The Structure of an Epidemic:

Modeling AIDS Transmission in Southern Africa

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The HIV/AIDS epidemic has reached prevalence levels in the order of 20-30 percent, maybe more among the general adult populations of several nations in Eastern and Southern Africa. While such prevalence levels imply a tremendous morbidity and mortality burden that clearly has demographic and social consequences, the contours of the demographic and social impact of the epidemic in the region are still not precisely established. This is due in part to the fact that demographic data for the region was already comparatively limited before the onset of the epidemic. In trying to quantitatively assess the impact of the epidemic, demographers have thus been constrained to study the most direct effect (i.e., of infection on mortality) with aggregate, non-parametric models designed to reproduce some of the observed empirical regularities of the epidemic and place minimum demands on data.

Micro-simulations models, on the contrary, can represent many of the social mechanisms that contribute to the spread of HIV or that are affected by it. The value of the quantitative assessments that can be derived from these models is limited by data requirements that far exceed what is currently available in much of Eastern and Southern Africa. These models are qualitatively more informative as they can illustrate some of the possible consequences of the epidemic, and through sensitivity analyses, they can also enable investigators to prioritize data needs as regards some of the determinants of the spread of the epidemic. Agent-based models are a new class of micro-simulation models, in which the model units (or agents) behave according to some basic rules that may depend on the state of the system (population of agents) at that time. They are thus particularly well suited to the study of the study of interactions between agents and the dynamics of macro-level norms or phenomena and micro-level rules of behavior.

Because the spread of the HIV epidemic depends on sexual interactions between people, the social structure in which individuals meet and the social norms that constrain sexual

behavior, agent-based models appear to us a promising avenue for studying the mechanisms that contribute to the spread of HIV and the multiple social impacts of the epidemic at high prevalence levels. In this paper, we describe our first steps in attempting to develop an agent-based model of HIV and population dynamics. Ours can be described as a mixed strategy between the "black box" approach of aggregate models and the "bottom up" approach of artificial societies. We start from a basic micro-simulation model based on aggregate distributions (e.g., age distribution at first marriage) that we gradually replace by behavioral rules (e.g., rules for partner search and marital decisions). This work is very much in progress and we are still at this time far from producing new knowledge on the future of the epidemic. What we are currently focusing on instead is the process of "docking" our basic model with extant knowledge about the epidemic, that is, making sure that it reproduces what is currently known about it, and on a gradual substitution of its parameters by the behaviors that are implicitly involved in the value of this parameter and that will become visible and susceptible to change.

Background

The Demographic Impact of an HIV Epidemic

The mortality consequences of an HIV epidemic were recognized first. As an untreated HIV-infection leads to death about ten years later (Morgan et al. 2002) and as most HIV-infected individuals are young adults that would otherwise be expected to live much longer (Heuveline 2003), an HIV-epidemic clearly has the potential to reduce life expectancy and to increase the population's death rate. To provide an order of magnitude, one can imagine that the number of new infections remains constant over time and that the survival time from infection to death is uniformly ten years. In that hypothetical case, the annual number of deaths becomes equal to the annual number of new cases (incidence), whereas prevalence is ten times larger than annual

incidence. While overly simplistic, this scenario provides a rule of thumb for the order of magnitude of the increase in the annual death rate that the HIV epidemic might induce: about one-tenth of the all-age prevalence level.

In the mid-1980s, it was then recognized that some HIV-epidemics in sub-Saharan Africa were of a higher magnitude than in any other part of the world, and could be reaching prevalence levels of 10, 20, or maybe even 30 percent.[1] As suggested above, such levels might translate in additional mortality in the order of up to 3 percent per year, which was about the annual growth rate of these populations. Some early models were indeed predicting population decline in sub-Saharan African (Anderson et al. 1991). Demographers then became involved in trying to assess the demographic impact of severe HIV-epidemic on death rates and rates of population growth (e.g., United Nations 1991).

The first demographic projections to explicitly incorporate the demographic impact of AIDS in actual populations (United Nations 1993) provided a slightly less alarmist picture. More specifically, while confirming the huge impact that these HIV epidemics might have on mortality, demographic simulations suggested that the impact on the population growth rate of the worst-affected populations was unlikely to lead to population decline. This surprising finding was largely the result of taking into account the age structure of the population, and the age patterns of incidence and fertility rates. To illustrate this with another simplistic example, imagine that a woman who becomes infected shortly after marriage continues to bear children at the high marital fertility rates that were then common in sub-Saharan Africa during the eight or so asymptomatic years that follow her infection. At these rates, we can expect her to bear three

[1] Higher *adult* prevalence levels have been reported, but while under-five prevalence can be as high or higher than adult prevalence, the overall prevalence under age 15 is generally lower than the adult prevalence and the all-age prevalence lower than the adult prevalence.

children on average, and with one-third a reasonable value for the mother-to-child HIV-transmission rate (The European Collaborative Study 1999; Gibb and Tess 1999; Leroy et al. 2001), we might assume that one of her children will die young, but that the other two might escape other causes of child mortality and survive to adulthood. It is counterintuitive but in fact possible that every adult woman becomes infected and that the population still replaces itself. This extreme example simply illustrates that in early and high-fertility populations, a high-prevalence epidemic might not necessarily lead to population decline.

An implicit but crucial assumption underpinning this conclusion, however, is that these early and high-fertility regimes are not themselves affected by the HIV-epidemic. The same demographic simulations can show that a modest compensatory fertility response can counter-balance the impact of HIV on population growth through mortality, or on the contrary, that by depressing fertility the epidemic could multiply its impact and then bring population decline (Heuveline 1997). A few precursors notwithstanding (e.g., Gregson 1994), the impact of the HIV epidemic on fertility has not attracted much attention from demographers, until the effect that infection itself had on the subsequent fecundity of HIV-positive women was recognized (Gray et al. 1998). Because of this effect alone, HIV-induced population decline can no longer be ruled out (Heuveline 2003).

However, there are many more aspects of a reproductive regime that may be affected by a severe HIV-epidemic and the increases in morbidity and mortality it induces. It is in fact untenable to assume that other than the biological effects of the virus on the fecundity and survivorship of infected people, everything else would remain the same in a population affected by a severe HIV-epidemic. Would the male and female age patterns of marriage, the probability of widows and widowers to remarry, the survival of children orphaned by the epidemic,

migration patterns be unchanged? The possible "downstream" effects of the epidemic are many (Barnett and Blaikie 1992), and while simulation models cannot substitute for data, they can illustrate the demographic consequences of different effects possibly induced by the epidemic and contribute to prioritize data collection needs.

Modeling HIV and Population Dynamics

Demographic projections are typically carried by macro-level discrete-time models, so-called cohort-component models that represent the survival and childbearing of different birth cohorts (Preston, Heuveline and Guillot 2001). Early projections of the demographic impact of the epidemic simply extended these models to incorporate exogenous projections of the number of AIDS-related deaths over time (United Nations 1993). In turn, the simplest AIDS-projection models use a mathematical function to fit extant HIV/AIDS trends and to extrapolate them into the near future. Developed at the World Health Organization, *Epimodel* for instance uses a gamma curve, trend in HIV-prevalence, and survival distribution from infection to death, to reconstruct and project trends in incidence and deduce future trends in AIDS-related mortality (Chin and Lwanga 1991). While the validity of the gamma curve has been questioned (for an assessment, see Salomon and Murray 2001), the curve captures an essential feature of an individual-to-individual infection, namely a sub-exponential growth in the number of new cases over time. Because HIV/AIDS projections make implicit demographic assumptions to link HIV-incidence, HIV-prevalence and AIDS-related mortality, the import of exogenous HIV/AIDS projections into demographic projections that make explicit (possibly different) assumptions may lead to some inconsistencies, which can be avoided by jointly simulating the HIV and population dynamics (Heuveline 2003).

While the overall shape of incidence over time appears similar across epidemics, those tend to have very different horizontal and vertical scales, that is, the time to reach a plateau ("saturation") and the prevalence level reached at that time vary across epidemics, for reasons that are still poorly understood. So called bio-behavioral models attempt to address these issues by modeling the different behaviors leading to HIV-infection. Macro-models do so by the allocating individuals to "risk groups", that is, grouping people according to their behavior and the greater or lesser risk that they become infected in a given period of time (e.g., Stanley et al. 1991). Micro-models do so by applying to individuals different distributions for the risk of experiencing a certain chain of events. Bio-behavioral models almost inevitably underestimate individual heterogeneity with respect to the risk of HIV-infection because the limited number of observable individual characteristics in surveys only allows for a poor proxy for the risk of infection. For instance, the number of partners in a given period obviously fails to represent the level of risk, since the highest risk of infection is actually for people who have only one partner and with whom they have sex regularly, when that partner is infected. This is perhaps the principal mode of infection for married women in Africa. We hence need to be able to go beyond individual characteristics and look at interactions between individuals, in particular "sexual mixing" patterns. This shortcoming is quite serious because models that underestimate individual heterogeneity actually misrepresent the saturation process that produces the sub-exponential growth in incidence, so that they eventually overestimate the epidemic (Brookmeyer and Gail 1994).

Another difficulty faced by bio-behavioral models is the modeling of the dynamics of the behaviors that may lead to infection. The initial distribution might be derived from a survey, for instance, but this distribution is altered over time by the differential rates of attrition precisely

because some behaviors are more likely than others to lead to infection and premature death. Thus, the model needs to make assumptions about the behaviors of new sexually active individuals for instance.

Because of such difficulties, macro-models that do not specifically represent the processes leading to infection but are based on an acceptable representation of infections over time are arguably preferable for the purposes of predicting an aggregate quantity such as future numbers of AIDS-related deaths. These models cannot, however, represent the many "downstream" demographic effects of the epidemic whose importance is now being recognized in high-prevalence populations.

Agent-based models are a new class of micro-simulation models that represent the behaviors and interactions of the different individuals or "agents." They have the potential to address the limitations of early bio-behavioral models described above, by modeling both the networks (e.g., sexual networks) that allow to understand individual to individual risk heterogeneity (the fact that the same behavior may carry different risks of infection), and the processes that contribute to individual engaging in these behaviors in the first place. They are also more adequate to study the social consequences of the epidemic because they model the relationships of HIV-positive individuals to other individuals.

Agent-Based Modeling

Over the last several decades, agent simulation has emerged as a novel methodology in the social sciences, one that integrates theory and empirical research, drawing premises and assumptions from the former, and generating aggregate patterns that can be compared with the latter. Although it takes somewhat different forms in the several disciplines, as a method it holds the promise of integrating the insights of multiple types of specialization into unified models.

The early work of Schelling (1978), Maynard Smith (1982) and Axelrod (1984)[2] provided a first wave of exemplars demonstrating the potential of a new approach to social simulation research.[3] Schelling's enormously influential model, which was essentially a thought experiment carried out on a checkerboard, was perhaps the closest to demographic concerns. With minimal technical resources, Schelling demonstrated that segregation at the aggregate level was possible without bias in the micro-level population. In general, these studies illustrate how relatively simple models can provide insights into complex issues, including those with potential policy implications.

A subsequent generation of agent simulation research, including Epstein and Axtell (1996), Axelrod (1997) and Young (1998), provides a second wave of exemplars. They respectively illustrate, *inter alia:* (1) how agent simulation can be applied to a range of interactive social processes, (2) the diversity of social topics that can be addressed using simulation based on simple agents, and (3) the emergence of social institutions and structure from the interaction of agent strategies.

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[2] For a critique of Axelrod's work from a game theoretic perpective, see Binmore (1998).

[3] The waves or generations of agent simulation exemplars identified here are drawn primarily from two areas of social simulation: complex adaptive systems and evolutionary game theory. Parallel developments were occurring in distributed artificial intelligence (AI, or multiagent systems, see Weiss 1999), demography (microsimulation, see Wachter, Blackwell and Hammel 1997), ecological modeling (individual-based simulation, see DeAngelis and Gross 1992), and computational organization theory (see Carley and Prietula 1994). Development in each of these areas followed a different pattern. Early and continuing contributions in distributed AI, for example, were primarily in a variety of technical and problem-solving domains (see Bond and Gasser 1988); only later did multiagent insights begin to be applied in the area of social simulation (cf., Castelfranchi and Werner 1994). It is of inherent interest how the same computational capabilities gave rise to similar innovations in various specialized areas of research.

Based upon such foundations, more specialized types of research began to emerge, for example in economics (Sargent 1993), ecology (DeAngelis and Gross 1992) and international relations (Cederman 1997). It is not surprising then that demography, with its tradition of microsimulation, came to apply agent-based techniques as well (Billari and Fürnkranz-Prskawetz 2003). Whether addressing migration, the evolution of the family dynamics, or important historical transitions, agent-based computational demography (sometimes abbreviated as ABCD) provides the means for more deeply probing the complexities out of which demographic processes arise.

Model Construction

As previously discussed, one of the strengths of agent simulation is its ability to model complex interactions. This potential, which provides a focal point for the expression of theoretical generalizations, is also what enables the capability of modeling the complex cultural and social structures through which the HIV virus is transmitted. To fully realize the potential of agent modeling, it is necessary to design relevant mechanisms, and also to structure the interactions among such mechanisms. This is fundamentally a theoretical enterprise, an activity that draws upon existing theory, and by which further theoretical insights can be refined.

The present research program has involved the design of four categories of mechanisms: (1) work-related migration, (2) networking and interaction, (3) disease and mortality, and (4) marriage and divorce. Each type of mechanism can be seen as contributing to the larger pattern of HIV transmission in Eastern and Southern Africa, where to date the highest prevalence levels have been found among general populations. In this paper, we discuss the construction of a basic version of the model, and its gradual elaboration. This basic model has a full architecture in the sense that the four mechanisms are represented, but they are initially represented by aggregate

statistical distributions only, as they would be in any other type of micro-simulation. These aggregate parameters can be thought as "place holders" in order to establish the architecture of the full model, but will be gradually replaced by modeling the rules of behavior and interactions between agents that determine the observed distribution. It is only when this is fully implemented that the full potential of an agent-based simulation will be realized. At this time, only the marriage and divorce module has been so implemented.

The Basic Model

Work-Related Migration. In Southern Africa, migration plays a role in the spread of the HIV virus (Hunt 1989; Chirwa 1997; Hampshire 2002). Young men migrate to urban areas and/or work camps where the HIV/AIDS rate and the risk of infection are significantly higher than in the villages and rural areas. Specific parameters will vary from population to population, depending *inter alia* on topology, population distribution and cultural patterns. However, the generalized effect of migration creates a two-tier structure to the spread of the disease to which an HIV/AIDS model must attend. The sources of variation can then be explored by conducting a sensitivity analysis of relevant parameter ranges.

Our baseline migration model is driven by an exogenously determined unemployment rate. At present, the structure of its distribution, which is relative to quasi-discrete bands, is artificially defined. Subsequent refinement can substitute a theoretical or empirical economic base, but the present goal is simply to capture the two-tier structure.

As is typical in employment-driven migration processes, we assume that the propensity to migrate is highest among young adult males. The frequency of migration for specific agents gradually declines as, over time, migrating workers age and marry. Seasonal effects also influence the rate of migration. In the model, the rate of migration return is determined by the

season, and the duration of the agent's current migration. The entire migration process can be visualized using a Geographical Information System (GIS) capabilities.

Networking and Interaction. Potential sexual partners are found within affinity networks of various types. In the basic model described here, affinity networks operate according to the following rules: (1) new acquaintances (and therefore prospective sexual partners) are introduced by mutual friends, (2) friendships without further contact decay over time, and (3) there is an upper limit on total friendships.

The number of sexual partners for a given agent is reduced by village residence, increased by migration, and is influenced by marital status (i.e., after agents marry, the number of sexual partners in a given time period is reduced). In the current model, the frequency of sexual intercourse is based on an empirical distribution shaped by the values of relevant parameters. As discussed before, as the model evolves, any particular component may be refined or replaced.

Disease and Mortality. In the basic model, we have two mortality schedules depending on HIV status. In other words, when an individual becomes infected, she leaves the original age-at-death distribution and her age at death follows a second distribution corresponding to her reduced survival chances.

The infectivity of infected agents is also duration dependent, that is, depends on the length of time between the time of infection and the time of a subsequent sexual contact. As suggested by epidemiological studies, infectivity is assumed to be highest during the first two weeks after the infected agent has been exposed to the virus and lowest immediately thereafter. Subsequently, there is a gradual increase correlated with the length of the agent's infection.

Marriage Formation and Dissolution. In our earliest models, marriage and divorce rates were based on a probability distribution summarizing empirical age patterns. During young adulthood (ages 20-29) both marriage and divorce rates are relatively high. Subsequently, both marriage and divorce rates drop to levels that are roughly equivalent. There are aspects of the African cultural context (e.g., polygamy) that have not yet been captured in this model.

Module Substitution

Marriage is the first example in which one of the underlying mechanisms has been refined by replacing the basic mechanism with one that it is more sophisticated and intuitive. Specifically, what might be called the Basic Plus model draws upon and extends the marriage formation model of Todd and Billari (2003). In this model, each agent has a base quality, aspiration level and courtship duration. Each is assigned randomly from a normal distribution. From about age 13 on, each agent surveys their friends in search of a friend of the opposite sex whose quality level exceeds their aspiration level. When one is found, an offer of courtship is extended. If the potential partner agrees, using the same criteria, a dating relationship is formed.

During courtship, agents continue to look for a better relationship with friends of higher quality. Each agent also has a waiting threshold. If they do not participate in a courtship for longer than that threshold, their aspiration level is reduced. If agents date someone whose quality is higher than their aspiration level, the latter is adjusted upward. Alternatively, if an offer of a relationship is rejected, the agent's aspiration level declines as well. Ultimately, if the relationship lasts longer than the courtship duration parameter of both agents, they get married.

This marriage formation model tries and captures the serial and contingent nature of relationship formation. We are also in the process of further elaborating the formalization of aspiration levels, which in the original model were represented by a mono-dimensional

parameter. In this present study, we try and represent aspiration levels as multi-dimensional parameters depending on observable characteristics of prospective partners. At this time, age and wealth have been incorporated so and we will next incorporate religious and cultural preferences.

Discussion

Agent Research Lifecycles

In assessing the status of the present initiative, it is essential to consider the development cycle of agent simulation research programs, especially those involving complex, multilevel models. Broadly, there are three phases: design, development and exploration.

During the design phase, research priorities are established, and component mechanisms are designed that contribute to the overall research focus. The tasks of this stage include identification of the parameters to be used in sensitivity analysis. During the development phase, computational models of the mechanisms are developed and integrated into a multilevel model. This stage includes sanity and plausibility testing. The exploratory phase involves exploration of parameter interaction, and validation studies, followed by assessment and refinement.

In the research program continues, this lifecycle may itself be iterative, as the research program is broadened or deepened into new areas. These target areas define the overall direction of the research program, as will shortly be discussed for the present initiative.

Current Results

The current study has completed phases one and two, and is presently moving into the exploratory phase. The baseline HIV/AIDS model for Eastern and Southern Africa has been designed and developed, and shown to produce a number of the gross qualitative structures that characterize the epidemic. We are now conducting a sensitivity analysis and, as data permits, will explore the alignment of these results with the varying infection patterns across Eastern and

Southern Africa. This phase initially focuses on how five population characteristics evolve over time as presented in Table 1.

Table 1. Sensitivity Analysis: Population Characteristics

Ratio of male to female currently infected with HIV/AIDS
Male & Female Adult Prevalence (% HIV positive in the population ages 15 to 64)
Proportion of population by age category (0-15, 15-25, 25-50, 50+)
Proportion of children who are orphans (maternal, paternal or both)
Proportion of adults age 50+ with at least one orphaned grandchild whose deceased parent was their own child

These endogenous variables are affected by the parameters governing each of the mechanisms of the model, including the prevalence of young adult migration, transmission rate (depending on agents' condom use), characteristics of affinity networks, and duration of the spouse search. The interaction of these effects will be demonstrated during the presentation.

The longer-term goal of these activities is to produce an analytical framework that can be used to derive theoretical insights and be applied to policy-related issues. To achieve this goal it is necessary to add to the social and demographic complexity of the model. It is to this issue that we now turn.

Future Plans

The future objectives of the present project involve introducing greater complexity in systematic steps. Two key types of complexity are family structure and cultural variation. Regarding the latter, we will define cultural groups to which agents will identify to a varying degree depending on their religion, ethnicity, and education. Regarding the former, we will define residential unit

(household) in addition to the existing marital and parental relationships. This will allow us to study the adaptive contours of households as the epidemic affects a community.

References

Anderson, R.M., R.M. May, M.C. Boily, G.P. Garnett, and J.T. Rowley. 1991. "The Spread of HIV-1 in Africa: Sexual Contact Patterns and the Predicted Demographic Impact of AIDS," *Nature* 352(6336):581-89.

Axelrod, Robert. 1984. *The Evolution of Cooperation.* New York: Basic Books.

_____. 1997. *The Complexity of Cooperation: Agent-Based Models of Competition and Cooperation.* Princeton, NJ: Princeton University Press.

Barnett T, Blaikie P: *AIDS in Africa: the present and future impact*. London: Bellhaven Press; 1992.

Billari, Francesco C. and Alexia and Fürnkranz-Prskawetz (Eds.) 2003. *Agent-Based Computational Demography: Using Simulation to Improve Our Understanding of Demographic Behaviour.* Berlin: Physica Verlag.

Brookmeyer, R. and M.H. Gail. 1994. *AIDS Epidemiology: a Quantitative Approach*. New York: Oxford University Press.

Cederman, Lars-Erik. 1997. *Emergent Actors in World Politics.* Princeton, NJ: Princeton University Press.

Chin, James and S. K. Lwanga. 1991. "Estimation and Projection of Adult AIDS Cases: A Simple Epidemiological Model." *Bulletin of the World Health Organization* 69(4):399-406.

Chirwa, W.C. 1997. "Migrant labour, sexual networking and multi-partnered sex in Malawi," *Health Transition Review* 7(suppl 3):5-15.

DeAngelis, Donald L. and Louis J. Gross (Eds). 1992. *Individual-Based Models and Approaches in Ecology.* New York: Chapman & Hall.

Epstein, Joshua M. and Robert Axtell. 1996. *Growing Artificial Societies: Social Science from the Bottom Up.* Cambridge, MA: MIT Press.

The European Collaborative Study. 1999. "Maternal Viral Load and Vertical Transmission of HIV-1: An Important Factor but Not the Only One." *AIDS* 13(11):1377-85.

Gibb, D.M. and B.H. Tess. 1999. "Interventions to Reduce Mother-to-Child Transmission of HIV Infection: New Developments and Current Controversies." *AIDS* 13(suppl A):S93-S102.

Gray, R.H., M.J. Wawer, D. Serwadda, N.K. Sewankambo, C. Li, F. Wabwire-Mangen, L. Paxton, N. Kiwanuka, G. Kigozi, J. Konde-Lule, T.C. Quinn, and C.A. Gaydos. 1998. "Population-Based Study of Fertility in Women With HIV-1 Infection in Uganda." *Lancet* 351(9096):98-103.

Gregson, S. 1994. "Will AIDS Become a Major Determinant of Fertility in Sub-Saharan Africa?" *Journal of Development Studies* 30(3):650-79.

Hampshire, K., 2002, "Fulani on the Move: Seasonal Economic Migration in the Sahel as a Social Process," *Journal of Development Studies* 38 (5), pp. 15-36.

Heuveline, P. 1997. "AIDS and Population Growth in Sub-Saharan Africa: Assessing the Sensitivity of Projections." *Population Research and Policy Review* 16(6):531-60.

_____. 2003. **"**HIV and population dynamics: a general model and maximum-likelihood standards for east Africa," *Demography* 40(2):217-245.

Maynard Smith, John. 1982. *Evolution and the Theory of Games*. New York: Cambridge University Press.

Hunt, C.W. 1989. “Migrant Labor and Sexually Transmitted Disease: AIDS in Africa,” *Journal of Health and Social Behavior* 30 (4):353-373.

Leroy, V., C. Montcho, O. Manigart, P. Van de Perre, F. Dabis, P. Msellati, N. Meda, B. You, A. Simonon, and C. Rouzioux. 2001. "Maternal Plasma Viral Load, Zidovudine and Mother-to-Child Transmission of HIV-1 in Africa: DITRAME ANRS 049a Trial." *AIDS* 15(4):517-22.

Morgan D, Mahe C, Mayanja B, Okongo JM, Lubega R, Whitworth JAG. 2002. “HIV-1 infection in rural Africa: is there a difference in median time to AIDS and survival compared with that in industrialized countries?” *AIDS* 16:597-603.

Preston, S.H., P. Heuveline, and M. Guillot. 2001. *Demography: Measuring and Modeling Population Processes*. Oxford, England: Blackwell Publishers.

Salomon, J.A. and C.J.L. Murray. 2001. “Modelling HIV/AIDS Epidemics in Sub-Saharan Africa Using Seroprevalence Data From Antenatal Clinics,” *Bulletin of the World Health Organization* 79(7):596-607.

Sargent, Thomas J. 1993. *Bounded Rationality in Macroeconomics.* New York: Oxford University Press.

Schelling, Thomas C. 1978. *Micromotives and Macrobehavior.* New York: W.W. Norton.

Stanley, E. A., S.T. Seitz, P.O. Way, P.D. Johnson, and T.F. Curry. 1991. "The United States Ineragancy Working Group Approach: the IWG Model for the Heterosexual Spread of HIV and the Demographic Impact of the AIDS Epidemic." Pp. 119-36 in *The AIDS Epidemic and Its Demographic Consequences, Proceedings of the United Nations/World Health Organization Workshop on Modelling the Demographic Impact of the AIDS Epidemic in Pattern II Countries: Progress to Date and Policies for the Future, New*

York, 13-15 December 1989, edited by the United Nations. New York: United Nations and World Health Organization.

Todd, Peter M. & Francesco C. Billari. 2003. "Population-wide marriage patterns produced by individual mate-search heuristics," pp. 117-137 in F.C. Billari and A. Fürnkranz-Prskawetz (Eds.) *Agent-Based Computational Demography: Using Simulation to Improve Our Understanding of Demogrqaphic Behavior.* New York: Physica-Verlag.

United Nations. 1991. *The AIDS Epidemic and Its Demographic Consequences, Proceedings of the United Nations/World Health Organization Workshop on Modelling the Demographic Impact of the AIDS Epidemic in Pattern II Countries: Progress to Date and Policies for the Future, New York, 13-15 December 1989*. New York: United Nations and World Health Organization.

———. 1993. *World Population Prospects: the 1992 Revision*. New York: United Nations.

Young, H. Peyton. 1998. *Individual Strategy and Social Structure: An Evolutionary Theory of Institutions.* Princeton, NJ: Princeton University Press.