c.

In Figure 8, c_{min} is the largest value of $CD4+$ for which the chance of an individual to become symptomatic is maximum, c_M is the smallest value for which the chance to become symptomatic is minimum, and c_{max} is the largest possible level of $CD4+$.
From (4) and (7), we conclude:

$$\begin{aligned} \frac{dx}{dt} &= -\lambda(c)x & x(0) &= 1 \\ \frac{dy}{dt} &= \lambda(c)x = \lambda(c)(1-y) & y(0) &= 0 \end{aligned} \tag{8}$$

Solving (8), we have:

$$\begin{aligned} x(t) &= e^{-\lambda(c)t} \\ y(t) &= 1 - e^{-\lambda(c)t}, \qquad t > 0. \end{aligned} \tag{9}$$

As discussed previously, the $CD4+$ level is the most useful information to follow HIV symptoms evolution. We assume that the microscopic model (3) describes the time behavior of the $CD4+$ level ($c(t)$) for the population as well. If we assume (9) and (7) we have $x(t) = e^{-\lambda(c(t))t}$. Moreover, if $\lambda(c(t))$ is differentiable, then $x(t)$ is the solution of the following differential equation

$$\frac{dx}{dt} = -\left[\lambda(c(t)) + t\frac{d\lambda}{dt}(c(t))\frac{dc}{dt}(t)\right]x \tag{10}$$

In practice, the value of $c(t)$ is uncertain because initially we have individuals with different $CD4+$ levels in the population. Therefore we assume that the initial value $C(0) = C_0$ is a fuzzy set. Hence, the population model suggested in this paper actually is a non-autonomous differential equation with fuzzy time-varying parameter.

Hullermeier[22] suggests that the solution of a fuzzy differential equation be a fuzzy function obtained from a family of differential inclusions. Mizukoshi et al.,[24] have shown that when parameters (coefficients and/or initial conditions) are fuzzy, the Hullermeier solution is the same as the one obtained from the extension principle. Therefore, we first obtain the solution of the classical differential equation and next we use the extension principle to get fuzzy solutions. To obtain a real-valued trajectory, we adopt the center of gravity to deffuzify the fuzzy solution. We note that, in principle, any defuzzification method could be chosen (see Ref. 9 for an alternative based on the Sugeno integral).

In the next section we show how to find a solution of the non-autonomous differential equation from the extension principle, given the corresponding autonomous equation solution and the fuzzy time-varying parameter values.

4. Method to Solve Asymptomatic HIV Population Model

The asymptomatic HIV population model is an instance of non-autonomous fuzzy differential equation, once (10) depends on a time-varying parameter, the $CD4+$ level, whose initial value is a fuzzy set. A method to obtain a solution for (10), given an initial fuzzy value for $c(t)$, is summarized in Figure 9.

First, we note that from the fuzzy rule base and inference system we determine the transference rate $\lambda(v, c)$ of the macroscopic model, and using the relationship between the $CD4+$ level c and viral load v (6) we obtain (7). Thus, the composition of (5) and (7), for t fixed, denoted by $x_t(c)$ is

$$x_t(c) = \begin{cases} e^{-t} & \text{if } c < c_{min} \\ e^{-\lambda(c)t} & \text{if } c_{min} \leq c \leq c_M \\ 1 & \text{if } c > c_M \end{cases} \tag{11}$$

where $\lambda(c) = \frac{c_M - c}{c_M - c_{min}}$. Next, assume that the population of HIV-positive studied has the $CD4+$ level initially characterized by a triangular membership function u_{C_0} for C_0, Figure 10:

$$u_{C_0}(c) = \begin{cases} 0 & \text{if } c \leq \underline{c} - \delta \\ \frac{1}{\delta}(c - \underline{c} + \delta) & \underline{c} - \delta < c \leq \underline{c} \\ \frac{-1}{\delta}(c - \underline{c} - \delta) & \underline{c} < c \leq \underline{c} + \delta \\ 0 & \text{if } c > \underline{c} + \delta \end{cases} \tag{12}$$

The parameter $\underline{c}$ is the modal value and δ the dispersion of the fuzzy set C_0, whose universe contains c_{min}, c_M and c_{max}.

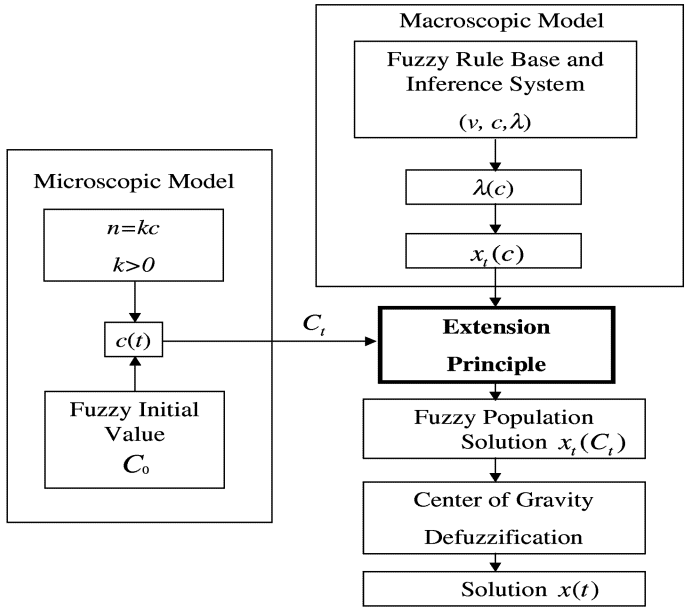

Figure 9. Method to find the evolution of the asymptomatic HIV population.

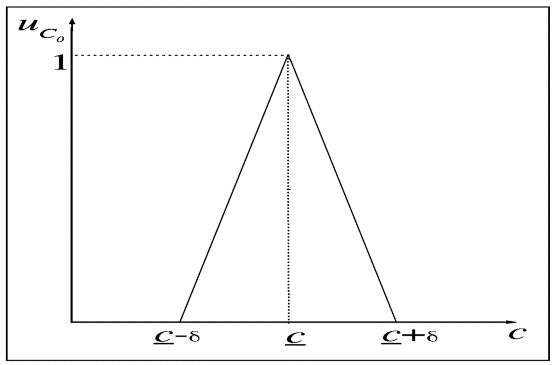

Figure 10. Membership function for C_0.

4.1. *Solution of microscopic model with fuzzy initial value*

As suggested in Refs. 22, 23 and 24, we solve the non-linear differential equations system (3) for each value of c_0 within the support of C_0, that is, for each $c_0 \in supp(C_0)$. We assume that the corresponding solution $c(t)$ has the same membership degree as does c_0. Therefore the solution of (3), given the fuzzy initial value C_0, is a fuzzy set C_t whose membership function is u_{C_t}. Figures 11 and 12 show the solution obtained for C_0 of Figure 10 with $\underline{c} = 0.89$ and $\delta = 0.1$.

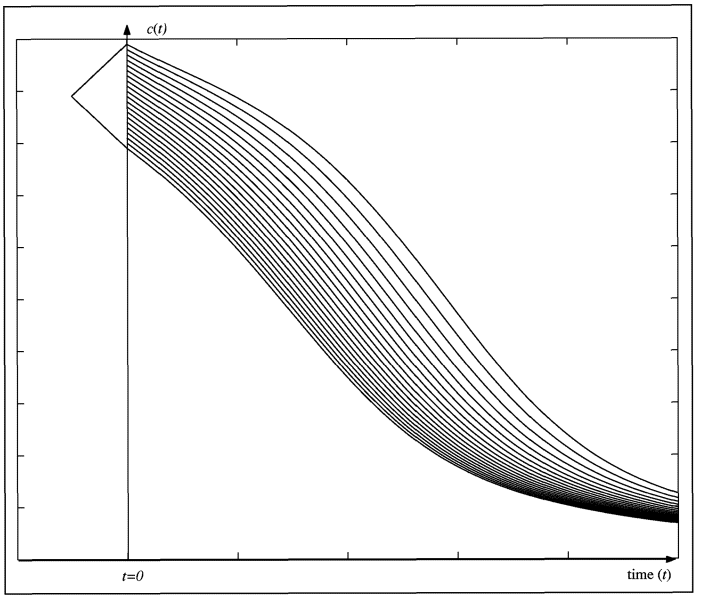

Figure 11. Solutions $c(t)$ for $c_0 \in supp(C_0)$.

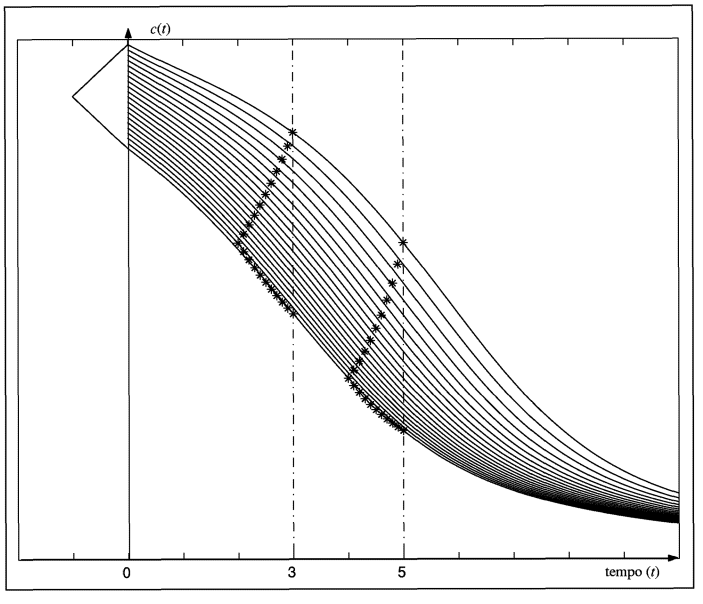

Figure 12. Fuzzy solution C_t at $t = 3$ and $t = 5$.

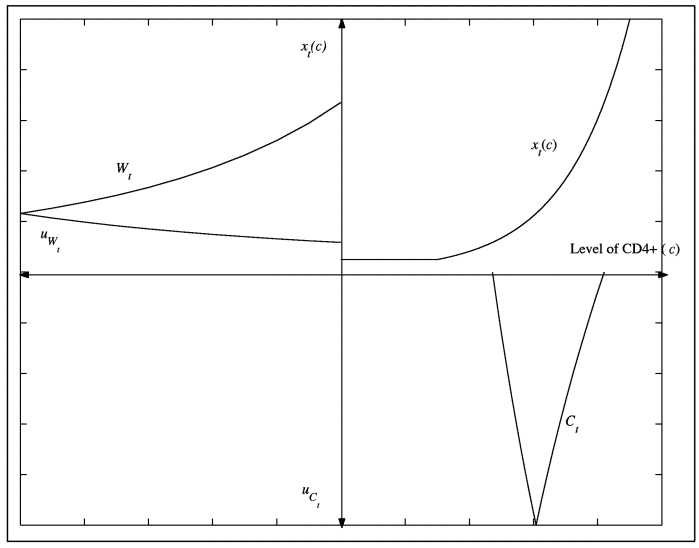

Figure 13. Computation of W_t at $t = 3$.

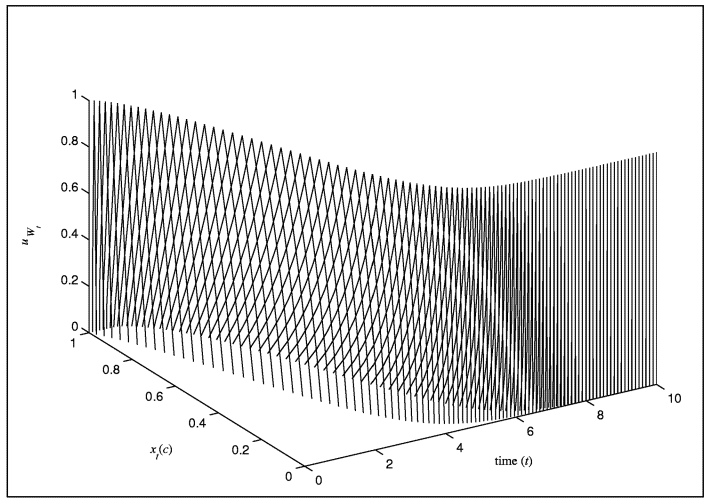

Figure 14. Membership degree of the proportion of asymptomatic population at each time instant t.

4.2. *Extension principle*

In this subsection, the extension principle is used to obtain the image of the fuzzy set C_t through function (11). More specifically, from extension principle we have, for each time instant t:

$$u_{W_t}(x_t(c)) = \sup_c u_{C_t}(c) \tag{13}$$

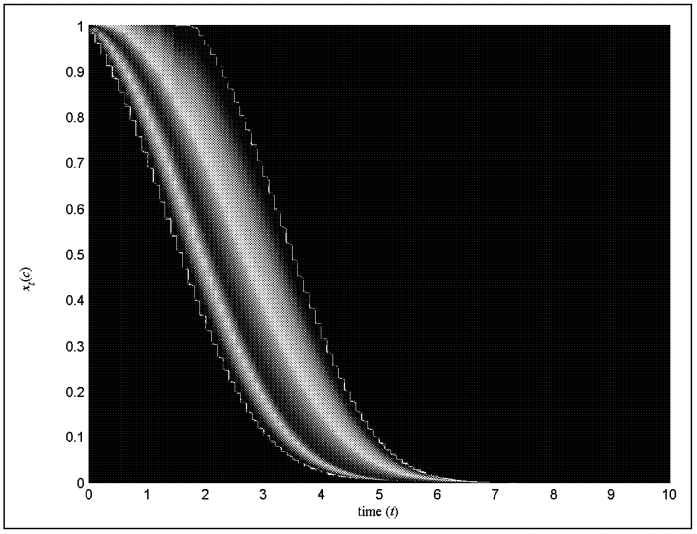

Figure 15. Fuzzy population solution $x_t(C_t)$.

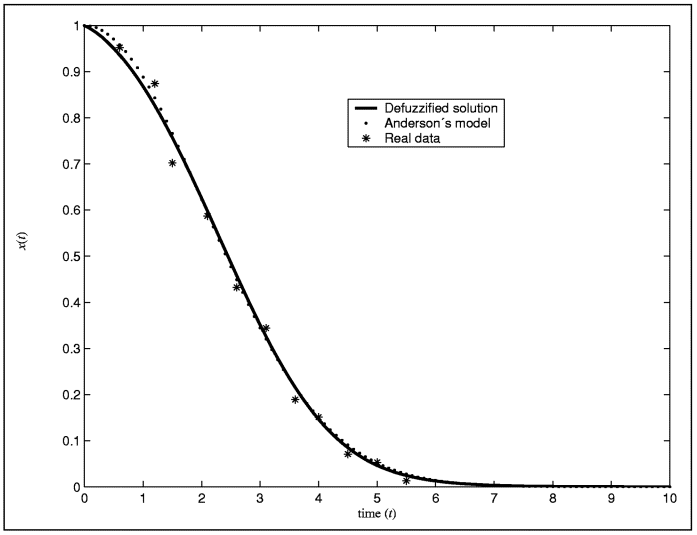

Figure 16. Comparison between the defuzzified solution and real data.

where C_t is the fuzzy $CD4+$ level at t whose membership function is u_{C_t}, and W_t is the corresponding fuzzy set at t with membership function u_{W_t}. Figure 13 illustrates W_t at $t = 3$ whereas Figures 13, 14 and 15 show the solution $x_t(C_t)$ assuming C_t evolving as in Figure 12.

4.3. *Defuzzification*

The last step of the method, as indicated in Figure 9, aims a representative solution. A common defuzzification scheme is the center of gravity method. Let u_{W_t} be the membership function of $x_t(C_t)$ and denote $x_t(c)$ by x_t to simplify notation. Then a real-valued output $x(t)$ is chosen, at each time instant t, as follows:

$$x(t) = \frac{\int\limits_{supp(W_t)} x_t u_{W_t}(x_t)dx_t}{\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t} \tag{14}$$

For instance, given the fuzzy solution shown in Figure 14 we obtain, using the center of gravity, the defuzzified solution depicted in Figure 16.

5. Justification of the Method

In what follows, we show that the center of gravity defuzzification of (14) is a solution of (10). Let

$$x(t) = \frac{\int\limits_{supp(W_t)} x_t u_{W_t}(x_t)dx_t}{\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t} \tag{15}$$

As $\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t$ is constant and $\int\limits_{supp(W_t)} \frac{u_{W_t}(x_t)}{\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t}dx_t = 1$, we have

$$x(t) = \int\limits_{supp(W_t)} x_t \frac{u_{W_t}(x_t)}{\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t}dx_t = E(x_t) \tag{16}$$

where $E(x_t)$ may be viewed as the expected value of x_t. Since $x_t = e^{-\lambda(c)t}$ we get

$$x(t) = E(e^{-\lambda(c)t}) = \int\limits_{supp(C_t)} e^{-\lambda(c)t} \frac{u_{C_t}(c)}{\int\limits_{supp(C_t)} u_{C_t}(c)dc}dc \tag{17}$$

From the medium value theorem for integrals, and because $f(c) = e^{-\lambda(c)t}$ is continuous and $p(c) = \frac{u_{C_t}(c)}{\int\limits_{supp(C_t)} u_{C_t}(c)dc}$ is integrable and positive, we have $x(t) = e^{-\lambda(c(t))t}$ for some $c(t) \in supp(C_t)$. Note that

$$e^{-\lambda(c(t))t} = \frac{\int\limits_{supp(W_t)} x_t u_{W_t}(x_t)dx_t}{\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t} \tag{18}$$

or,

$$\lambda(c(t)) = -\frac{ln\left(\frac{\int\limits_{supp(W_t)} x_t u_{W_t}(x_t)dx_t}{\int\limits_{supp(W_t)} u_{W_t}(x_t)dx_t}\right)}{t}, t \neq 0 \tag{19}$$

From (19), $\lambda(c(t))$ (see Figure 17) is differentiable in t. If $\lambda(c(t))$ is differentiable in t, then $x(t)$ (15) is a solution of the non-autonomous differential equation (10). That is, from an autonomous differential equation, the method generates a representative (defuzzified) solution $x(t)$ that actually is a solution of the non-autonomous differential equation (equation (10)).

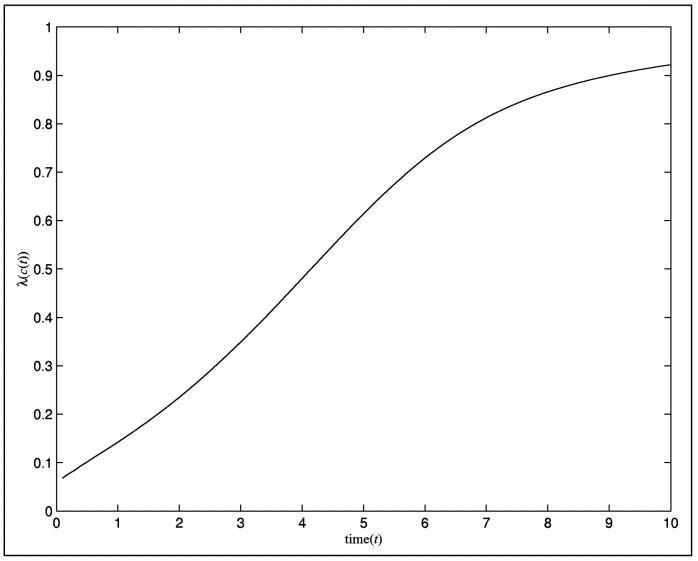

Figure 17. $\lambda(c(t))$.

We notice that in the method suggested in this paper, the idea is to obtain $x(t)$ following the steps shown in Figure 9 instead of to solve an equation such as (10) since this equation is unknown *apriori*. The solution $x(t)$ is obtained from a family of classic differential equations, but it does not coincide with any solution for a fixed c. However, for each t, $x(t)$ is a value that belongs to the unique solution of the family of equations parameterized by c. Therefore, what differs the solution derived with the method suggested here from the deterministic solutions is that the deterministic solutions all uncertainties are excluded in the beginning (defuzzify at $t = 0$ and solve) whereas here the uncertainties evolves and defuzzification occurs at the time instant of interest (defuzzify when needed).

6. Comparing the Fuzzy Set-Based Solution with Real Data

As discussed in Section 2, Ref. 13 presents the Anderson's model (1) adjusted with a best-fit procedure to find a to the data of Ref. 17. We use the same data of Ref. 13, as shown in Figure 1. Since it gives the values of $\frac{dy}{dt}$, we integrate the original data to get $y(t)$. Next we compute $x(t)$ from $x(t) + y(t) = 1$. To find

the solution of the infected population given by the fuzzy model (8), we assume $c_{min} = 0.01$ and $c_M = 0.95$. As we note in Figure 16, the defuzzified solution closely matches the original data, as does Anderson's model. However, differently from the Anderson's model, the solution obtained via the fuzzy set-based methodology comes from expert knowledge and biological principles and does not depend on the availability of population data.

7. Conclusion

This paper has suggested a methodology to determine the evolution of positive HIV population and manifestation of AIDS, focusing on the nature of the transference rate of asymptomatic to symptomatic, and on the fuzzy character of the $CD4+$ level values. The main difference between the classic model (1) and the fuzzy model (8) introduced in the paper is that the fuzzy model exploits expert knowledge and the inherently imprecise values of biological parameters, whereas the classic model does not. In a sense, the classic model is a particular instance of the fuzzy model. In addition, Anderson's model parameter is derived from a best fit to data procedure while the fuzzy methodology is constructed from biological principles and biological information. The fuzzy set-based methodology provides a clear and meaningful characterization of the asymptomatic population behavior once it is compatible with medical knowledge and perception of its dynamics. The solution has shown to be close to data reported in literature.

The contributions of the paper are manifold. It suggests a scheme to combine microscopic (individual) and macroscopic (population) models to study the dynamics of populations and act in individuals as it is actually done in practice; it provides a mechanism in which uncertainty is considered as a part of the solution and representative values of the time trajectories are computed whenever necessary to evaluate population state and to decide on the proper health policies; it proposes a simpler method to obtain non autonomous differential equations with fuzzy initial conditions than more sophisticated and general methods such as differential inclusions, and fuzzy differential equations such as those of the Hullermeier class.

Future work will evaluate the effect of viral load and $CD4+$ levels in the transference rate of symptomatic to asymptomatic individuals considering populations with regular adhesion to treatment, and to model HIV positive populations with irregular adhesion to anti-retroviral therapy.

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