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The impact of HIV/AIDS epidemic on the population age structure of Zimbabwe

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Introduction

The end of this century has been a period of great transformation for Zimbabwe.
As many other developing countries in this period, the nation have gone through a demographic transition, particularly evident during the eighties. This kind of transition is characterized by a sharp decrease in fractional death rates, due the rapid improvement in life conditions, and a slow corresponding decrease in fractional birth rates, which are strongly related to sexual habits and traditions.
Zimbabwean demographic transition, however, has been, and actually is very atypical and dramatic, due to the enormous HIV/AIDS epidemic. The fractional death rates have begun to re-increase during the nineties, while birth rates kept on decreasing, due to cultural factors and to the reduction of reproduction capacity of the population, related to the erosion of the fertile population and to the AIDS kids generation caused by the epidemic.
The population growth has slowed down and the big wave of population born during the eighties will ultimately increase the work force only partially, due the strong “filter” effect of the epidemic.
Owing to the fact that the HIV/AIDS epidemic affects only specific age classes from the population, the resulting age structure of Zimbabwe at the beginning of the new millennium is consistently different from what otherwise could have been. The ratio between the work force and the total population, in particular, is considerably lower than the level it would have reach in the hypothetical case in which the epidemic would not have occurred. What is worst is probably that the major effect of the epidemic in this sense is still to come: if the main conditions related to HIV diffusion would not change, during the next decade the work force would be around twenty percent lower than the level we would have observed if the virus would not have spread.
These disequilibria in the population age structure can seriously compromise the development possibilities of the nation, leading to even worse life conditions, which will finally again affect the population development.
The HIV/AIDS multistage model presented here has been expressly developed and adapted to the specific Zimbabwean situation to evaluate the effects of the implementation of different possible policies, which are intended to fight the Virus diffusion, on the population as a whole and specifically on its age distribution.
Very interesting insights have been gained by testing the model with different actual policies, specifically referring to the introduction of the HAART treatment, which is able to

stretch the expected lifetime of infected subjects and improve their life conditions and/or the introduction of Viramune/Nevirapina, a drug particularly efficient in reducing the risk of virus transmission from mother to child.
The results of these kinds of interventions have also been compared with those of more soft oriented policies, such as sensitization or information campaign, and the opportunity to integrate one with each other has been evaluated.
The resulting model behaviors in these hypothetical scenarios have in some cases been counterintuitive and ultimately lead to reflection on the recent development of HIV/AIDS international agreements.

An introduction to the model

The main scope of this work is to build a structure reflecting the most important aspects, on a highly aggregated level, of the spreading of the HIV/AIDS disease, with an endogenously generated infection rate, to test the effect of the implementation of different policies on the population's age structure. The model should represent the main mechanism of spread of the disease, to keep trace of the subjects infected, of their age, sex, and clinical evolution. It should incorporate the effects of changes in the population, both of its total number and of its age and sex distribution on the diffusion of the epidemic. It should allow the client to test different possible scenarios, considering changes in people sexual habitudes, in infectivity of the disease and in the incubation times. The model should also take care of the problem of HIV positive kids from infected mothers.

In this model, the infection rate will be modeled as a function of infectivity of the disease, the number of potentially dangerous contacts generated by each person and the probability for an infectious person to have a contact with a susceptible one, given by the ratio between susceptible and the total population. These assumptions mainly reflect the work firstly made by Kermack and McKendrick in 1927 with the SIR model that has already been applied to the study of many different diseases, here re-elaborated with some important differences. In particular the infection rate is calculated on the basis of the number of contacts generated by the infected population and of the probability for them to encounter a susceptible person, and not vice-versa as in the SIR model. This distinction, while mathematically is not relevant, becomes conceptually relevant when we assume that infected population is divided in subgroups with different behaviors.
In fact in the SIR model the infection rate is calculated as:
IR=(ciS)(I/N)
where: "c" is the contact rate, "i" is the infectivity (the probability to be infected when in contact with an infectious subject, "S" is the total number of susceptible persons, "I" represent the infectious population and "N" is the sum of "I" and "S". This formulation, calculating the number of contacts generated by the susceptible population (c*S) and multiplying it by the infectivity and the probability to meet an infectious subject (I/N) does not permit to distinguish between different categories among infectious subjects, with different behaviors or infectivity. Therefore, the formulation adopted in this model is calculated as:
$IR=(c_1*i_1*I_1*)(S/N) + (c_2*i_2*I_2*)(S/N) +\ldots+ (c_n*i_n*I_n*)(S/N) = \sum_{i=1,n}(c_{i*}i_{i*}I_i)(S/N)$

where c_i and i_i are the specific contact rate and infectivity respectively, for the group of infectious I_i. This formulation allows the modeler to distinguish between different subgroups of infectious people, with different epidemiological characteristics.
This distinction made, the main forces driving the diffusion and the eventual depletion of the disease are the same of those considered in the SIR model (fig.1)

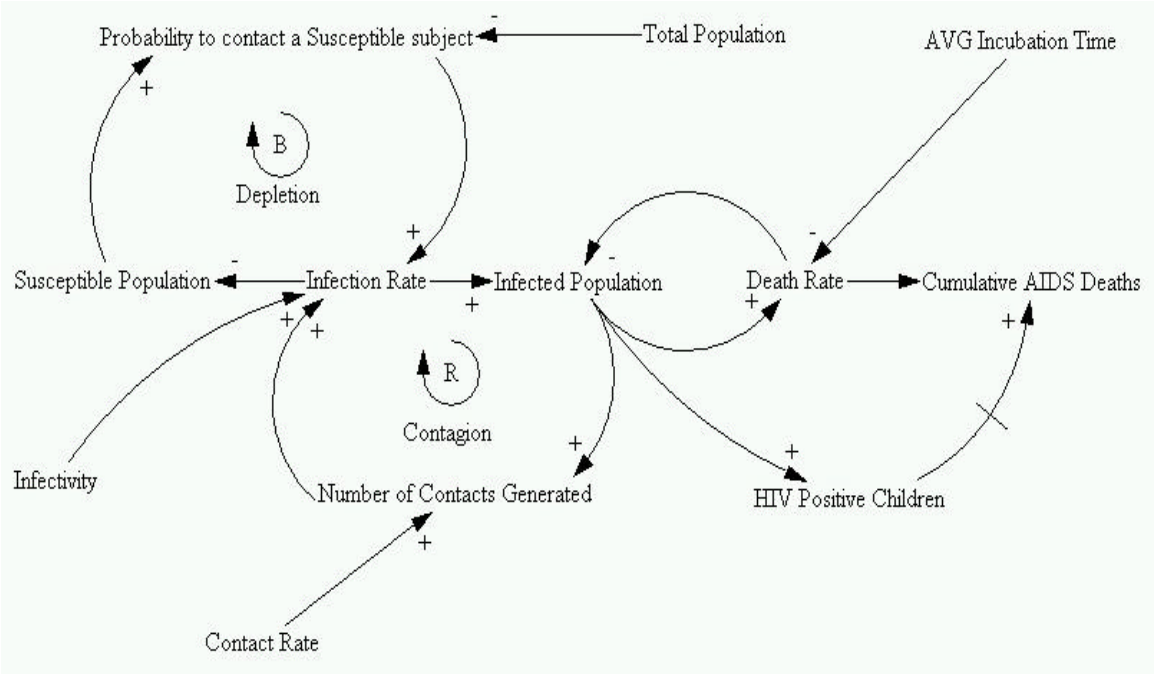

Fig.1

This simplified causal loop diagram highlights the main feedback loops relevant in the system. In this case the only reinforcing loop is the "contagion" loop, which highlights the idea that if the number of infected people increases, assuming that the number of contacts each person is able to generate remains constant, the total number of contacts generated by infected people will increase. This, ceteris paribus, would increase the infection rate, that would increase again the number of infectious people. Although as more people will get infected, the number of subjects in the susceptible population stock will decrease and the ratio between susceptible and infected population will be reduced, which means that the probability for an infectious person to meet a susceptible one decreases: this is the effect described by the susceptible population "depletion" loop. As well, when the infected population increases, with a certain delay (the incubation time) the death rate will increase, draining subjects to the cumulative HIV/AIDS related deaths stock. This will reduce again the level of infected people, closing the "AIDS Deaths" balancing loop.
Besides, while people are located in the infected population stock, they can generate HIV positive kids that will eventually die as well (the incubation time is in this case relevantly shorter).
Naturally, the relative strength of those loops will not be constant during the course of the epidemic: particularly important is the switch in dominance between the reinforcing "Contagion" loop and the balancing "Depletion", that will cause the system, under normal conditions, to produce the typical s-shaped behavior in the cumulative number of

infections. At the beginning of the epidemic the reinforcing loop will be dominant, driving an exponential growth in the number of infected people. In the mean time, the balancing loop will become stronger and stronger while subjects are moved from the susceptible to the infected population, and it will finally become dominant. That will be the moment when the infection rate will reach its maximum value, corresponding to the inflection point in the cumulative number of infections function. The system will then show a goal seeking behavior in the cumulative number of infections, driven by the "Depletion" balancing loop, until the infection rate will eventually fall to zero.

As it clearly appears from the infection rate function description and from the causal loop diagram, the population stock has a major dynamic role in the system: any external influence on this stock, as migrations or food scarcity, would have a major effect on the course of the epidemic.
This requires the model to be integrated with a population model, taking into account the evolution of the susceptible base and of the population as a whole.
In fact the whole model has been built as a population model, where people transit from one stage of HIV/AIDS to another as they are still part of the population (fig.2), aging and being subjected to natural death causes, just as the rest of the population is.

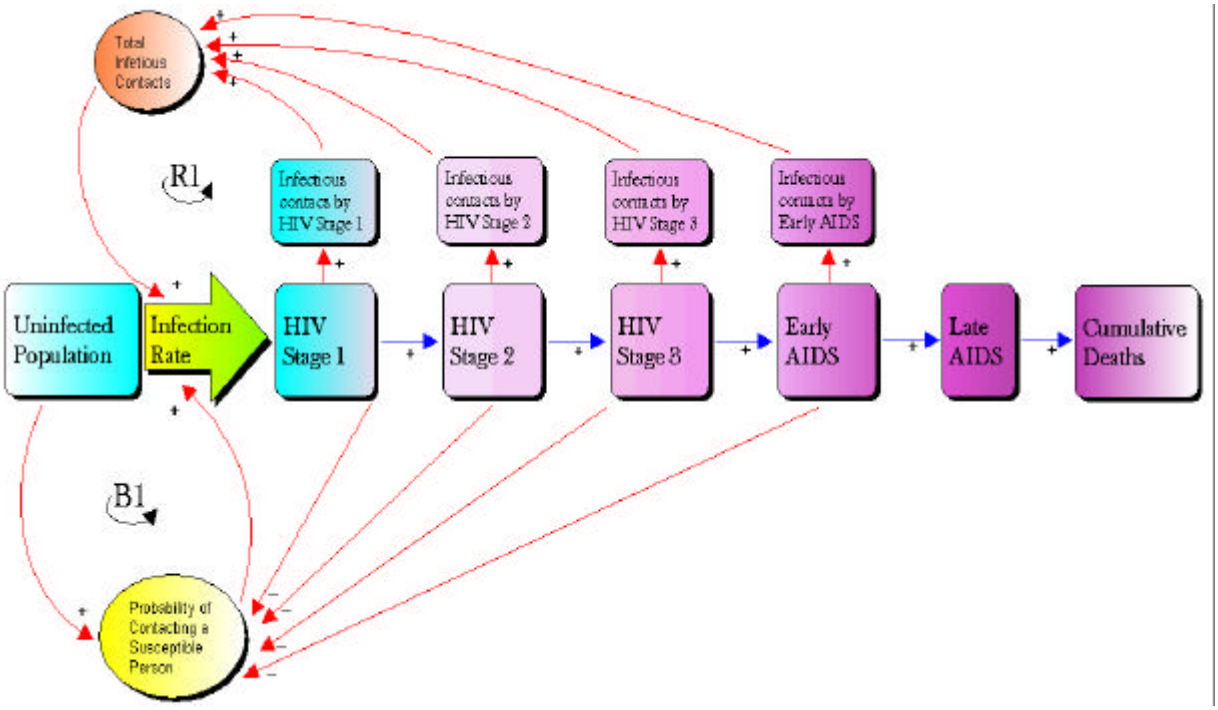

Fig.2

As shown in figure 2, the total living population is divided in six different stocks: the uninfected population and five other stocks representing different stages in the development of the disease in the subjects. In each HIV stage and eventually in the Early AIDS stage people generates a certain number of infectious contacts depending on the particular

infectivity and contact rate of that stage. These contacts can eventually be with a susceptible person, depending on the ratio between the part of the uninfected population that can generate this kind of contact and the part of the total population that can generate this kind of contact. The two main loops, R1 and B1, responsible respectively of the initial exponential growth in the cumulative number of infections and of the following goal seeking behavior, are highlighted by the figure.
People are moved from the uninfected population stage to the first HIV stage by the infection rate, and then to the following stages depending on the specific incubation time associated with each stage. Moreover in each stage people are subjected to the same natural death rate (deaths not related to AIDS).
Births in the model can be generated both by the infected and the uninfected population: while in the latter case the totality of births would flow into the uninfected population stock, in the former case a percentage of the kids, depending on the probability of HIV transmission from mother to child (known as perinatal transmission), would flow into a separate stock of HIV positive children whose lives will be very short, lasting in most of cases between one and two years.

The idea of differentiating the HIV/AIDS population into five different stages is based on the research work conducted in 1999 by B. Dangerfield and C. Roberts and in 2001 by B. Dangerfield, C. Roberts and Y. Fang. The justification of this kind of structure comes from a medical and a social analysis of HIV/AIDS subjects (the analysis has been conducted in Great Britain, but most of the results can be extended to the same study field in different nations).
From a medical point of view, subjects infected with HIV pass through different stages before eventually dying. In the following description, incubation times are considered as the average time spent by subjects in each stage, if they do not assume any treatment.
Straight after the infection, the organism of the infected person reacts very strongly to the virus and, while any symptom is unlikely to appear, the subject infectivity is very high. However, this period normally lasts only for about 4 months. In the next stage, which lasts on average around nine years, the subject's infectivity decrease considerably and no symptoms are normally shown. This is the longer stage characterizing the development of the disease, and due to the lack of symptoms and information, in many cases (not just in developing countries) the subject still have no idea about his/her condition.
The third stage is characterized by a new increase of infectivity and by the eventual appearance of minor symptoms. This stage normally lasts about one and a half year. The development of major AIDS symptoms is associated with the transition to the early AIDS stage: in this period of about six months the subject is still able, in most cases, to conduct an almost normal life, and is normally not yet hospitalized. In the last three months of life, hospitalization is required and the patient must be assisted.
In developing countries, in many cases, the subjects realize their seropositivity condition only when major symptoms appear, in the forth or fifth stage, when sometimes they even die without any idea of the cause. This implies that they did not have any reason to change their sexual behavior and they have been helping to spread the disease for about ten years; therefore, when they realize their condition it is too late to change their habits, because in many cases they can't be sexually active and infect anyone.

As it clearly appears from the description above, the distinction in the model between these different stages is extremely important, due to the different degrees of infectivity characterizing the subjects in the different moments.
From the point of view of the social relationships, this distinction is also very important. In fact the development of symptoms can reduce the capability of the subject to generate more contacts, because of a physical incapacity (as in the late AIDS stage) or because of the reduced desire of contacts of the subject and the limited possibility to attract partners (as is more likely to happen in the early AIDS stage).
The model is particularly focused on the representation of the mechanism of spread of this disease in the sub-Saharan nations, where the main way of transmission of the virus is via heterosexual contacts. Transmission via blood (for example used needles) is not considered and the distinction between homosexual and heterosexual contacts is assumed to be not relevant, because of the extremely low percentage of homosexual males in these countries.
The time horizon chosen reflects the long-term perspective of the analysis: to go far enough in the future to understand the effects of the epidemic on the development of the population and to capture the effects of potential policies actually implemented, I decided to set the end of the simulation in the year 2050.
While the first HIV infections seems to appear in the late seventies, I decided to start the simulation well before, in 1965, when surely no infections where detected. This allows the user to observe the trend of the evolution of the population before the spread of HIV and to get an idea on how it would have been without the diffusion of this disease and therefore about the real impact of the virus on the population development.
This 85 years long time horizon is consistent also with respect to the huge delays involved in the biological development of the virus within the body, giving an enormous inertia to the epidemic.
In fact, the average life span after the infection is around ten years without any treatment, a period that can be increased significantly with the introduction of the HAART treatment.
This means that even if we would be able to stop immediately the diffusion of the virus, the epidemic would still have major effects on the population for ten to fifteen years to come.

This basic structure, together with the formulation of the infection rate above illustrated, is able to represent the main spreading mechanism of the disease, to keep trace of the subjects infected, of their age, sex, and clinical development. It can guarantee the model the capacity to capture the effects of changes in the population due to food scarcity, migrations, or other external shocks, on the disease development. The possibility to test different policies, introducing changes in infectivity and incubation time, due for example to the introduction of new treatments, or changes in the sexual behavior of subjects has also been included. Changes in the people sexual behavior can be introduced in two ways: the first possibility is to assume that the subject's behavior can change while progressing in the different HIV/AIDS stages, because of the increasing consciousness of the disease; the second possibility consist of considering the sexual behavior of whole population to change over time, while they become more sensitive to the problem of the epidemic diffusion.

Model Boundaries

The purpose of this model is not to explain endogenously the whole population dynamics of one Nation, but to capture those dynamics related to the HIV/AIDS epidemic. That is why the model is not demanded to generate endogenously all the rates influencing the population stock. In particular the fractional birth rates are exogenous to the model and are based on statistical estimations. The utilization of exogenous fractional birth rates also implies another assumption: the fractional birth rates are not assumed to be influenced by the development of the epidemic. In fact, a causal relationship between the diffusion of HIV and a decrease in the fractional birth rates seems to me difficult to find: only if we assume an increase in the utilization of condoms, due to an increase in the perceived risk of infection, we should consider a reduction in the birth rates, but only with respect to the undesired part of births. If we consider the situation of a nation in which this part of the birth rate is forecasted to be very high for the years to come, we could easily incorporate such an effect.
The natural death rates can also be considered as exogenous to the model. In fact their estimation is based on a reference year death rates, multiplied by the effect of the average daily number of calories ingested per person on the death rates. The data relative to the evolution of the alimentation conditions are based on statistical estimations.
The net migration rate is exogenous as well: particularly it is exogenous to the model the first immigration of HIV infected people. In fact there are still no scientific proves of the origin of the human forms of the HIV virus and the I assumed an immigration of a little number of infected people as the initial cause of the epidemic.
These are the main variables considered as exogenous to the system: more specific assumptions relative to each sector of the model is treated in the “Appendix 1”, where a sector-by-sector analysis of the model is conduced.
In the “Appendix 2” the conclusions of the analysis of the validity of the model from a structural point of view are reported.

Fitting a real nation’s situation

The HIV/AIDS epidemic in Zimbabwe

The structure of the model has been designed particularly to represents the virus diffusion in sub-Saharan developing countries, where the diffusion of the virus is mainly related to the heterosexual contacts, where sexual habits are particularly free and people’s knowledge of HIV/AIDS is very limited. Some specific modifications have then been introduced to fit the Zimbabwean situation.
I decided to concentrate the research work on Zimbabwe because of many factors. Firstly Zimbabwe is among the first five countries with the highest HIV prevalence, with an adult esteemed rate higher than 25% at the end of 1999. This very high prevalence rate, together with the relatively high population, which was exponentially growing before accusing the effect of the epidemic, and with the average degree of economic and social development characterizing Zimbabwe, suggest this nation to present interesting and highly representative data about the epidemic development and its impact on the population. Another very important factor determining this choice is the relative political stable period that the country experienced from the end of the seventies until 1999. In fact even the

economic crisis the country is living since 1999 is not very relevant if compared to the recent political events in many other sub-Saharan countries. This allowed the collection of good data series, while in most developing countries the quality of such data is not very high, both on the population and on the epidemic development. In this second case, in particular, the only data we can consider as affordable is the number of the cumulative AIDS related deaths reported, which represents only one part of the total AIDS related deaths. Many estimations based on this basic data and made by different organizations are also available. Unfortunately data relative to the period between 1966 and 1981 are mainly missing or not affordable, due to the civil war that the country experienced in that period. A last factor that pushed me to choose this nation to implement a first application of the model, is the strong impression I personally had in this country in 1999 about the heavy impact of the epidemic, both on people as individuals, from the point of view of the human sufferance affecting a huge part of the population, and on the entire nation, from the point of view of the substantial limit to the socio-economic development the disease implies, basically destroying the work force and increasing importantly the medical assistance costs.
To give an idea of the dimension of the problem, AIDS is actually the major cause of death in Zimbabwe and 1,5 millions of people were estimated to live with HIV/AIDS in 1999. Considering the prevalence rate data resulting from statistical tests conducted in many areas, this numbers seem to be an underestimation of the real situation. In fact recent data talk about an ascertained prevalence rate in pregnant women oscillating between 30% and 50% depending on the specific country region, and in standard male hospital patients constantly over 50%, reaching the peak of 71% in some urban areas.
The real impact of the epidemic is still far from being discovered, due to the scarce monitoring system and the enormous delays involved in the development of symptoms related to HIV/AIDS and therefore in the emergence of cases.
All the specific modifications implemented in the model to fit the Zimbabwean situation are reported in the “Appendix 3”.

Sensitivity Analysis

With the above-mentioned specific adaptations, the model seems to fit very well the available data both on population and on the HIV/AIDS epidemic.
During the following analysis the model will be compared with the most affordable statistical data referred to the period from 1965 to 2000. For the period from 2001 to 2050, the model’s results will be compared, when possible, with other authoritative forecasts.

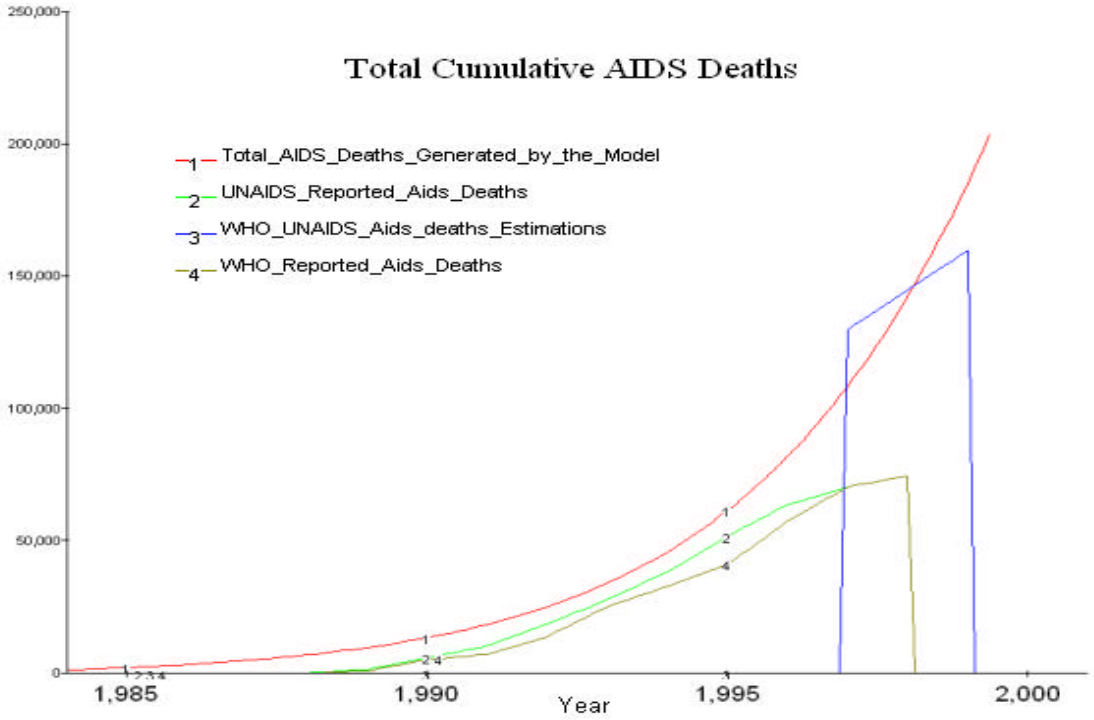

Fig.3

These two figures (fig.3 and fig.4) show the adherence of the model to statistical data respectively on population development and AIDS cumulative deaths.

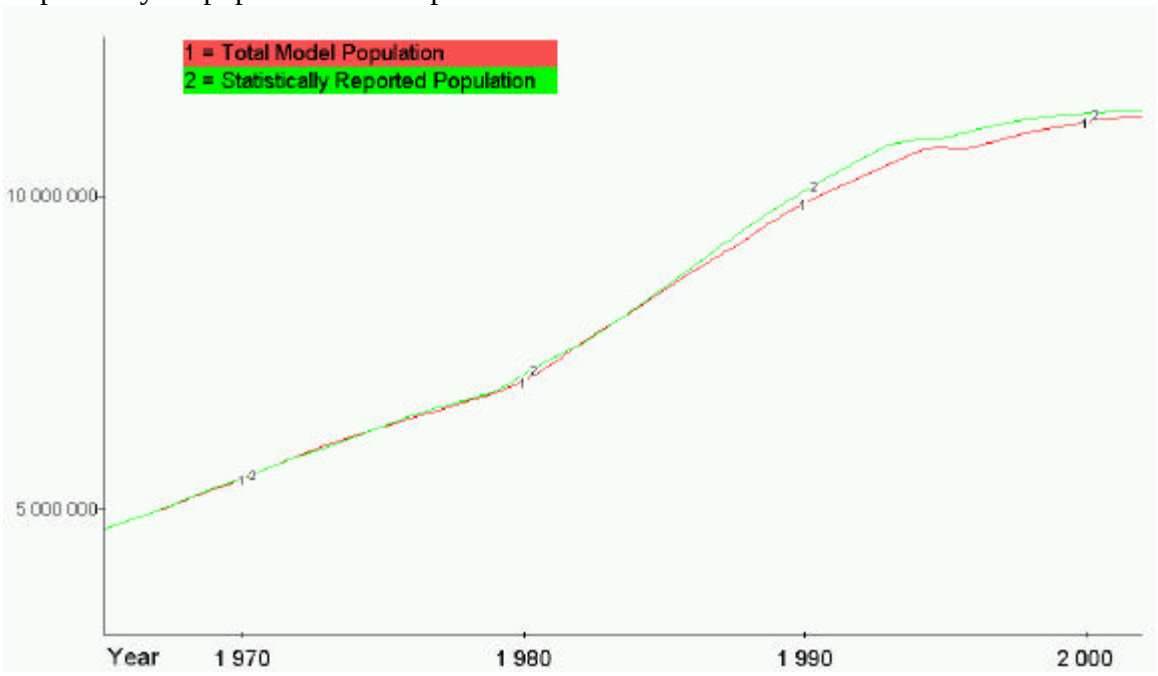

Fig.4

These fittings are obtained without any variation during the simulation of the parameters setting: in particular the sexual behavior of the population is supposed to remain constant in this period.
In both cases the data fitting seems to be very good, but the analysis of the quality of these results in terms of statistical adherence to the available data is outside the scope of this work and it would also be not very relevant due to the relative affordability of the data. Here only the results from the model sensitivity analysis for the most important estimated parameters are reported.
The first variable that will be taken into consideration is the effect of the civil war on the birth rate table function. To test the sensitivity of the model to changes in this parameter I initially simply run a simulation excluding completely any effect of the civil war on the birth rate. The result of the simulation is not surprising: the model overestimates the growth of the population in the period of the civil war, but after 1980 the patterns of growth of the population in the basic and the modified runs are again very similar, differing numerically but not qualitatively. However the net difference between the two data series reaches a maximum of approximately 11% in the year 2000. What is more important, the effect of this change in the population is not particularly amplified on the development of the epidemic, and the difference in the cumulative number of AIDS deaths observed in the year 2000 in the two cases is also roughly 11%. On the other side, similar results are obtained by setting an effect of civil war on birth rate double than the one utilized in the base run: the model population in this case will be depressed more than in the base case in the period of the civil war and will cumulate a gap lower than 6% of the total population in the year 2000. Accordingly the difference in the cumulative number of AIDS deaths between this hypothetical situation and the base run at the end of the millennium is lower than 6%.
The second variable taken into consideration will be the effect of calories per capita on death rate table function. Setting the model to consider no effects of the alimentation on the death rates, the model's population will grow more rapidly than in the basic case, particularly in the 80's and in the 90's, when the food availability in the country was seriously decreasing. However this discrepancy between the population calculated in the base run and the results generated in this case is very little, about 5% percent in the year 2000, and the difference in the cumulative number of AIDS deaths is even lower, around 1.6%. Similarly a change in the effect of calories per capita on the death rate in the opposite direction will cause the population to grow slower than in the base case, particularly between 1980 and 2000. Setting a double than normal effect the resulting population will be less than 5% lower than the normal one and the AIDS cumulated deaths number only 1.6% lower than what normally produced by the model.
Changes in the distribution of the war deaths over time have extremely low effect on the population, due to the overall low relevancy of the number of people drained by the population stock (only 25000 persons over 7 millions) and to the short time range in which can be reasonable to place the majority of these deaths (accordingly to the history, only in the second half of the seventies).
In the case of the three variables now analyzed, which represents the only cases in which table functions have been used, it is evident that the model sensitivity to the changes introduced is only numerical, while no behavioral mode sensitivity or policy sensitivity have been observed. Moreover, the most important thing is that no particular amplification

effects have been observed on the cumulative number of AIDS deaths due to these changes in the population level. Considering all the above, I can conclude that, with respect to the purpose of the analysis, the model's sensitivity to these parameters is perfectly tolerable.

Different is the case of the base contact frequency parameters, as well as the normal contact frequency parameter. These parameters represent the average number of sexual contacts that people have every year, and they may differ from an HIV stage to another, if we would assume that there is a great part of the infected population that knows of their infectiousness and decides therefore to modify their sexual behavior as a consequence. In the case of Zimbabwe, I assumed that most of the infected people do not know about their infectiousness and therefore the base contact frequency for the different HIV stages is considered to be equal to the normal contact frequency for uninfected people.

As already discussed, affordable data for this parameter is particularly difficult to find, while this is one of the parameters to which the model is more sensitive. In fact, a very small increase in this parameter from 19.25 to 20 (approximately a 4% increase) generates a number of cumulative AIDS deaths in the year 2000 higher than the one in the base case of almost 75%, and a reduction of the same magnitude creates a reduction in the AIDS deaths of more than 44%. This very high sensitivity highlights the necessity to be very careful in the estimation of this parameter, even in the case of a very good data fitting, as here is the case. Good data fittings can in fact also be obtained by other couples of values for the contact frequency and the initial number of infectious people "injected" in the population: in fact also the value assumed for this second variable is an estimation and can be subjected to errors. Even if the very little number of infectious people assumed to immigrate in the population at the beginning of 1978 is consistent with the information available, we do not have specific and exact data on this topic.

However, with respect to this variable, the model does not show a particular sensitivity: if we set the number of infectious immigrants to a value 1/3 higher or lower than in the base case, the model would show a variation between the cumulative AIDS deaths calculated in the base run for the year 2000 and those calculated with the modified parameter of 37% and 29% respectively.

What is more important, if we assume a value for this parameter lower or higher than its normal value of 33%, and then we adjust the contact frequency parameter for a good data fitting, the final results generated by the model, even in the long run, are not particularly different from those generated in the base case. In fact, if we consider the number of cumulative AIDS deaths in the year 2010, the two alternative simulations would calculate a value for this variable 10% higher and 10% lower respectively than the value obtained in the base run.

These results are particularly important meaning that not only from the point of view of the qualitative behavior and of the policy evaluation, but also from the numerical point of view the model does not seem to show a particularly high sensitivity to the couple of values chosen for the indicated parameters to fit the data.

A sensitivity analysis with respect to the infectivity and the incubation time parameters does not seem to be useful, due to the fact that both data comes from very accurate scientific estimations (Dangerfield and Roberts 1999 and Dangerfield, Fang and Roberts 2001) and that they are not likely to spontaneously change in nature. In fact there is still no proven possibility to change the infectivity of the virus, even pharmacologically, while

there is the possibility to change the incubation times by the application of the HAART treatment: the effect of such changes will be discussed in the policy analysis part.
The results of more accurate discussions on the structural validity of the model are reported in the "Appendix 2".

A long-term behavior discussion

To capture the real dynamics of the epidemic and to capture its effects on the population development and age structure, as it has already been discussed, I decided to go with the simulation far in the future until the year 2050. Up to now, the model behavior has been compared with the statistically reported data on population and on the epidemic evolution. From the year 2000, instead, the model behavior will be compared with the estimations on the population growth made by the U.S. Census Bureau.
Before focalizing the analysis on the age structure of the population, it is necessary to discuss the behavior generated by the model relative to the population development until 2050 as well as to explain the major assumptions made for the years to come.
If we continue with in the simulation with the parameters set defined for the base run until 2050, what clearly appears is that the population in the model grows initially more than what is forecasted by the U.S. Census Bureau. However, after the year 2015, it falls dramatically beneath the forecasted value, reaching in 2050 a level lower than its initial level back in 1965 (fig.5).

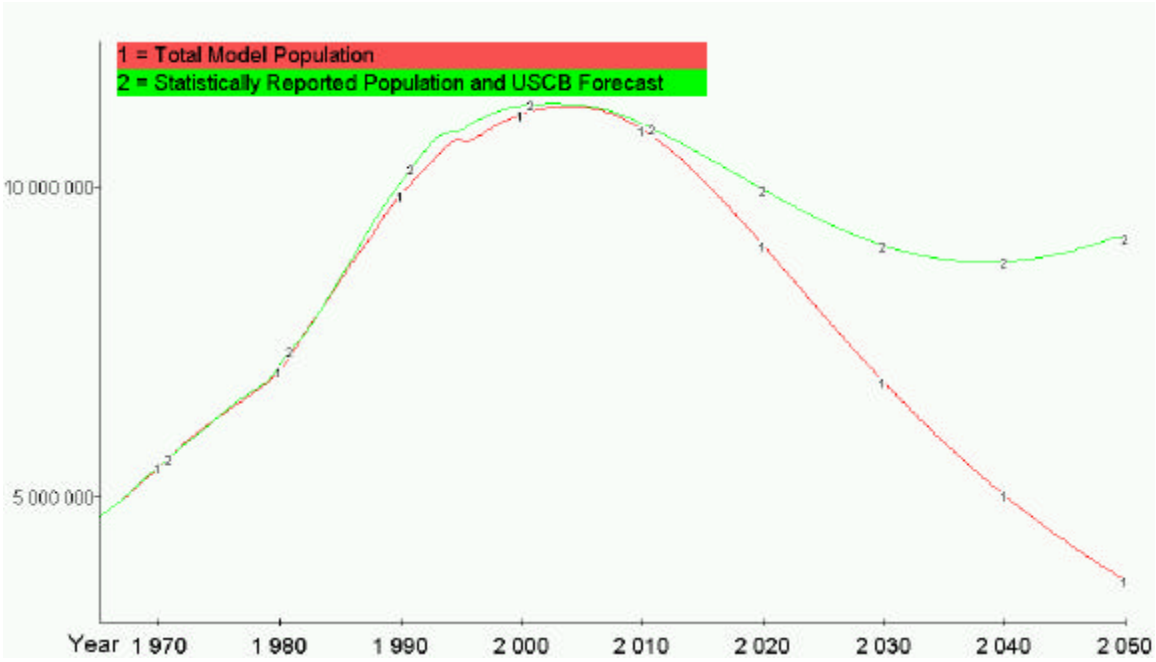

Fig.5

If we have a look on the development of the epidemic in figure 6, where the infection rate and the cumulative number of infections are reported, we can see that the infection rate reaches a peak in 2007, naturally corresponding to the inflection point in the cumulative function: the correspondence is highlighted by the black line (note that the two functions

are normalized in the graph: the higher scale refers to the infection rate, the lower one to its cumulative function).
We can also observe that the infection rate is not symmetric with respect to its peak, but after the peak it decreases very slowly, forming a long tail.
The peak of the infection represents the point of shifting in the dominance from the reinforcing "Contagion" loop to the balancing "Depletion" loop (fig. 1): from that moment the addition of more people in the infected population has a stronger effect in decreasing the probability of having an infecting contact than in increasing the number of contacts generated by infectious people.

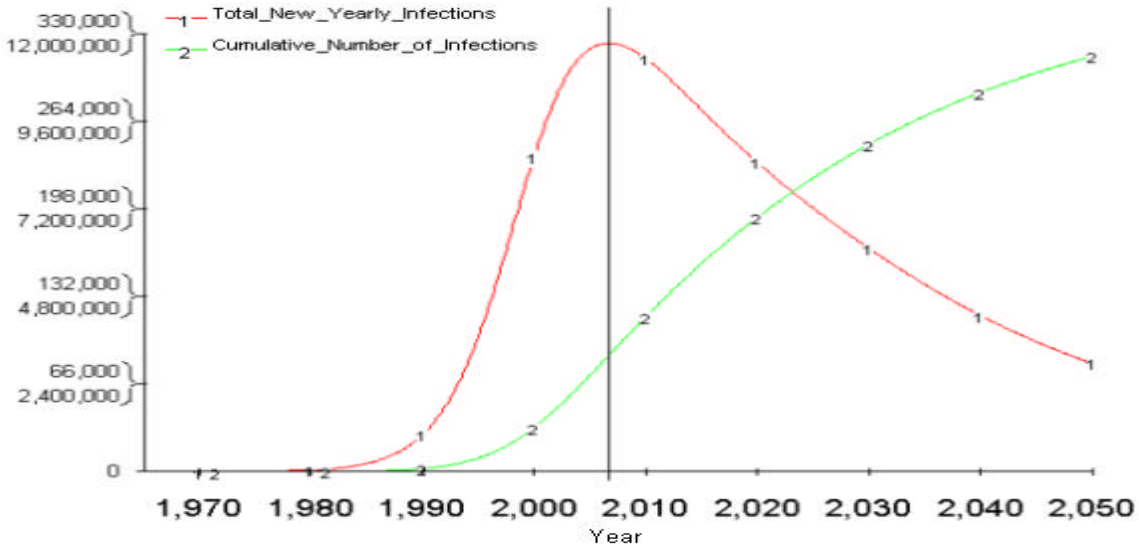

Fig.6

But why is this shift of dominance happening, and why does it happen in this moment?
To better understand the behavior of the HIV/AIDS population, figure 7 reports the infection rate, the adults HIV/AIDS population and the relative total death rate, comprehensive of deaths for AIDS and for other reasons.

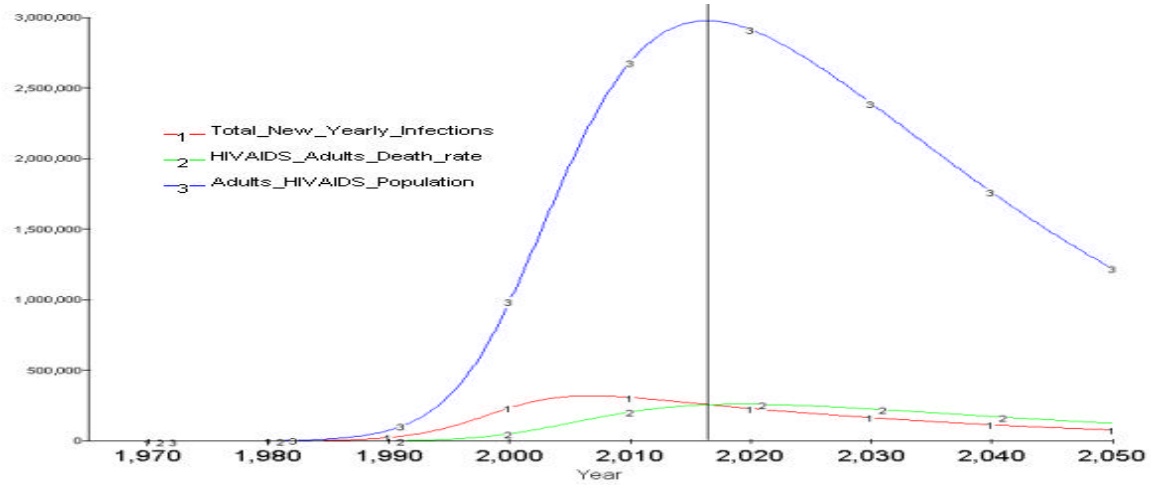

Fig.7

Intuitively, we know that for the epidemic to occur, the infection rate must be higher than the death rate relative to the HIV/AIDS population. If this condition were not respected, the few number of infectious people we introduced into the population would have died before infecting an equal number of people. Writing this condition in a very simplified mathematical form, without taking into account the distinction between different stages of the disease that are characterized by different infectivity and contact frequencies, we obtain:

$I*c*(S/N)*i>I/d$

Where "I" is the infectious population, "S" is the uninfected population, "N" the total population, "i" is the infectivity, "c" is the contact frequency and "d" is the average duration of the disease.

The left hand of the equation represents the infection rate, which is given by the infectious population multiplied by the contact frequency (that gives the number of contacts the infectious population generates), multiplied by the probability that a contact will be a contact with an uninfected person (S/N), and multiplied by the probability that such a contact would result in a new infection (i).

The right hand of the equation represents the death rate from the HIV/AIDS population stock, which is given by the level of the stock divided by the average duration of the disease.

If we bring the I from the left hand of the equation and take it to the right hand and we do the opposite with d, we obtain:

$(S/N)*c*i*d>1$

This indicates that for the epidemic to occur the multiplication of the factors in the left hand of the equation must be greater then 1: the higher the value of any of these factors, the higher the possibility for the epidemic to occur.

This equation is known in epidemiology as the *threshold theorem*, and in particular the c*i*d ratio is called *contact number* and the expression (S/N)*c*i*d is called *reproduction rate* for the epidemic.

Due to the fact that in the case of HIV, while "i" is relatively low, "d" is a very long period and "c", especially in the developing countries, is consistently high, we can see that, at the beginning of the epidemic, when (S/N) is very close to 1, the epidemic is extremely likely to occur.

Considering that the contact number (in this simulation) is constant in time, we can easily see that the major factor that will condition the epidemic development is (S/N), that is the probability that a contact generated by an infectious person would be with an uninfected one.

It is the dynamic of this ratio that will shift the dominance from the reinforcing "Contagion" loop to the balancing "Depletion" loop. In fact (S/N) will decrease with the growth of the infectious population (I) and the depletion of the uninfected one (S), causing the infection rate to reach a peak and then decrease. From that moment any increase in I will have a higher marginal effect in decreasing (S/N) than in increasing the number of contacts generated by the infected people. This is the moment when the shift of dominance from the reinforcing to the balancing loop happens. The reduction in the (S/N) ratio will also finally cause the reproduction rate to fall under 1. That is the moment when the infection rate in figure 7 crosses the death rate and therefore, as it is clearly highlighted by

the black vertical line, it is also the moment when the stock of HIV/AIDS adults reaches its maximum level.

Observing figure 8, we can see that the HIV/AIDS population will start decreasing only about ten years after the infection peak: this period of delay reflects also the fact the death rate does not increase instantaneously and proportionally to the HIV/AIDS population as we could deduce from the previous simplified formulation of the threshold theorem, because in the model the delay is formulated as a fifth order delay and not as a first order delay as in the indicated equation (note again that the values of the functions are normalized, the higher scale referring to the infection rate, the lower one to the HIV/AIDS population).

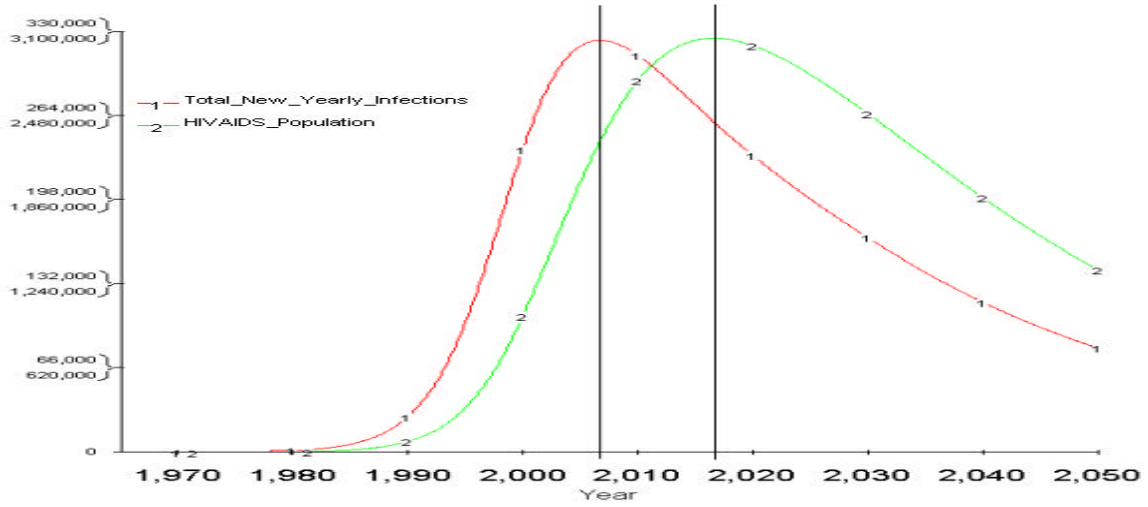

Fig.8

The total death rate relative to the HIV/AIDS population, reducing this stock that represents, when it reaches its peak, about one third of the entire population, greatly contributes to the erosion of the overall population, and causing its dramatic fall in the second quarter of this century.

The fall observed in the total population level, is also caused by the fact that, having the children from AIDS parents only a 70% of probability to survive for more than 3 years, the reproduction capacity of the population is seriously compromised in the moment the HIV/AIDS population starts representing a huge portion of the total population.

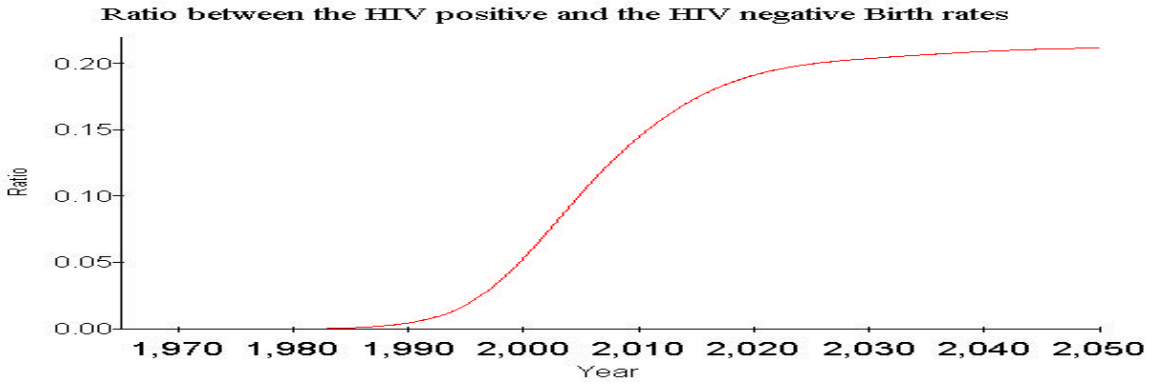

Fig.9

The relative importance of the part of births that is seropositive is shown in figure 9, where the ratio between the infectious children birth rate and uninfected children birth rate is reported. In the final part of the epidemic the infectious children birth rate is more than one sixth of the total, which implies a serious effect on the development of the population.
This decrease in the uninfected children birth rate also causes a decrease in the number of person that will enter the sexual active age, reducing the probability for infectious people to have contacts with uninfected ones, and closing a balancing loop that, with a consistent delay, will partially contribute to the reduction of the infection rate.

In the base run, we assumed no changes in the behavior of the subjects during the entire period of the simulation: this implies the assumption that either people will not be able to learn from the experiences of others and that no information about the disease will be available, or that this information will be not relevant in determining the subjects behavior. This assumption is clearly unrealistic, and as we can observe in many developing countries, governments and international organizations are making big efforts to diffuse information about the ways of contagion and its consequences. Even if the effect of these campaigns is extremely difficult to measure, evidence indicates that a reduction in the average number of dangerous contacts should be expected.
A particular weight to contribute to changes in people's sexual behavior should also be given to the experiences of AIDS lived by friends or parents of the subject, in the cases in which the cause of death is recognized as AIDS. Basing on these assumptions I decided to introduce in the model the effect of sensitization on contact frequency table function, which is calculated on the basis of the ratio between the actual population and the cumulative number of deaths for AIDS: figure 10 shows the shape of the function.

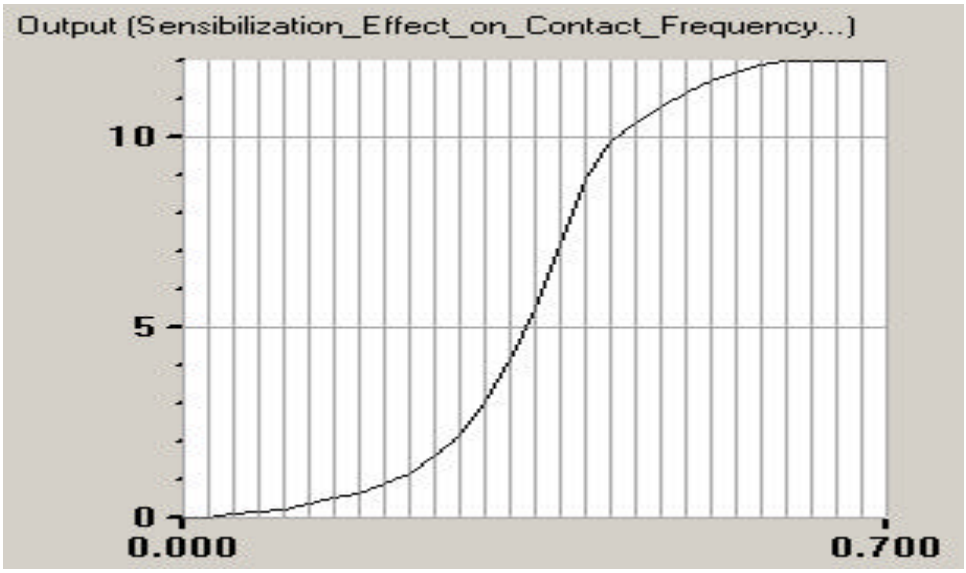

Fig.10

The input of the table function is the ratio between the actual population and the cumulative number of deaths due to AIDS and the output is a dimensionless value: to obtain the actual contact number, the normal contact number will be divided by the table function output plus one. The shape of this function reflects the idea that a higher number in the cumulative

deaths for AIDS, with respect to the population level, will cause the people to perceive a stronger risk of being infected, because of either some of their friends or familiars may be dead for AIDS or an increase in the effort made by the government to sensitize the people, what is reasonable to expect when the dimension of the problem increases. An increase in the number of cumulated AIDS deaths over the total population in the range between 3/10 and 5/10 will generate a particularly strong increase in the effect, while the flat head and tail of the function imply a reduced increase in the output with respect to the increase in the input at the beginning and at the end of the epidemic. This shape of the function, while not based on any scientific data, seems to be perfectly reasonable, considering natural that at the beginning of the epidemic, until the number of death is under a certain "threshold level", the perceived risk to be infected will be quite low and then, once reached this level, the information and the fear of the virus will start to spread within the population. Naturally the function cannot grow indefinitely: whatever will happen a part of the population will never completely change their sexual behavior and therefore the function saturates for a value of the input of 0.7. The scale of the input is also an estimation and implies a great reduction in number of dangerous contacts in the final part of the epidemic. For example when the equivalent of four tenths of the living population will be dead for AIDS, people will reduce of eleven times the yearly number of unprotected and potentially dangerous sexually contacts they would have otherwise had.

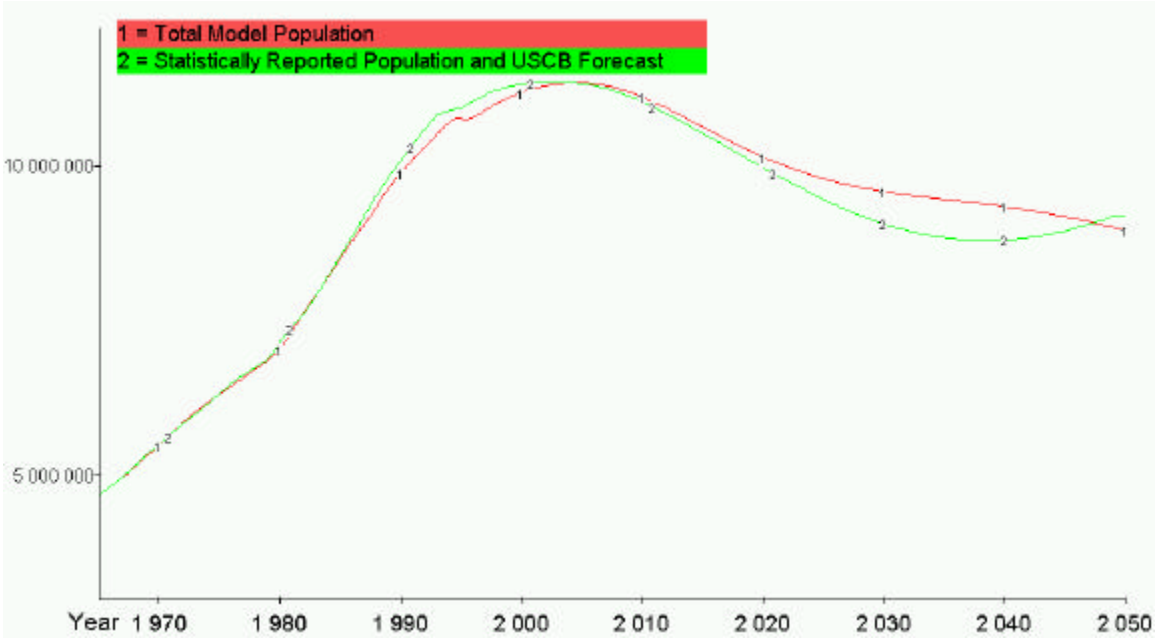

Fig.11

With the application of this effect, the development of the population, on the long-term, takes a complete different path, much more similar to the U.S. Census Bureau forecasts than previously, as shown in figure 11.

The reason of such a dramatic change with respect to the previous simulation results, is that thanks to the effect of sensitization on contact number we added to the model, we made the contact frequency, that is what in the threshold theorem equation is called "c", decreasing

with the increase of the number of HIV/AIDS deaths: this caused the epidemic to reach its peak earlier and to cause a lower number of deaths.
The magnitude and the characteristics of the table function inserted in the model are extremely subjective and perfectly contestable. However, independently from these factors, we can derive important considerations from the result of this simulation: for the population to stabilize on the level indicated by the U.S. Census Bureau in 2050, if we do not consider the introduction of any particular medical treatment, a major change in the sexual behavior of the people is necessary. Accordingly to the scale of the output of the table function, to obtain the result shown in figure 11, at the end of the epidemic the number of dangerous contacts generated will be reduced of thirteen times: this implies not a little adaptation, but a complete transformation of the sexual habitudes of the population.
Many other assumptions can be modified to obtain different results from the model, as the assumption that no one of the people infected decide to modify its sexual behavior because of an understanding of the danger they represents for other people or the assumption that the incubation times are constant. These changes will be tested in the policy evaluation part, as the effect of the introduction of sensitization campaigns or of the use of multi drug treatments.

The analysis of the age distribution of the population generated by the model and its evolution, with the same assumptions discussed above, can give a first idea of tremendous demographic dynamics that are taking place and will take place in the future of this nation.

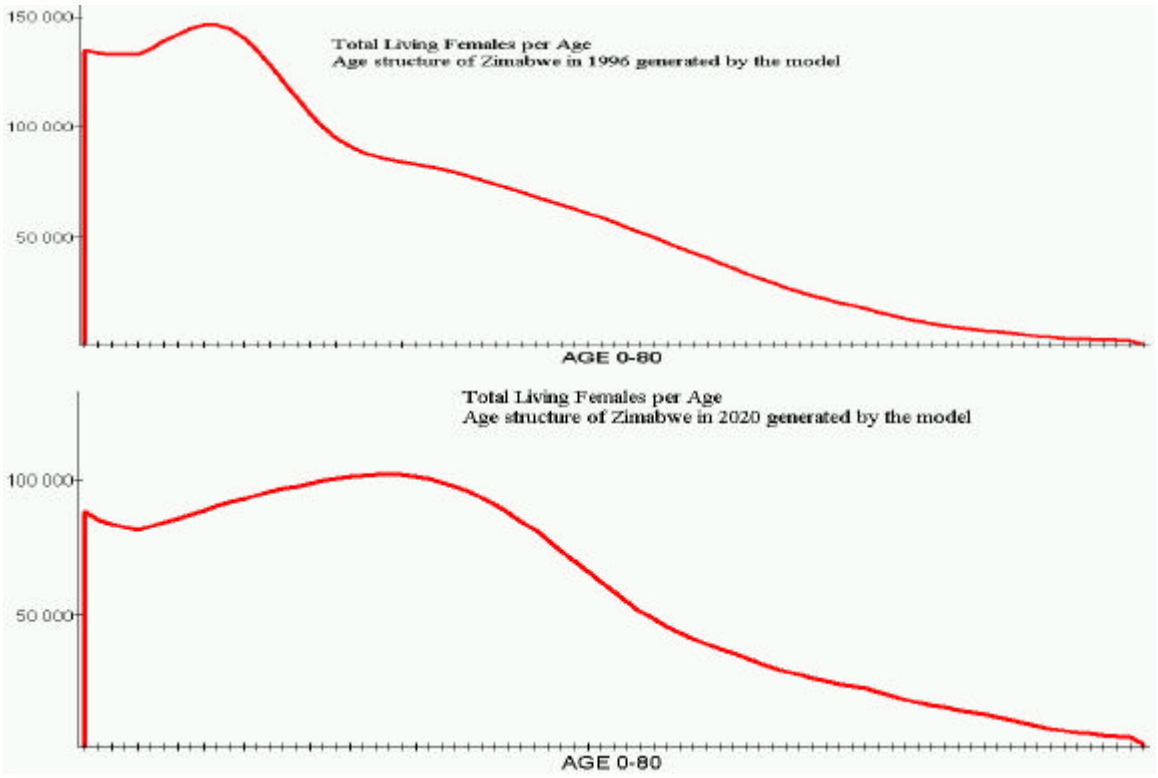

Fig.12

Above are reported two static pictures of the demographic structure of the population (particularly of the female population) in 1996 and in 2020 (fig.12), and to give an idea about the qualitative validity of the data generated by the model, these figures are compared with the age structure of Zimbabwe in 1996 and 2020 estimated and forecasted by the U.S. Census Bureau (fig.13).
The different results cannot be numerically compared, due to the fact that the population in the model is smoothly divided into 81 age classes, while the data from the USCB divide the population into only eight major age groups of different dimension. However, if we smooth the peak corresponding to the central age group in figure 13, which comprehend people from 20 to 44 tears old, we can easily observe a significant similitude between the age structures generated by the computer and those estimated and forecasted by the U.S. Census Bureau.
The "waves" in the age structure of the population are caused by the dramatic variations in the birth rate due to exogenous and endogenous effects, and by the flow of AIDS deaths, which drain people only from specific age groups.

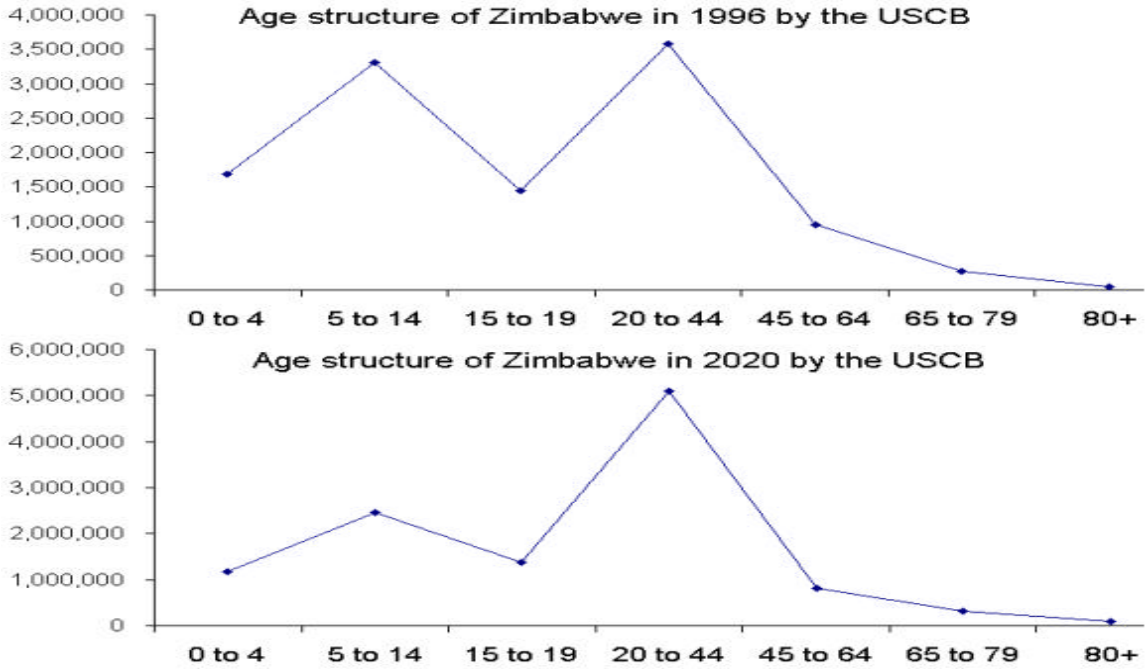

Fig.13

It is particularly interesting to observe the evolution of the demographic structure of the population dynamically during the simulation, to understand how the enormous waves of population, generated by different phenomena in different periods, move into the age structure before ultimately disappear, and to see how the population is depressed by the effect of the epidemic of AIDS, as snow that slowly melts under the rain.
To fully understand the effect of the HIV/AIDS population on the age structure of the population, in figure 14 the workforce ratio (the ratio between the workforce and the total population) development is reported for the same scenario previously analyzed and it is compared to what it would have probably been without the HIV/AIDS epidemic.

Particularly, it can be observed immediately how the effect of the epidemic on certain age groups can dangerously compromise the equilibrium of the population seriously eroding the workforce. From the beginning of the second millennium the gap in the workforce ratios generated with the two different scenarios becomes important: in this moment, in fact, the huge wave of people born in the early eighties start reaching the working age (considered from the age of 20 to the age of 60). In the case the epidemic would not have appeared, this wave would have had a full impact on the workforce, which would have exponentially increased until 2010 to eventually reach a plateau of about 62% in 2040.
In reality the effect of the wave of people born in the eighties on the increase of the workforce has been only partial, due to the strong effect of the epidemic. We can observe the full effect of the epidemic in this sense looking at the behavior generated by the model for the second and the third decades of the new millennium: the workforce grows slowly and accelerate only after the year 2020, due to the correspondent reduction in the birth rates and in the population over 60 years old.

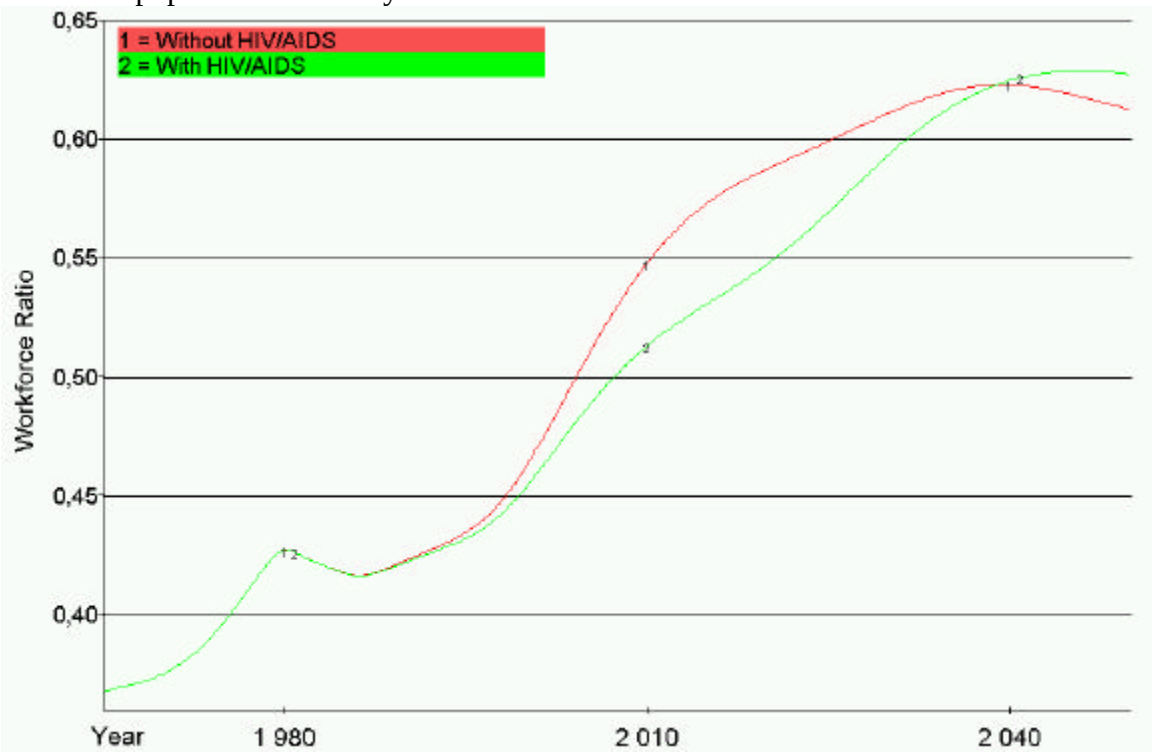

Fig.14

The workforce erosion is even more evident if we look at it in absolute terms: given this scenario, the model generates a loss of people in the working age of about two millions units. Such slowing down in the growth of the workforce for a developing country in very delicate economic situation, as Zimbabwe actually is, can have serious repercussions on the national social and economic system. The labor force is still the major economic resource for many developing countries, which usually base their economic development, in these phases of rapid change, on the price gain they can have with respect to the developed countries in the production of basic goods, due to the abundance and the low cost of the labor. Therefore, the slow growth in the workforce generated by the model can introduce

important delays in the socio-economic development of the country. To understand the full impact of the disease in this sense we also have to consider that the epidemic has a strong effect in reducing the whole population, as discussed before, and therefore in reducing the National demand.

Policy analysis

In the model proposed in this work, many are the possible policies that could be tested: representing the model the main forces driving the epidemic development, and being clearly explicated the mechanism of contagion and the stages the subjects go through during the course of the illness, many are the parameters that can be modified to reflect the implementation of a particular policy, and the model will show a different sensitivity to each of them.

This policy analysis is concentrated only on three particular policies and I will confront the resulting behavior generated by the model in the three cases, to draw some general conclusions.

The first policy I will test refers to a decision taken in the summit of the World Trade Organization (WTO) that took place in Doha (Qatar) in the month of November 2001. Many newspapers reported as the most important agreement resulting from the meeting the decision of giving the possibility to the developing countries to buy or produce pharmaceutical products for the HAART treatment at a very low cost, without paying any royalties to the pharmaceutical firms who holds the patent for such drugs.

This will be probably the first step in the process of introduction of the HAART treatment in the developing countries: what will be the effect on the population and on the development of the epidemic of such a policy?

The main direct effect on an infectious person of the HAART treatment is a consistent increase in the expected life, the length of which depends on many factors, as for instance the moment in which the subject starts the treatment or the continuity in the assumption of the antiviral drugs. While the first introduction of the HAART treatment has been only in 1995, from the observation of the slow development of the virus in the subject under treatment, it has been estimated that thanks to these antiviral drugs the overall incubation time of HIV can result even doubled. Many experts also argue that the treatment can have the additional effect of reducing the infectivity, because of the reduction in the viral load observed in many patients: however this hypothesis is still not proven and will not be considered in this study.

To test this policy the model's parameters have been set as in the base case, with a normal "effect of sensitization on contact frequency" as the one utilized to obtain the behavior in figure 11, and the average time spent by individuals in each HIV/AIDS stage has been increased of a value of 50%, from the beginning of the year 2002. I decided to use this value on the basis of the assumption that not every infectious people will have access to the treatment and that a part of those who will make use of the antiviral drugs will not be constant in following the prescriptions and therefore will benefice only of a part of the effect of increase in the expected life. However the sensitivity of the model to the choice of this parameter is only numerical, and there are absolutely no behavior mode or policy sensitivity. The development of the population in the case of the HAART treatment introduction in the year 2002 is shown in figure 15. This figure should be compared with

figure 11, being the set of parameters identical to that case with the exception of the incubation times. We can observe that the population grows after the year 2002 more than in the base run in figure 11, but after the year 2020, it tends to decline more rapidly and it finally falls on a level lower than the one it would have reached without the introduction of the treatment.

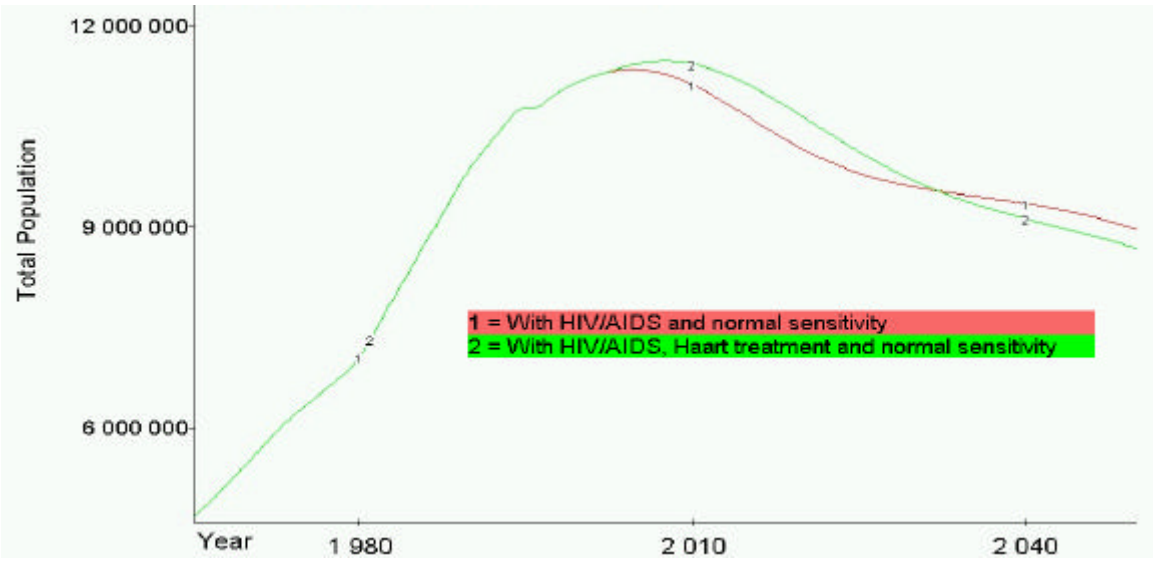

Fig.15

Why the introduction of a treatment that should help the people living with AIDS has such a negative effect on the population development on the long run?

In figure 16 we can see the HIV/AIDS adults stock development as well as the inflow (the infection rate) and outflow (the death rate) to this stock in the normal case of no introduction of any treatment, corresponding to the population development illustrated in figure 11. We can observe that the data series relative to the infection rate and to the death rate are smooth, and they cross at the beginning of the second decade of the century, marking the maximum for the HIV/AIDS population stock. This stock reaches a maximum level around 1.9 millions, before rapidly declining, drained by the death rate that, with a certain delay, reflects the behavior of the infectious population.

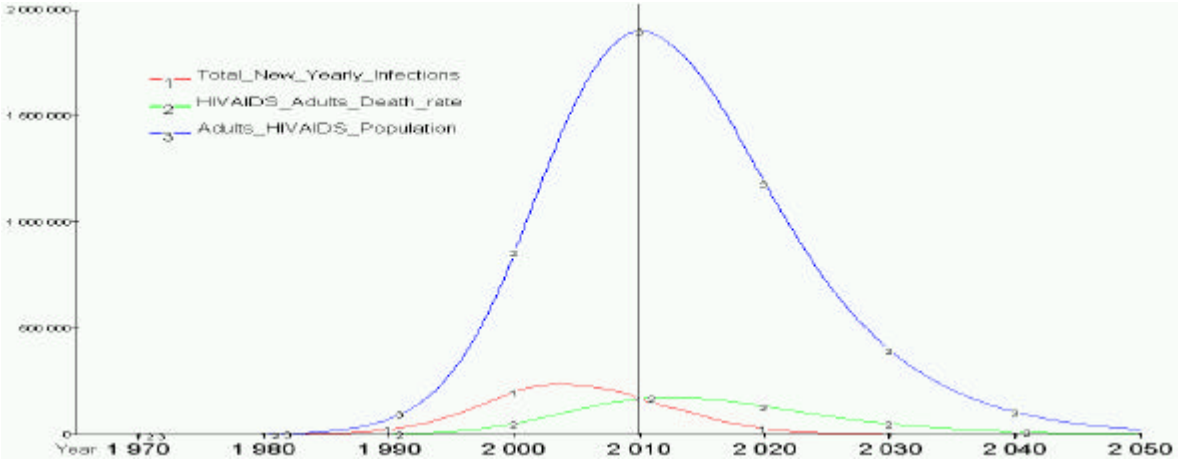

Fig.16

Comparing the previous graph with the one reported in figure 17, we can immediately observe some important differences, which allow us to better understand the reasons of the negative impact of the HAART treatment on the population development on the long-term.
We can observe, in fact, that right after the introduction of the antiviral drugs treatment, the death rate falls, as we would expect. This causes HIV/AIDS population to increase faster than normal, owing to the fact that the death rate drains a lower number of subjects from this stock. This increase in the HIV/AIDS population is accompanied by a parallel increase in the infection rate, due to the fact that a higher number of infectious people will now generate a higher number of contacts and will eventually infect a higher number of persons. This will increase even more the infectious population stock that will reach its maximum level only in the middle of the second decade of the century, at a value of about 2.5 millions.

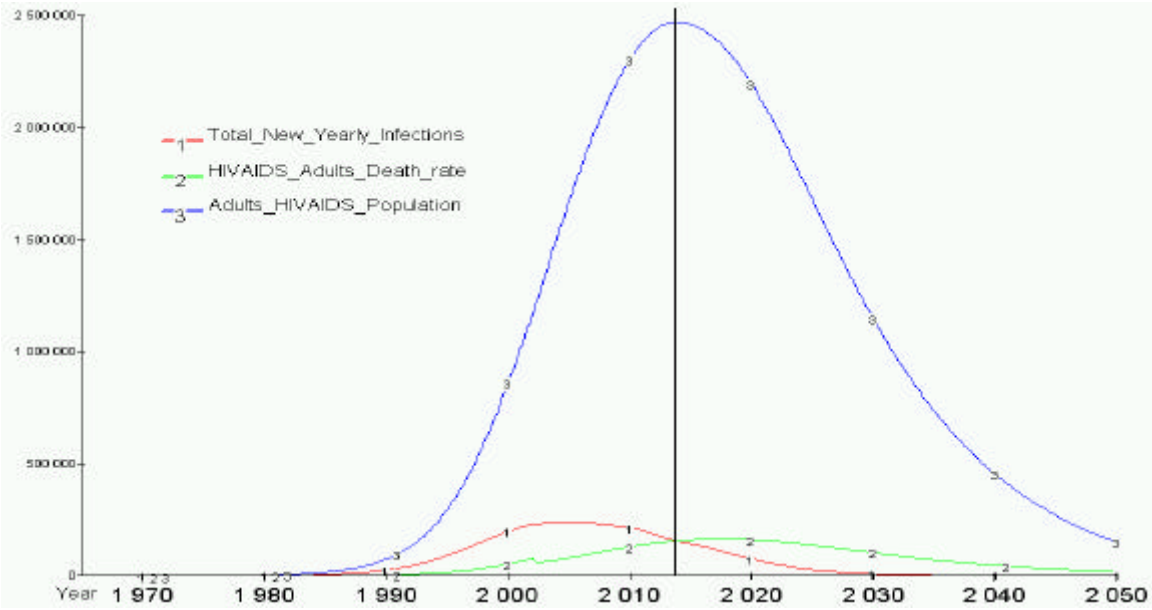

Fig.17

However, after a certain delay, the death rate will reflect the enormous growth in the HIV/AIDS population and will start draining out people from this stock, reducing considerably the total population.
Note that the difference in the maximum level of the HIV/AIDS stock in the two cases is really consistent, with more than half a million people. We can conduce the same policy test with any value for the increase of the incubation time due to the introduction of the HAART treatment, and the result would not qualitatively change.
On the other side, it is also very interesting to observe what happens in the age structure of the population, once this kind of treatment is introduced. Figure 18 shows the behavior of the workforce ratio with the same scenario representing the introduction of the HAART treatment.

Clearly the introduction of the treatment initially is able to close a part of the gap between "With" and "Without HIV/AIDS" workforce ratio curves. By increasing the average life of the infectious subjects, the antiviral drugs consent to the HIV population to be for a longer period part of the labor force, before shifting to AIDS and ultimately die. However, on the long term, the introduction of the treatment, increasing the infectious population (as shown in figure 17) cause the workforce ratio to grow more slowly and to ultimately reach a similar equilibrium level than the one it would have reached without any treatment, but with a consistent delay.

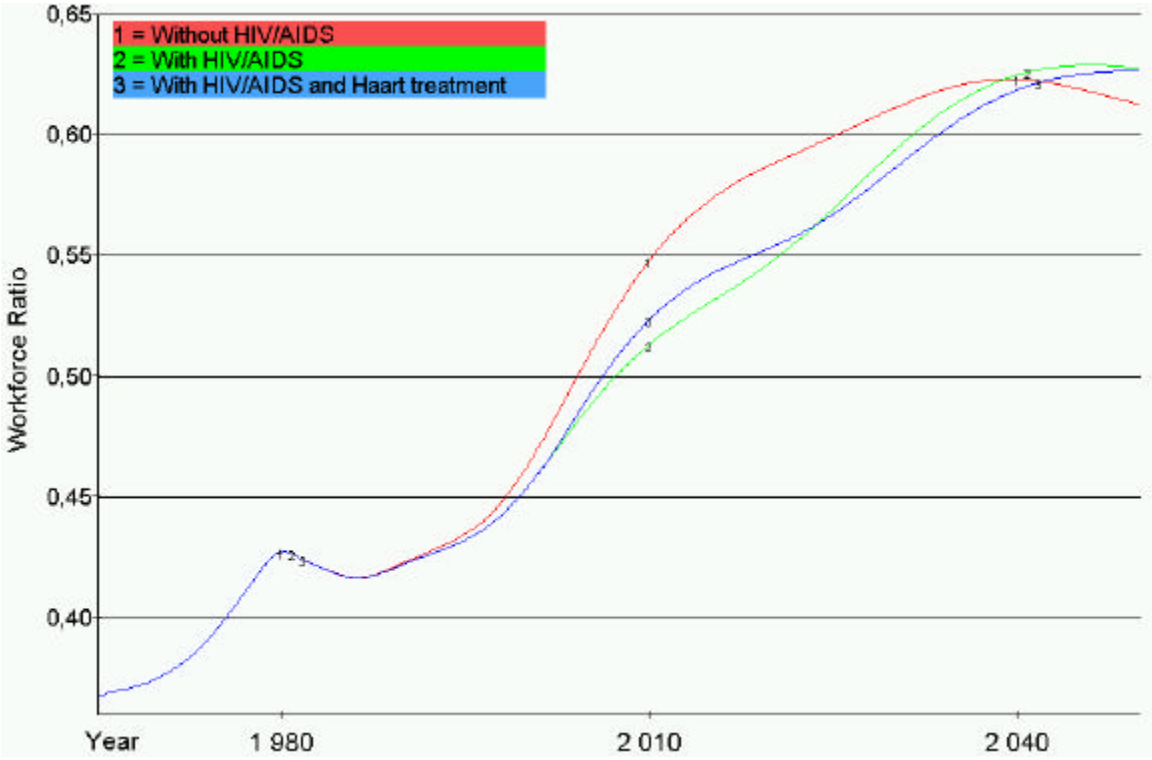

Fig.18

On a short-term horizon, we can observe how the introduction of the HAART treatment has a benefic effect on the population as a whole and how it can partially enhance the workforce ratio with respect to the level it otherwise would have been.

On the contrary, on a long-term horizon, the introduction of the antiviral drugs seems to have a negative effect, delaying the peak of the epidemic, causing a drop in the population and particularly in the workforce.

Without going into a political discussion, we can simply conclude that these results raise some doubts on the opportunity of this kind of intervention as well as they show some major risks in the implementation of such a policy.

However, independently from the long-term effects of this policy, the pressure from people on governments to have an immediate benefit and to give a more normal life to already infected people will make almost impossible for policy makers to refuse the introduction of the HAART treatment in the developing countries.

That is why the second policy that will be tested here is not considered as an alternative to the previous one, but as a measure to try to reduce the negative effects on the population development of the antiviral drugs introduction.
A first observation comes from the fact that a subject must be aware of his clinical condition to start the HAART treatment. In order to do so he/she must be subjected to specific medical tests: this implies that it would be possible, in theory, for the medical personnel to identify these people. Therefore it should be possible to enroll them into sensitization programs, as long as they are under treatment. This should reduce the number of unprotected contacts these people generate and therefore reduce the impact of the longer time they spent in the HIV/AIDS stages. Contemporarily the government should introduce general information campaigns, to reduce the contact frequency of the whole population.
If together with the introduction of the HAART treatment we assume a reduction in the base contact frequency of people in the HIV stages two and three (it is almost impossible to get to know about being infectious in the first months after the infection) from 19.25 to 15 and if we assume from 2002 an effect of sensitization on contact frequency a 50% higher than normal, we will obtain the behavior described in figure 19.

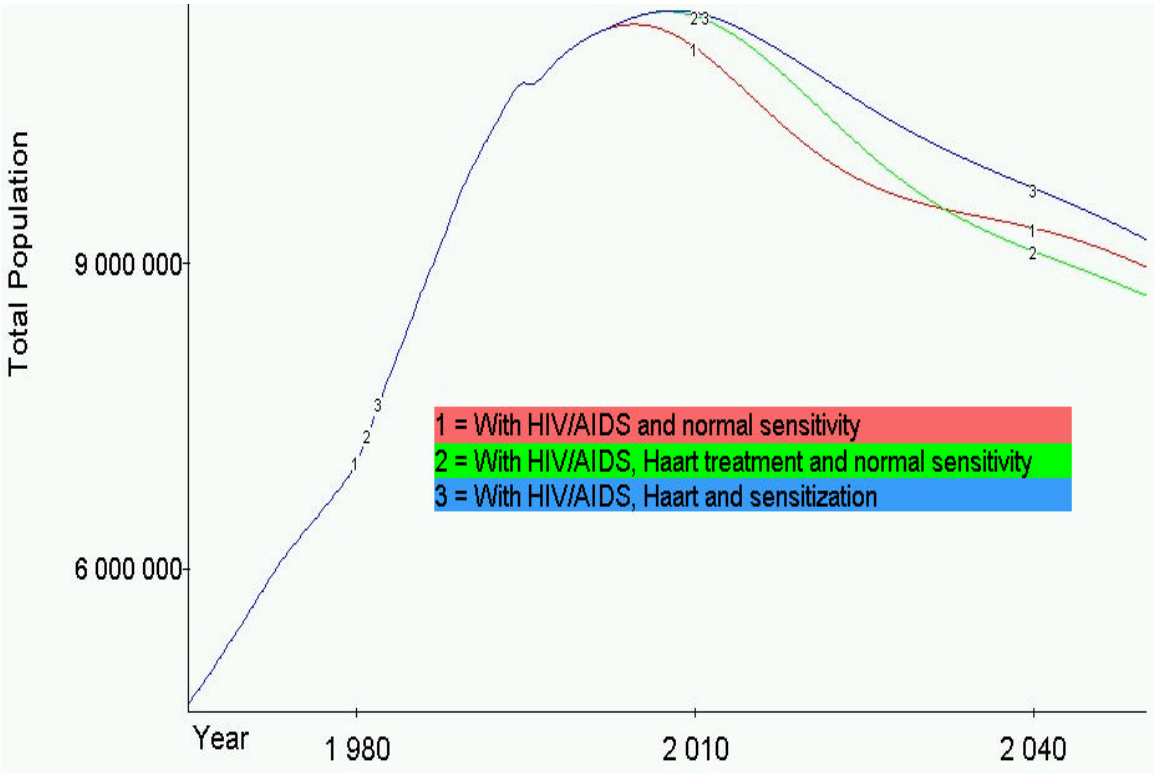

Fig.19

The reductions in the contact frequencies of the different categories of people can partially absorb the negative effect of the introduction of the HAART treatment and, as figure 19 shows, the population will tend to be constantly on a higher level than before the introduction of this second policy.

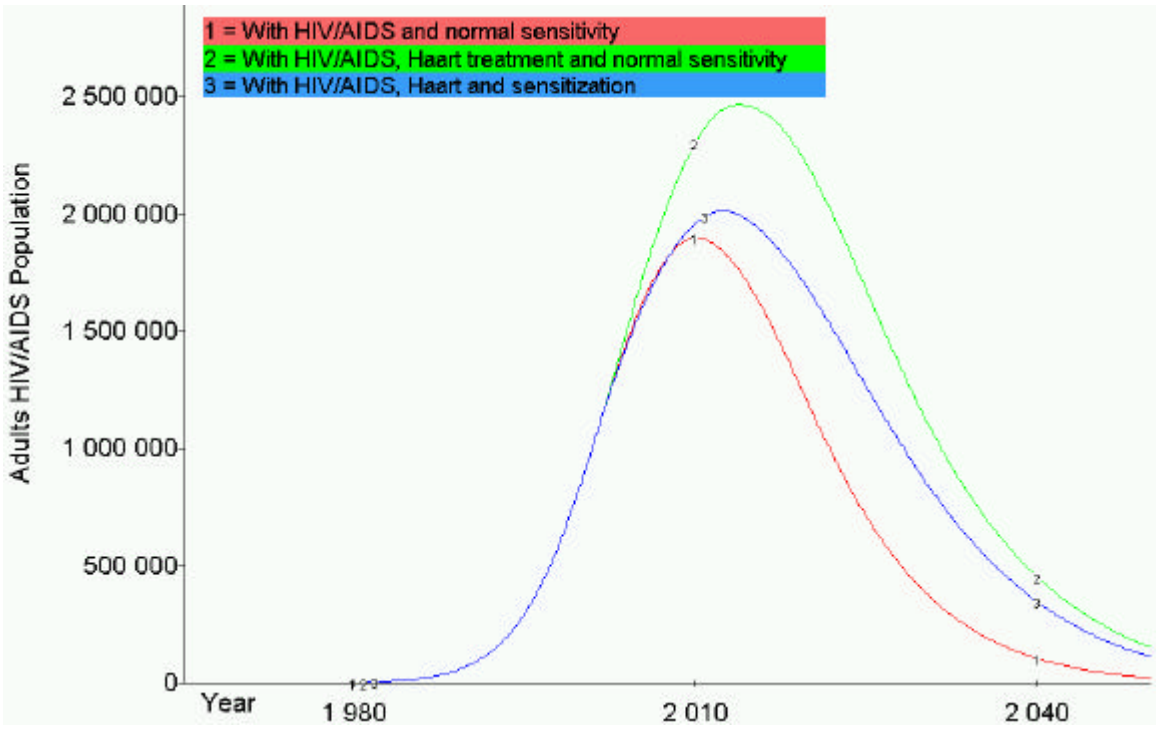

Fig.20

In figure 20 is reported the Adults HIV/AIDS population for the three cases of no intervention (case 1), HAART treatment (case 2) and HAART treatment joined to a sensitization action (case 3). As it clearly appears from the graph, at the moment of the introduction of the different policies, while the infectious population in case 2 grows very fast with respect of case 1, in case 3 is not shown such an extreme behavior and it finally reaches the level of only 2 millions.

The same kind of benefic effect is also observed on the workforce ratio and on the absolute level of labor force, as it is shown in figure 21 and 22.

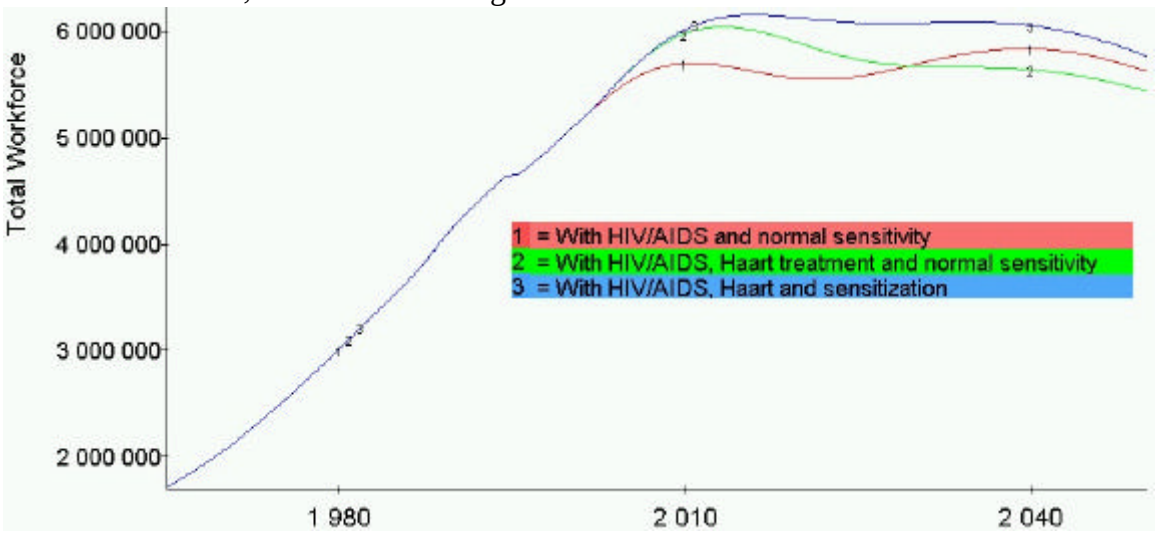

Fig.21

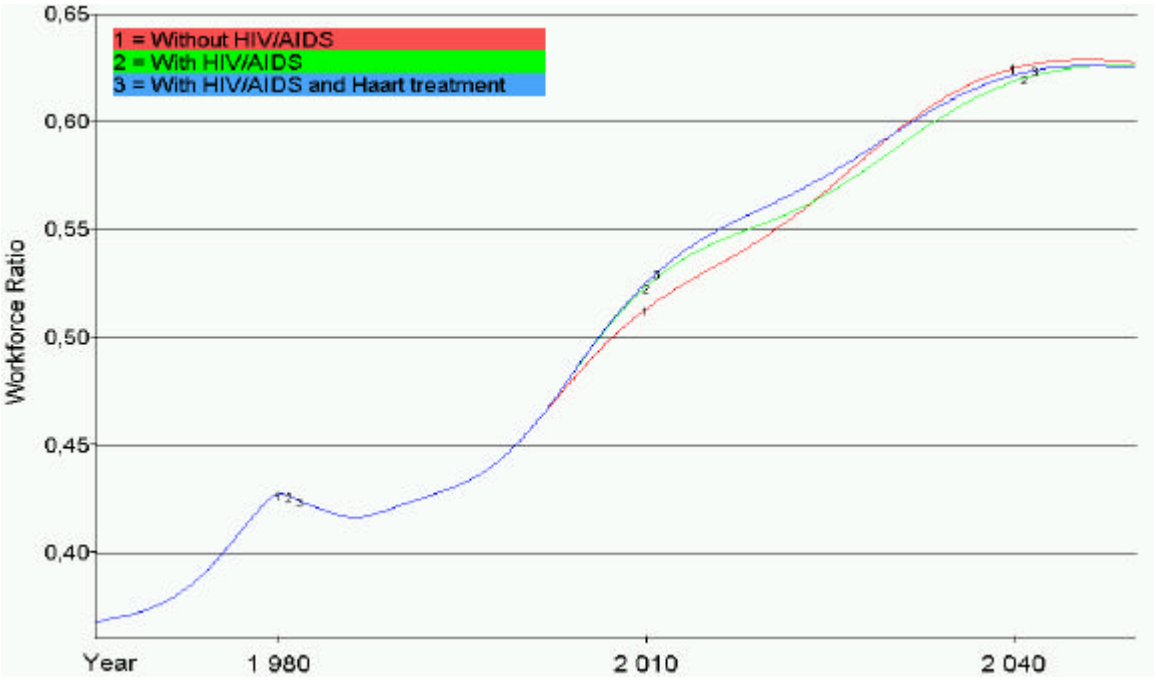

Fig.22

Therefore, this policy seems to be able to act in the sense of constraining the negative effect of the introduction of the HAART treatment on the population development. Although, if we consider the huge reduction in the contact frequency we assumed to obtain this result, we can understand that the effort required from the government to implement such a policy is very high and that probably not all the developing countries would be able to sustain it. In fact, this policy assumes an immediate reduction of about 25% in the contact frequency for people in the second and third HIV stages, in addition to an increase of 50% in the overall sensitivity of the population to this problem: these are not results that can be obtained immediately (as we did in the model) and, in some cases, even in the long-term it will be particularly difficult to achieve such a goal.
Anyway, the effect of this kind of policy is surely positive for the limitation of the HIV diffusion, therefore an effort in this sense, independently from the magnitude of the intervention, could surely limit the effect of the epidemic.

The third policy that will be tested concerns the introduction of the Viramune/Nevirapina, a drug particularly efficient in reducing the risk of virus transmission from mother to child. The children of HIV positive mothers can be HIV positive or not depending on several factors, as hygienic conditions during the delivery of the kid or as the duration of the breast-feeding period, among others. In most of cases this is strongly depending on the quality of the medical assistance, before and after the delivery. The overall probability of transmission of the virus from mother to child is represented by the perinatal transmission rate. The reference value for this parameter has been estimated to be 30%, consistently with the average hygiene conditions of the nativities in the country.

To test the effect of the introduction of a Viramune/Nevirapina treatment on pregnant women, I assumed a reduction from the year 2002 of the perinatal transmission rate of 50%. In reality, if well conducted, this treatment can reduce much more than 50% the transmission rate on a single woman, but here a reduction of 50% has been considered assuming that only a part of the pregnant women can afford this treatment and that not all of them will follow it perfectly.
For this new simulation only the perinatal transmission rate parameter has been modified, while the rest of the parameters setting corresponds to the one used to represent the implementation of the HAART treatment joined with a sensitization action.
Figure 23 shows the immediate results of the introduction of such a treatment on the pregnant women.

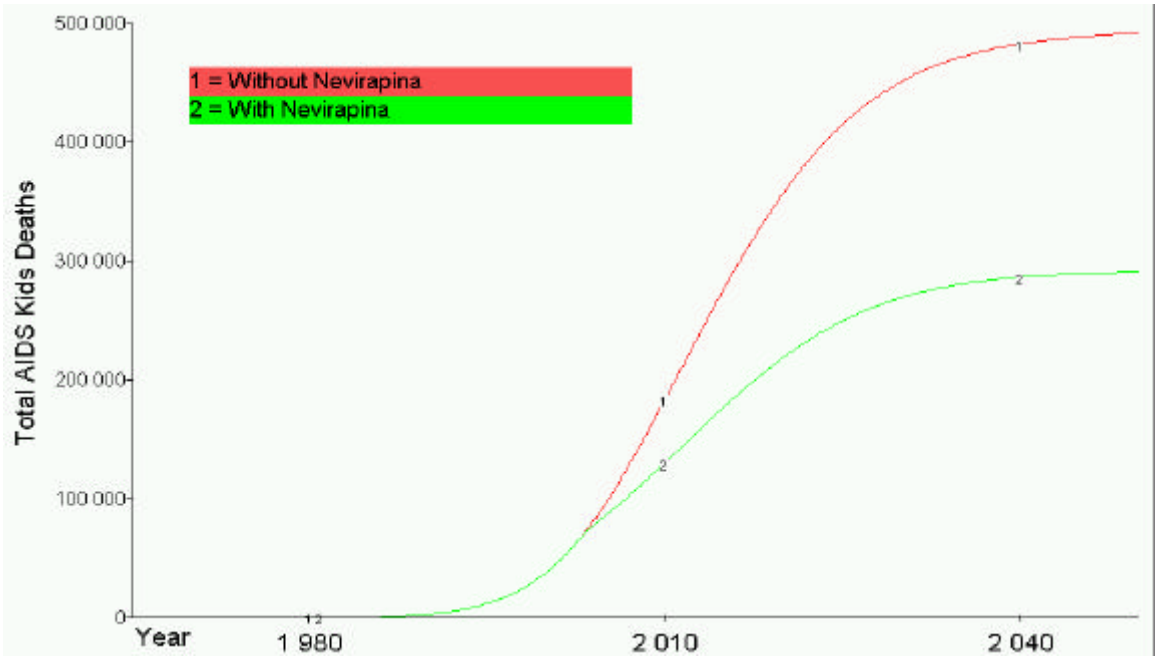

Fig.23

As it clearly appears, the implementation of this kind of policy, strongly reducing the number of children born with AIDS, has a major effect in reducing the number of death due to AIDS in this age class. The magnitude of the effect is particularly relevant and the simulated number of children dead for AIDS in 2050 is in this case about half of what is shown without this treatment.
The results on the whole population of the implementation of this policy are reported in figure 24. The effect observed is quite small: the behavior pattern of the population development does not change and its absolute value is constantly higher than what was previously observed, but only of about one hundred thousand units. Naturally, the effect of this policy on the workforce is even smaller and the results of the simulation are almost identical of those obtained without the introduction of the Nevirapina treatment.

This happens for two main reasons. Firstly the AIDS children population represents only a low percentage of the whole and a great reduction in it has a small direct relative effect on the total population. Secondly, the increase in the flow of non-infectious births caused by the introduction of the treatment, with a certain delay, will have the effect of increasing the stock of non-infectious adults, alimenting the hotbed of the epidemic.

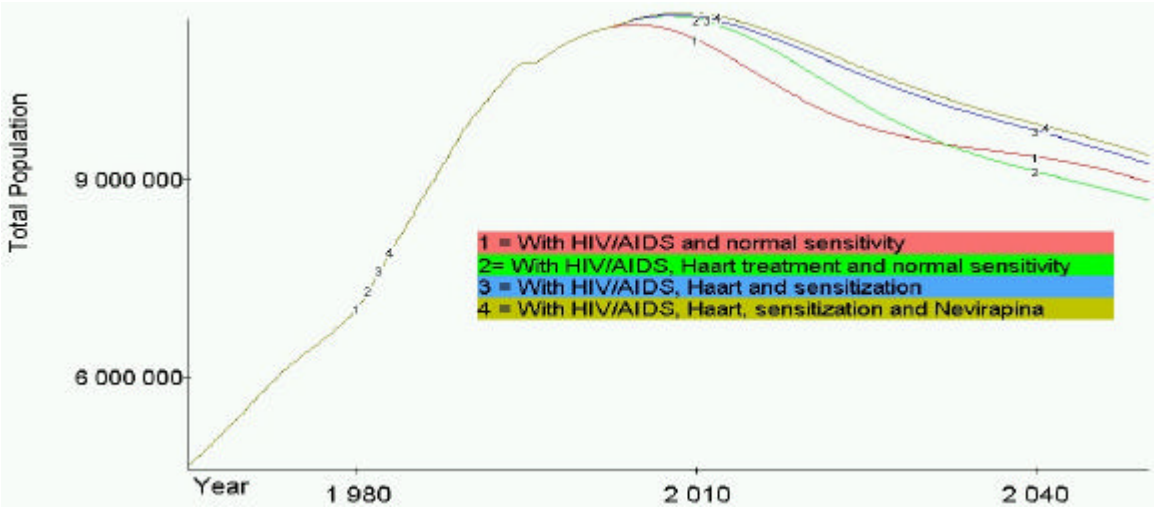

Fig.24

This will eventually act in the sense of partially balancing the positive effect of the policy implemented. In fact, a correspondent increase in the adults HIV/AIDS population is shown in figure 25.

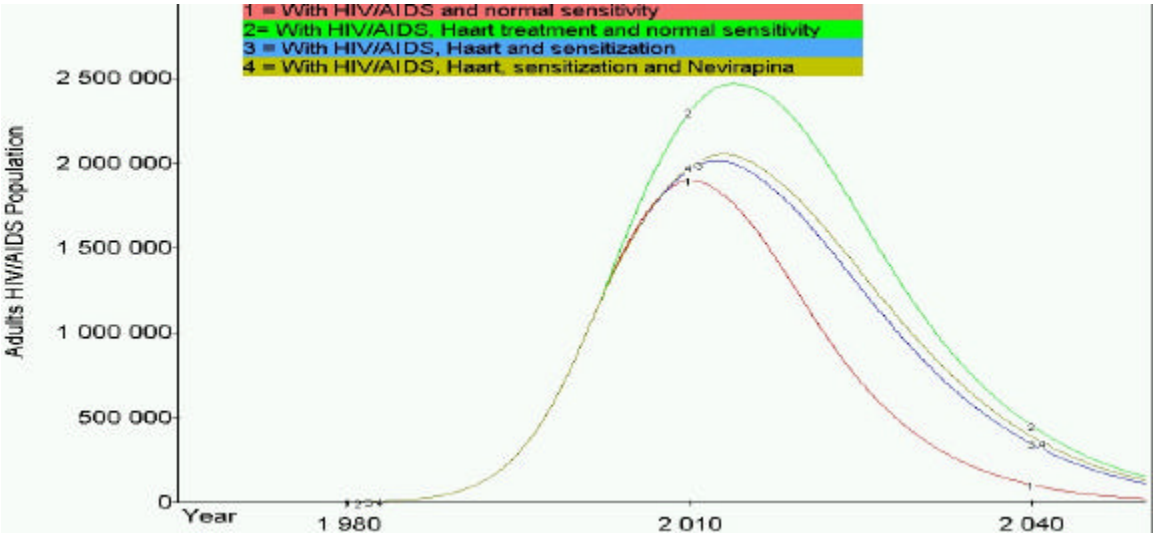

Fig.25

However, even if on the population as a whole and on the labor force this policy seems to have only a very small effect, this effect seems to work in the right direction. If we also consider the relatively low cost of the introduction of this treatment with respect to the potential cost of the implementation of the policies analyzed before, and the enormous benefit it generates on the children of infectious mothers, we can conclude that this specific policy should be taken into consideration.

Conclusions

From the analysis conducted during this work, I can draw three major conclusions.

A *first conclusion* is related to the results of the long-term analysis of the impact of the disease on the Zimbabwean population. From the many simulations conducted we obtained frightening results, highlighting a situation that should not be considered any more under control and that is not likely to change in the next decade.

It is extremely difficult to evaluate all the effects of this epidemic on the long term from the social and economic point of view. Although, simply looking at what could be the direct effect on the population development of the diffusion of HIV, it is clear that this phenomenon is going to change the history of the country.

Even in what it seems to me an extremely positive scenario, assuming that people consistently reduce to a level thirteen times lower than the actual level the number of unprotected sexual contacts, we should expect four to five millions of AIDS victims in this first half of the century.

The subtraction of this enormous amount of people from the more productive age groups, will have dramatic effects on the domestic product, therefore also on the life conditions of the youth and the elderly. This should reduce the population even more, compromising the possibility of the nation to go towards a sustainable development.

A *second conclusion* I can draw refers to the analysis of the three policies more likely to be implemented in the next few years in Zimbabwe, as in many other developing countries. On the side of the possible introduction of the HAART treatment, it seems to me that policymakers will be challenged with a difficult trade-off between a short-term benefit and a long-term one in terms of human lives loss. However, independently from the possible long-term effects of this policy, the pressure from people on governments, to have an immediate benefit, will make almost impossible for policy makers to refuse the introduction of the HAART treatment in the developing countries. To limit the long-run effects of the introduction of the treatment, a sensitization and information campaign should be introduced. This, independently from the magnitude of the intervention, could surely reduce the effect of the epidemic. Finally, to these first two kinds of interventions, a Nevirapina treatment can be usefully added, in consideration of the relative low cost of the implementation of this policy and the great benefit that can derive from it for the target children population.

The *last conclusion* is related to the necessity, raised during this work, to integrate the actual model in a wider socio-economic model. In fact, the model here presented is able to consider only the direct effect of the epidemic on the population and therefore on the workforce, which will eventually affect the development of the local economy. On the other side, the economic situation of the country will certainly affect the development of the population and the possibilities of the government to implement policies to fight the Virus diffusion, all aspects that the current model is not designed to take into consideration. The possibility to “close” these two powerful reinforcing loops could give new and deep insights into all the main aspects of the impact of the HIV/AIDS epidemic on the population of Zimbabwe and of many other developing countries.

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Most of the data utilized for the model's parameters setting has been taken from the UNAIDS, WHO, U.S. Census Bureau IDB and FAO. This data can be consulted in the following web sites:
www.unaids.org
www.who.int
http://apps.fao.org/debut.htm
http://www.census.gov/ipc/www/idbprint.html

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