ASSESSING THE LEVELS AND IMPACT OF MORTALITY IN CRISIS SITUATIONS

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INTRODUCTION.

Population crises characterized by sudden increases in mortality were a recurrent feature of preindustrial populations. As a result, the study of their nature,intensity,causes, and impact on demographic and social structures has been largely the task of historical demographers. Long after their disappearance from the European scene, crises still reappear with some regularity in overburdened populations although with different intensity, regularity, and geographical scope. To the evidence revealing devastating catastrophes occurring during the end of the XIXth century in India and China, other information has been added about crises of significant intensity and scope in more recent periods: the Great Bengal famine of 1943,the Ethiopian famines of 1973 and 1974, the Russian famines in the late 1920's, the Bangladesh famines of 1971 and 1974, the China famine of 1959-61, and the Sahelian drought of 1970-1972. In some cases the cause is the outbreak of political upheavals accompanied by pervasive and protracted warfare. In others, the culprit is the persistence of climatic conditions that lead to the collapse of normal production and distribution of food. With a few exceptions (for example the Great Malaria epidemic of 1934-35 in Sri-Lanka), crises generated or dominated by the presence of epidemics have been absent. The spread of diseases, however, remains an important

mechanism through which wars or adverse climatic conditions increase mortality, depress fertility, and alter the regional distribution of the population.

Population crises have long been thought to be a ' natural ' mechanism through which a homeostatic equilibrium is regained once populations experience more or less prolonged drifts away from it. Malthus saw them as a court of last resort, the archetype of the positive checks that prevent population growth beyond the carrying capacity. In the past two decades or so, the debates about the relations between population and socio-economic well-being have recast the role that population crises play in the absence of deliberate population control. They are seen either as the ultimate consequence of failure to bring down the rates of population growth (Meadows, et al., 1972) or as exaggerations, the product of misreading of population and economic statistics (Simon, 1980). The wave of renewed interest has also prompted efforts to reorder our notions about the role of population crises in the preindustrial period (Watkins and Van de Walle, 1983). Partial evidence suggests that, at least in the long run, they could not have provided sufficient relief from population pressure build-up (Watkins, 1985). More recent events suggesting the rapid world-wide spread of a deadly AIDS epidemic could alter the terms of the above-mentioned debates, as the death toll in certain areas may mount to unforeseen levels and if unchecked, upset the

functioning of entire economic, political and social systems.

The aim of this paper is to study the nature of population crises, to describe the techniques for measuring their intensity, and to assess the potential role that they play as a check on population growth. In the second section, I present a model of the mechanisms leading to population crises. This model is a generalization of a simpler one suggested by Chen and Chowdhury (1977) for the study of famines. It will be used to formulate a typology of crises and to define the expected behavior of common demographic indicators. In the third section, I discuss the typical trajectory of demographic indicators immediately before, during and immediately after a crisis; allowance is made for variations due to the nature, magnitude and duration of crises. In the fourth section, I present an assessment of alternative measures of the intensity of population crises. I show that proper formulation and application of these measures would benefit from a prior examination of the mechanisms that trigger the crises. In the fifth section, I deal with the impact of population crises on prospective population growth and on selected aspects of the social structure. Here I contest the argument according to which the 'effectivness' of crises as mechanisms of population control is only of minimum significance.

II. THE NATURE OF POPULATION CRISES.

a) Basic variables and relations.

Although the causal chain that leads to the outbreak of population crises is quite complicated and variable, a few universal features can be highlighted. First, there is always a triggering event that, under certain conditions, will lead to reponses of mortality, fertility, and migration. The triggering event may be purely exogenous or endogenous. Thus, the occurrence of flooding or droughts or other vagaries of climatic nature is an exogenous event since it is not directly induced by social design or population conditions. It may well be that population pressure on land, patterns of land utilization, and settlements could, on the long haul, affect weather patterns and through soil degradation, increase the sensitivity of output to changes in weather. In these cases, however, the role of population as a cause is remote and can be safely disregarded. On the other hand, political unrest accompanied by more or less systematic warfare or the implementation of economic policies affecting food production or its distribution,shall be considered endogenous triggering events. In most cases the outbreaks of epidemics are part of a chain initiated by an exogenous or endogenous triggering event. However, there are situations when they are the result of long-term changes in a delicate balance between agents of disease and human hosts. In these cases, they shall be considered

exogenous events. This distinction is introduced here to dispose of the idea that all crises are endogenously produced, the result of excessive pressure on resources. The connection between the occurrence of the crises and population pressure should not be postulated a priori but left to investigation. It may be that the triggering event and the chain of reactions that follows it would not have taken place in the absence of rapid population growth or unbalanced population distribution. Yet, this is a proposition for falsification, not an axiom on which interpretations should be based. Second, the triggering event may lead to two alternative and possibly complimentary responses. The first is the production of food shortages and the second is the disturbance of the patterns of entitlement to goods and services for all or a part of the population. The distinction between these two mechanisms was first introduced by Sen (1980) and later used in other research on the effects of crises (Watkins and Menken, 1985; Watkins and Van de Walle, 1983; Chen and Chowdhury, 1977; Bongaarts and Cain, 1981). Sen argued that in many cases where famines had been documented, there was no evidence of a collapse of food production and a consequent food shortage. Rather, what led to starvation and its sequelae was the deterioration of terms of trade for some groups of the population. For groups whose claim over goods and services is based on self-subsistence, famines MAY occur as an outcome of adverse conditions affecting

production if and when the capability to command resources outside the boundaries of self-subsistence is also compromised. By and large, this takes place when there is a general collapse of food availability. It may also occur, however, when total food production is unaltered but the institutional mechanisms to make it accessible to the self subsistent population are lacking. More often than not overall output is not greatly affected only by local deficits. What leads to famines is the inability of the self-subsistent groups to find adequate alternatives. Groups whose claims over goods and services depend on exchange of products or factors of production face adverse conditions when relative prices and market conditions are altered even though production of basic foods remain unchanged. It follows that the use of food production or availability per capita as an indicator of actual availability may be meaningless inasmuch as it masks transformations in the system of entitlements. Serious disruptions of food production may lead to changes in the systems of entitlements for groups other than those relying on self-subsistence and, vice versa, alterations in entitlements may eventually cause a drop in food production. But,although the two mechanisms are related, their effects have to be considered separately. More importantly, the triggering event may effect only one of them without necessarily affecting the other.

The relations just described are illustrated on the top

panels, A and B, of Figure 1. They represent what we may consider the 'background' variables affecting population responses. The third level of Figure 1, Panel C, illustrates the relations between selected 'intermediate' variables and the demographic responses. Whether due to food shortage or disruption of entitlements, a first impact is felt in the levels of nutrition of the population affected. Two additional responses are highly likely: a decrease in nuptiality and an increase in outmigration. The levels of marital fertility are affected directly by nutrition (Bongaarts and Delgado, 1977), by spouse separation due to outmigration or by decreased frequency of sexual intercourse. Finally, mortality increases due to the incidence of infectious diseases-- facilitated by lowered resistance of hosts-- and the spread of agents of diseases due to higher population mobility out of the affected area and aggravation of crowdiness. Starvation itself can, of course, become a major direct cause of higher mortality. Feedback mechanisms are quite important in this representation. First, an increased incidence of diseases aggravates malnutrition and reinforces outmigration. Second, an increase in infant mortality will disrupt patterns of breastfeeding, reducing the length of postpartum amenhea and lowering intervals between conceptions. Third, increased mortality and outmigration disrupt production and distribution of goods. The first two feed-back mechanisms occur within the set of

intermediate variables, whereas the third leads from effects of the latter on the set of background variables. The inclusion of feed-back mechanisms is useful for the study of crises whose effects are dampened over time as well as of those that have a tendency to self-reproduce, aggravating the impact of initial effects. Also, the representation is general enough to apply to situations where the triggering event is the presence of a disease, as was the case during preindustrial times, rather than the vagaries of weather, natural disasters or military actions.

The triggering event has a direct impact on variables contained in level C rather than B and, through it, leads to changes in food production and system of entitlements. It thus generates another set of responses among the variables in level C. Figure 2 represents the two alternative types of mechanisms leading to population crises.

b) Types of crises.

The distinction just made permits us to define two main types of crises: one in which the triggering mechanism operates directly through level B and the other in which the first impact of the triggering mechanism is on variables in level C. Two additional types of population crises may be distinguished depending on the importance of food shortages relative to changes in entitlement systems. In each of these cases, two further types may be defined according to the most typical demographic response:

in some, outmigration will dominate over changes in the patterns of nuptiality and marital fertility. In others, the most frequent response will be postponement of marriages, separations and delayed childbearing. Finally, within each of these types one could further distinguish crises according to the nature of the mortality response. Although crises may result in age-sex-invariant (approximately neutral) mortality increases, it is more common to find strong age and sex differentials in mortality excesses.

With no further elaboration beyond the simple sequence of dichotomous types that I have established before, there are a total of 16 types of crises. To these one may add a final set depending on the intensity of the mortality response and the duration of the crisis (Charbonneau and Larose, 1979). Since the dimensions used to construct these types are correlated, it is likely that only a small subset will be of any empirical relevance. Table 1 suggests a classification of a handful of population crises according to a small number of criteria. The distinctions highlighted by the typology are useful to distinguish the expected behavior of various demographic indicators immediately before, during, and immediately after the crisis. In turn, the latter can be used as evidence to infer the type and the characteristics of the crisis.

III. DEMOGRAPHIC RESPONSES AND THEIR INTERACTIONS.

In the previous section I made no mention of specific demographic indicators and instead preferred to identify demographic responses in general. This is because the trajectory of demographic indicators depends on which ones are used. Typically one relies on the easiest ones to compute, the crude rates (Bongaarts and Cain, 1981; Chen and Chowdhury, 1977; Anderson and Silver,1985; Appleby, 1978; Hollingsworth, 1979;Livi-Bacci, 1978). These, however, are affected by changes in the age structure of the population which occur as a result of the sudden variation of vital events. A large increase in infant mortality, for example, will set a favorable stage for future reductions in the crude death rates even if the adverse conditions were not altered over a period of several years. This suggests that one should first trace the response in age specific rates and then attempt to infer the trajectory of more conventional demographic indicators.

a) Mortality.

a.1) Crises due to relative food shortages.

When the triggering event affects directly the levels of food production or system of entitlements, the age specific rates should rise rather slowly for an interval of time after food shortages become evident. This is because at the onset of the crisis the population may use inventories or resort to liquidation

of assets in exchange for access to food as a means of coping with relative scarcity. Unless the pre-crisis levels of nutrition are too low to begin with, the increase in mortality accelerates only after the scarcity of food leads to widespread undernourishment, chronic deficiencies in the immune system and increases in crowding. As food production and distribution return to normal levels, the force of mortality progressively decreases. Infant mortality is expected to rise due to malnutrition among children older than one month and less so due to decreases in birth weight affecting the neo-natal force of mortality. In most societies, however, the practice of breastfeeding provides a protection for all infants and makes them less vulnerable to sudden shifts in food availability unless there is pervasive malnutrition among the mothers. Mortality in the age group 1-5 is highly sensitive to crises triggered by relative shortages of food. Young children depend on solid foods and their immune system is not completely developed. Unlike infants, they do not enjoy the double protection offered by breastfeeding. The combination of food scarcity and spread of infectious diseases has had devastating consequences within this age group in almost all documented cases (Chen, 1973; Curlin et al., 1971; Chen and Chowdhury, 1977; Kane, 1984; Ashton et al., 1985; Turpeinen, 1979). Sex differentials in mortality are exacerbated within this age group. Overmortality of females has been reported even in cases where this is not a characteristic

feature of pre-crisis mortality patterns (Kane, 1984; Ashton, et al. 1985). Mortality above age 60 is also increased more than proportionately. This occurs as a result of higher suceptibility to diseases as well as to the unfavorable shifts in the distribution of food and services that takes place under the stress of the crisis. Unfavorable distribution of resources within the household aggravated by the stress of lactation or childbearing is at the root of female excess mortality within the reproductive period. In most documented cases, female mortality increases are higher and the absolute levels may reach higher values than for males (Curlin et al., 1971).

a.2) Crises due to wars.

The onset of the rise in the force of mortality arrives sooner and is steeper when the initial stage of the crisis is characterized by warfare, as it should reflect the excess deaths due to military casualties. As the latter account for a substantial amount of excess deaths, the cessation of hostilities brings a return to normal levels of mortality at a faster pace than in other types of crises. This should hold at least for the age-specific deaths corresponding to ages 15-50. The age pattern of mortality excesses shows higher relative increases for males in the ages 15-50 than in other types of crises. However, since wars bring disruption of food production and distribution, both the levels and the age-sex pattern of increases in mortality may

eventually be undistinguishable from the one described above. Differences would only be discerned during the initial stages of mortality increase and in the tail end of the crisis, as mortality declines less rapidly when the main triggering event is war.

a.3) Crises due to epidemics.

In these cases the onset of mortality increases depends on the speed with which the disease is diffused in the population. The pace of its decline depends on the relative accommodation of host and agents of disease, the proportion of individuals who are not susceptible and the efficacy with which the infected population is isolated from the rest to halt transmission. At least during the initial stages of the epidemic, the levels and age-sex patterns of mortality increases are dependent on its virulence and age-sex specific incidence. Thus, for example, before the introduction of the vaccine, 50% of the excess deaths caused by a smallpox epidemic was accounted for by children younger than 5. On the other hand, the age patterns of mortality excesses during a cholera epidemic are strongly biased toward adults and old people (Salvini, 1977). As can occur with population crises triggered by wars, those triggered by disease may just be the initial stage of a protracted period of crisis during which the effects are exacerbated by disruptions in food production and distribution. If this occurs, the absolute levels and the age-sex specific patterns of mortality excesses will

eventually follow the profile described in (a.1): to the pattern of excesses due to disease alone is superimposed another one due to the feed-back mechanism.

In all three types of crises, there is an underlying selection process which will affect the post-crisis levels of age-specific mortality. On the one hand, when the selection process leads to an elimination of the most frail, post-crisis population composition should be biased toward the least frail groups. The discrepancies should be higher at ages where the selection is stronger. As a consequence, the levels of mortality will be somewhat lower for a time after the crisis has run its course. On the other hand, when the selection process disproportionately affects those who are least frail, one would expect higher levels of the force of mortality over the short run during the post-crisis period than during the pre-crisis period. If the triggering event leads to food shortages or loss of entitlements, we would expect to have heavy selection of the <u>least</u> frail among the youngest. If the triggering event is warfare, we would expect heavy selection of the <u>most</u> frail at ages when recruitment into the military is most likely.

Table 2 summarizes the relations between levels, age-sexpatterns, and timing of mortality excesses on the one hand and types of crises on the other. One caveat is necessary here. So far I have assumed a homogeneous population, one with no social

inequalities. In reality, pre-crisis mortality differentials are exacerbated during and immediately after a crisis. The historical record shows that when the latter have been generated by the diffusion of diseases, the wealthier groups stand better chances of surviving (Livi-Bacci, 1978). Yet, the contrasts are not nearly as high as those experienced in contemporary developing societies (Alagmir, 1980). Regional differentials will be consistent with the regional patterns of food shortages and with regional differences in access to markets. In general, however, it is the urban areas which are least affected.

b) Fertility.

Fertility responses to crises have two main components. The first corresponds to reactions of the nuptiality pattern. The second corresponds to effects on frequency of intercourse, length of postpartum amenorrhea and fecundity.

b.1) Nuptiality.

The sensitivity of nuptiality patterns to crisis situations is enhanced whenever marriages are dissociated from menarche. This was the case in societies characterized by the ' Western European marriage pattern ' (Watkins and Van de Walle, 1983). The flexibility of this mechanism is less so in contemporary developing societies where the association between marriage and menarche is stronger. Yet, all documented cases show evidence of significant changes in nuptiality. Transaction costs, negative

future expectations and actual physical displacement of potential spouses, all contribute to reduce the probabilities of marriage in prime ages. As indications of serious disruption of food production and distribution of food become evident, households reallocate scarce resources to meet expected shortages. As a result, marriage contracts depending on dowry payments are reduced. Similarly, increases in the costs of adopting a spouse as a new household member or drastic changes in the prospective costs of setting up a separate household lead to systematic avoidance of new marriages (Caldwell et al , 1984; Bongaarts and Cain, 1981). When wars are the triggering event or when other triggering events lead to generalized outmigration, the normal course of nuptiality is, in addition, interrupted by sheer contraction of the marriage market. Depending on the intensity of the crisis, the onset of the nuptiality response can be swift, massive, and frequently becomes an early warning of the impending crisis (Kane, 1984). By the same token, the post-crisis levels of nuptiality may rise very rapidly, as marriages postponed over a rather extended period of time take place within a short interval of time. Such response is either attenuated or altogether prevented when the crisis has more or less permanent effects on marriage markets. This occurs with wars and wholesale outmigration. As in the case of mortality, nuptiality responses may vary by social groups. Available evidence shows that postponment of marriages is intentionally used by

certain groups as a strategy for survival whereas others experience little if any changes (Caldwell et al., 1984).

b.2) Marital fertility.

The response of marital fertility is somewhat swifter and depends on three factors. First, frequency of intercourse is reduced significantly. This occurs as a result of stress and poor physical condition, as a result of physical separation due to war and outmigration, or is due to deliberate abstinence. Second, fecundity is diminished as stress or malnutrition impair normal amenorrhea and anovulatory cycles. Third, higher levels of infant mortality lead to shorter postpartum amenorrhea periods (via reductions in the length of breastfeeding) thus offsetting somewhat the trend towards lower fecundity. Its strongest impact will be felt one year or so after the outbreak of the crisis but it is likely to be dwarfed by effects with opposite signs associated with the other two factors. At any rate, the downswing in fertility will be of lower magnitude than when breastfeeding is rarely practiced. Unlike the onset of the response in nuptiality which precedes the outbreak of the crisis, the earliest indication of marital fertility reductions will take place several months into the crisis. Similarly, whereas the post-crisis nuptiality levels may experience an immediate boost, restoration of the pre-crisis levels of marital fertility may take as long as nine months after the peak of the crisis is passed. The post-crisis rate of

increase of marital fertility will be a function of the effects that the crisis has had on marital disruption due to mortality or to migration. If the latter are unimportant, the increase may be substantial even in the absence of any intentional behavior to 'replace' the lost population. This is because the composition of female by susceptibility status will be unusually favorable to higher fertility (Bongaarts and Cain, 1981).

Table 3 summarizes the most important associations between types of crises, timing and intensity of the response of nuptiality, and effects on marital fertility.

c) Outmigration and return migration.

Intense and sudden flows of migrants out of the areas most stricken by the crises has been well documented in preindustrial and contemporaneous populations (EA Meng-Try,1981; Livi-Bacci, 1978; Ashton et al., 1985; Kane, 1984; Chen and Chowdhury, 1977; Caldwell, 1975). These flows may permanently alter the regional distribution of population (no return migration) or they may produce temporary relief for as long as the crisis lasts. Apart from the fact that outmigration itself can lead to higher mortality among those who migrate (dislocation, inability to adapt to new environment) and in the areas of destination (diffusion of diseases), it is unclear just what effect they actually have on the conditions at the origin. It has been argued that the outflows relieve pressure on resources and hence lead to more

rapid recovery (when only food shortages are the cause of the crisis). However, it is also the case that just like mortality, migration is also selective and may involve the most active and productive members of the sending population. If this were the case and no substantial return migration took place, the sending areas should deteriorate even further and their post-crisis mortality should remain at levels higher than the pre-crisis levels as those who remain are the most frail. Massive displacements of population are an invariable characteristic of crisis triggered by warfare (e.g., the cases of Central America, Eritrea, Afghanistan, Cambodia). Movements of such magnitudes are regularly accompanied by dismal health conditions and extraordinarily high levels of mortality at all ages. This is why protracted warfare, even though localized, may end up having a devastating cumulative effect on the demography of a population. Massive displacements of population not only lead to deterioration of health conditions; they also have a deep impact on fertility and, more generally, on household and family organization. In extreme cases, the latter disintegrates and isreplaced by alternative institutional forms (Montes, 1985). On the other hand, although fertility is initially depressed, it may experience important and sustained surges as pre-crisis restrictions in cohabitation and sexual intercourse break down. Reproduction may begin to take place among couples not regularly

in unions with the consequent disarray in the system of child care and child rearing. This reinforces the conditions keeping high levels of mortality.

Migration has also been the response to epidemics (Livi-Bacci, 1978; Charbonneau and Larose, 1979) and appears as an early warning of relative food shortages (Caldwell et al., 1984; Caldwell, 1975; Ashton et al., 1985; Lardinois, 1982). In both cases, return migration is more likely to occur than when wars are the triggering event since, unlike wars, epidemics and food shortages in general do not involve destruction of the environment. Similarly, the age and sex pattern of migration is likely to be more selective than in the case of wars.

d) The behavior of demographic indicators.

The foregoing considerations lead to inferences about the most likely course of selected demographic indicators. Consider the case of a crisis triggered by food shortages with no feed-back mechanisms. As indicators of mortality, I will take the probability of dying before age 5 (Q5) and the crude death rate (CDR). As indicators of fertility, I will use the total fertility rate (TFR) and the crude birth rate (CBR). Assume that generalized food shortages appear at time To and that they last about a year. Inmediately before To there will be a slight increase in Q5 and CDR as important segments of the population experience gradually worsening conditions due to a lack of inventories and funds for

protection. During the initial phase of the crisis, CDR will increase rapidly as the force of mortality at all ages increases. A similar trajectory will be observed for Q5. Within the year after the end of the crisis, the CDR will begin to decline, reproducing the decline in age-specific rates. However, its level may dip further down than Q5 (and lower than the pre-crisis levels) as a consequence of the changed composition of the population. First, as a result of short-term reductions of fertility and higher levels of mortality in infancy, the age structure of the population becomes more 'favorable' to a lower CDR. Second, the composition of the population by frailty is biased toward those least frail, and the age specific mortality rates ought to be on the whole somewhat lower than the pre-crisis levels. Figure 3 displays the trajectories corresponding to CDR and Q5. As a result of either an increase of outmigration or of a tighter nuptiality regime, both the CBR and the TFR begin to decline <u>before</u> the onset of the crisis. Nine months into the crisis, they reach their 'crisis levels.' This occurs as a result of lowered conceptions during the previous nine months. The fall in CBR is slower than in TFR since the former also reflects the changed age composition of the population which is more favorable toward population in reproductive ages. If return migration takes place and the level of marital disruption caused by adult mortality is insignificant, then the restoration of pre-crisis

levels of nuptiality and marital fertility occurs in two stages. First, there is a sudden surge of nuptiality immediately after the end of the crisis. The CBR and TFR remain at low levels until about a year past the end of the crisis. The second stage begins with increases in CBR and TFR as a consequence of the surge of marriages and of resumed marital relations. Both indicators will increase to levels exceeding those observed during the pre-crisis period. The 'excess' in TFR will be due to the unusual composition of the female population by susceptibility status. The increase in the CBR will be the direct consequence of increased TFR as well as of an age structure which has higher proportions in the childbearing ages. Figure 4 displays the trajectory of TFR and CBR. Figures 3 and 4 should be contrasted with Figure 5 where I have plotted the trajectory for selected indicators of mortality and fertility in Bangladesh, China, Finland, and Sweden during periods bounded by and containing crises. Finally, Figures 3,4, and 5 should be compared to Figure 6 which displays crude rates for simulated regimes of crises (see Section V).

IV. MEASUREMENT OF THE INTENSITY OF CRISES.

Historical demographers have developed a long list of alternative measures of crises intensity. Virtually all of them focus on mortality alone and most are based on counts of deaths that are not tied to counts of the population exposed to the risk of dying (Charbonneau and Larose, 1979). An important

preoccupation is the distinction of mortality increases or fluctuations due to extraordinary events from increases or fluctuations that can be expected from sampling variability. This is explicable by the historical demographers' focus on small populations and should not be of great relevance when dealing with large aggregates. In all these cases the idea is to estimate the excess deaths that are due to the occurrence of a crisis or, equivalently, the number of deaths that would not have occurred had 'normal' conditions persisted.

a) Hollingsworth's index.

This is the result of a series of criteria used to highlight the objective and 'subjective' impact of a crisis. The index is defined as follows:

$$I = Q/(1-Q) * (n)^{2/3} * (t)^{-1/3} \qquad (1)$$

Q is the proportion of the population who dies within period of length t, and n is an estimate of the total population. The index has been applied in a variety of contexts but its magnitude is dependent on and varies substantially with the selection of the period t. Further, the exponents in the formula are parameters whose values have been fixed for convenience when a more suitable procedure would have them estimated directly from the data.

b) Dupaquier's index.

This closely resembles a normalizing measure in that it relates the number of deaths within a year (or month or other time interval that the researcher may find suitable) to an average (' normal') number of deaths and to the average deviation of the number of deaths from their mean during such periods. The index is defined as follows:

$$II = (M(t) - U)/S \qquad (2)$$

where M(t) is the number of deaths in the year (or time interval) t, U is the mean number of deaths in the 10 preceding years and S their standard deviation. Dupaquier suggested classifying the intensity of crises depending on whether or not the value of index II fell within one of several ranges that were a priori defined. Unlike index I, index II does not incorporate a subjective dimension. The only 'subjective' item here is the selection of the number of years for the period of reference. It is likely that the value of the index in any one case varies considerably depending on the choice of the reference period, e.g. the time interval for which U is calculated.

An alternative to index II is to fit a functional form to a subset of deaths observed within a period. The intervals of crises are then determined in much the same way as one would identify conspicuous residuals in general linear models (Madai,

1979; Guttman, 1980). However, this procedure also has limitations, however. First, the choice of functional form is not trivial, although for most purposes a linear form would suffice. Second, the selection of the interval of time within which the function is fitted may have important consequences for the identification of large deviations. Finally, identification of large residuals is closely dependent on the method used to estimate the functional form. Least squares techniques are too sensitive to outliers and hence do not permit a clear-cut differentiation between normal and non-normal periods. In most cases, it would be preferable to use one of a variety of resistant procedures that are robust to outliers. For example, one could first divide the set of observations corresponding to a period of say n years into two groups. Then one could estimate the slope and intercept of a line using the median values in the two groups. Finally, the deviation of each observed value from the estimated line yields a measure of its closeness to a 'normal' year.

The various modifications suggested to these procedures attenuate the influence of random fluctuations and of the levels of mortality created by the crisis itself. Although their degree of success is somewhat different, all of these procedures are quite efficient in identifying the years of crisis. But none of them is good enough to actually measure the actual intensity of the crisis. Using 800 simulated populations experiencing crises

with different frequencies, durations and intensity during a period of 200 years (see Section V), it was found that Dupaquier's index, the index based on fitting a line through ordinary least squares and, lastly, the one based on fitting a line using resistant procedures successfully identified the crisis at least 8 out of 10 times. Failures occurred more often in simulations combining a pattern of crises with the highest rate of occurrence and the longest durations. However, in only a handful of cases (1 out of 80) was any index successful in estimating the intensity of the crisis (measured by the excess of crude death rates in relation to a normal year). In most cases there was a downward bias in the estimate of mortality excesses. This occurs because none of the procedures takes into account the reduction in the crude death rates that occurs as a consequence of lower birth rates immediately before and during a crisis. It should be noted that their performance in this regard would have been worse had the simulations incorporated the effects of heterogeneity changes in the age structure on the indicators of mortality.

The foregoing limitation is all the more relevant when estimates of excess deaths are obtained from two population censuses bracketing the period during which the crisis occurred. In this case, however, the estimated excess are too high (rather than too low) since the estimated overmortality assumes unchanged fertility and mortality during the period of crisis. The

sensitivity of these estimates to the choice of assumptions about normal mortality and fertility is too high to place any confidence in them (Anderson and Silver, 1985). However, if supported by careful internal consistency checks, they may result in good approximations (Kane, 1984; Ashton et al., 1985).

c) Indeces of replacement of population lost.

An alternative to conventional indeces of intensity of crisis is to incorporate mortality jointly with a measure of the capability of the population to recover the losses. This suggestion was first made in connection with the study of crisis in Toscana (Del Panta and Livi-Bacci, 1979) but has not received much attention. I present here a pair of elaborations and extensions of the original measure.

Assume that normal female fertility rate in the age interval x,x+dx, is given by f(x)dx, that maximum potential fertility in the small interval x, x+dx is given by (M * f(x)dx), and that the normal probabilities of surviving from birth to exact age x are p(x). The net reproduction rate is then given by:

$$NRR = \int_{o}^{\beta} p(x)\, f(x)\, dx \qquad (3)$$

If a crisis of a one-year duration increases the force of mortality at age x, x+dx by a fraction γ, then the cohort aged x at the time of the crisis will require an increase F in fertility

to restore the pre-crisis value of the ratio between generations. F is calculated as:

$$F = (S(x))^{-\gamma} \qquad (4)$$

where S(x) is the probability of surviving from age x-1 to x. If the population's normal rates of increase hover around zero, the value of F is that required to guarantee replacement of the cohort affected by the crisis. Panel a of Table 4 shows the values of F when the cohort considered is the one born during the year preceding the crisis (x=1). The values are high only for crises with intensity representing doubling or quadrupling of pre-crisis mortality levels (γ=1,3). In none of the contemporary documented crises has the intensity reached such levels. All other values of F are easily attained through post-crisis rebounds of the levels of fertility. Naturally, these values are harder to be attained by populations that have higher levels of fertility, e.g., their pre-crisis levels of fertility are closer to a maximum. The ratios of F to the maximum possible increases are displayed for a set of combinations of fertility and mortality regimes in panel b of

Table 4.

Assume now that a cohort just born is exposed more or less continously to excess mortality due to crises during its first fifteen years of life and that the average increase in the force of mortality between ages 0 and 15 (the beginning of the reproductive period) is γ, then F is calculated as:

$$F = (p(15))^{-\gamma} \qquad (5)$$

where for simplicity I have assumed that no mortality increases occur above age 15. To evaluate the implications of (5) from a somewhat different angle than (4), I have calculated the minimum increase in mortality that would be necessary for the cohort to be unable to replace itself. This will occur whenever the fertility multiplying factor,F, is higher than the minimum value that would be required to bring the current fertility up to the maximumn fertility observed in human societies (corresponding to a GRR of about 7.5). Table 5 displays some results for selected combinations of fertility and mortality regimes. In a society with high fertility (GRR=4.00) and high mortality (Eo= 35), the crises to which the cohort is exposed up to age 15 should lead to average

increases of mortality equivalent to 4.25 times the levels prevailing in normal times for replacement to be unattainable. In a society with high fertility but lower mortality (Eo=50), the crisis should increase mortality eightfold for replacement to be impossible. Note that the dimensions of the crises would have to be trully catastrophic for the affected cohorts to be unable to replace themselves. In fact, on average it would require that only a 17% of the original cohort attained age 15. Just from this information alone one may conclude that unless they are recurrent and severe, population crises are not the most efficient devices to check population growth.

A diagnostic of the severity of the crisis using the above described measure is, of course, strongly dependent on what one considers to be the maximum reproductive potential of the population. Since the latter is not unique, this measure of a crisis's intensity will vary due to factors other than those strictly related to the intensity of the crisis.

V. MEASURING THE IMPACT OF A POPULATION CRISIS.

1. Existing approaches to measurement.

An interesting question that one may pose is whether or not population crises can be taken as effective checks on population growth. This, after all, is the assumption that was at the core of Malthus's catalogue of positive checks. It also appears from time to time in contemporary debates as a rather strategic, if

seldom documented, premise. The answer to the question depends largely on the measurement instruments designed to assess quantitatively the magnitude of the effects of a crisis. I will review three different strategies, each having merits and disadvantages. It turns out that the underlying model for determining how crises are generated is essential for drawing conclusions. Quantitative assessments that rest on a static conception of crises lead to the conclusion that crises cannot be seriously taken as efficient checks on population growth. A model that explicitly accounts for periodicity, intensity and duration leads one to expect a considerable reduction of population pressure in the medium to long term. The degree to which crises actually check population growth depends on a stochastic mechanism which simultaneously determines their rate of occurrence, intensity and duration.

i) Calculation of the time for recovery.

In a series of papers (Watkins and Van de Walle, 1983; Watkins and Menken, 1985; Watkins, 1985) it is shown that a population with characteristics similar to those of pre-industrial societies will take only a short time to reach the pre-crisis population size after having experienced a crisis of high intensity. In fact, the time for recovery has been calculated to be between 6 and 50 years depending on the initial rate of growth and the severity of the crisis. This range is considered to

be sufficiently low to make only a minor dent in the population pressure experienced by the society. However, equally compelling is the calculation of the relative sizes of two populations, one experiencing and one not experiencing crises. Using the same data presented by Watkins (Watkins, 1985) I calculated that the ratio of the absolute size of the latter to that of the former twenty years after the crisis would be 1.06 if the crisis were minor and 1.26 if the crisis were major. The ratios would be 1.08 and 1.32 respectively 95 years after the crisis. These figures are invariant with the original rates of growth of the population. The slowdown of growth implied by these ratios is relevant if the initial size of the population--the level achieved immediately before the crisis strikes-- is below sustaining capacity. Calculations based on simulations to be presented later show that if a crisis of major proportions took place, on the average, every 30 to 40 years, the size of the population unaffected by crises could become 9.0 times larger than the expected size of a population subject to crises in the relatively short interval of 200 years. This is hardly a token reduction in population growth. Thus, in addition to the calculation of the time to recovery, one needs to consider the population growth that was foregone as a consequence of the crisis. The only case in which the latter is irrelevant is when the initial size of the population was already the equilibrium size.

ii) The foregone population growth under stability.

The suggestion stated before can be better and more elegantly implemented using some regularities that hold in stable populations. These have been used elsewhere to study the long-term impact of outmigration (Keyfitz, 1977), and have been applied to examine the long term impact of some types of population crises (LeBras, 1969; Salvini, 1977). It is well known that under conditions experienced by contemporary developing societies, a policy of year-after-year outmigration would require that about 30% of the population at the peak ages of childbearing migrate in order to keep the population from growing at more than .02 per year. The requisite outflow is evidently higher if the target was to keep the population at stationary levels. This suggests from the start that a one-time shock induced by a population crisis will be even less effective to check population growth in the absence of reductions of fertility. In fact, if the losses of population due to a crisis were equivalent to a fraction D of all members between the ages 0 - 50, the ratio of the ultimate population (affected by the crisis) to the ultimate population (not affected by the crisis) would be 1-D. The contribution of each age in the range 0-50 to this total loss t years after the crisis is given by the following quantity:

$$(D/v) * N(x) * v(x) * \exp(r*t)/b \qquad (6)$$

where v is the mean age at childbearing in the stable population, N(x) is the absolute number of the population aged x at the time the crisis strikes, v(x) is the reproductive value at age x, r is the rate of growth and b the intrinsic birth rate. The values of r,b, and v(x) are those associated with the patterns of mortality and fertility in the pre-and post-crisis situation. Since v(x) peaks at ages 15 or so, the losses experienced at this age weigh more heavily on the calculation of the ultimate losses due to the population crisis. Thus, crises in which the population 0-15 is more affected are the ones with the highest potential effectivness for reducing ultimate population growth.
In order to generate a standard formula for the calculation of ultimate losses to be attributed to excess mortality in the most strategic age group, 0-15, I derived the following formula:

$$\text{Ultimate Loss} = j* \exp(r*t)/(b*v) * C(15)* B(15) \qquad (7)$$

where j is the factor by which the crisis increases mortality (will equal 1 if mortality is doubled, 2 if mortality is tripled etc.), r, b, and v are defined as before, C(15) is a weighted average of the reproductive values within the age range 0-15, and B(15) is the number of deaths that would take place in the

population in the ages 0-15 exposed to the crisis if subjected to the pre-crisis force of mortality. The weights are given by:

$$w(x) = (N(x) * \mu(x)) \div \left(\int_0^{15} N(x)\, \mu(x)\, dx \right) \qquad (8)$$

where $\mu(x)$ is the force of mortality at x in the pre-crisis pattern of mortality and N(x) is defined as before. Formulae (7) and (8) are valid if mortality in the age segment 0-15 follows a Gompertz curve. The latter has been shown to be a quite reasonable approximation to a disparate set of mortality patterns. To illustrate the magnitudes attained by (7), I have assumed a stable population with a rate of growth of .01 per year and a life expectancy of about 50.00 (implying a crude birth rate equal to .029) and a mean age at childbearing of 26. If the crisis lasts between 1 and 2 years and mortality increases to a level two times as high as the pre-crisis levels, the quantity B(15) is about 24 per thousand of the original population, and the value of C(15) is 1.50. Thus the amount of the loss after 20 years is close to 58.32 per thousand of the original population. After 50 years the loss is in the neighborhood of 79 per thousand of the original population. Since in these calculations I have not accounted for the post-crisis upswing in births, the figures

presented should be taken as upper limits. As they stand, however, they do not appear to be insignificant. They are perhaps small enough to shatter the idea that a crisis may be any more effective than outmigration to keep populations from growing above certain targets. However, they are significant enough to make of recurrent crisis if not a court of last resort at least a highly disruptive occurrence in the trajectory of a population.

2. An integrated framework for measuring the impact of a crisis.

The two alternative forms of measurement presented above rest on models of generation of crises that disregard : a) the repetitive nature of crises and b) the trajectory of vital events that result from a crisis (see Section III). To assess the quantitative impact of crises, it is not sufficient to follow the consequences of ONE of them in the absence of any repetitions. Recurrence of crises is a fundamental characteristic of societies that have been vulnerable to them. Similarly, full and accurate estimation of the impact of a regime of crises requires appropriate modeling of the trajectory of vital events. As indicated in Section III, these trajectories depend on the nature of the crises themselves. In order to eliminate these two deficiencies, I proceeded to simulate the trajectories of several populations exposed to several regimes of fertility, mortality, migration and crises. As indicated below, the parameters for each of these regimes were estimated from known records of population

crises in the pre-industrial period and during the XXth century. The results that I obtain indicate that some crisis regimes that are likely to affect contemporaneous populations can be devastating if sustained for relatively short periods of time. The simulation models provide a more complete and richer picture than models resting on the assumption of a single shock.

a) A random mechanism for the occurrence of crises.

To incorporate the possibility of multiple crises in an interval of time, it is necessary to postulate a mechanism of recurrence. In the absence of any theory about it, I assumed that crises are generated by a Poisson process. Correspondingly, the intercrises waiting times are independent and exponentially distributed. Estimates of the rate for the exponential distribution were obtained from several historical records of crises (Charbonneau and Larose, 1979; Del Panta, 1980) and from summaries of crises in contemporary developing countries (See Appendix). Although it would have been possible to estimate type and country (region)-period specific rates, I thought it sufficient to estimate a range. A low frequency of crises corresponds to a regime with a rate equal to 30.00 per thousand years (or a waiting time for a crisis equal to 33 years). A regime with high frequency is characterized by a rate equal to 100 per thousand years (or a waiting time for a crisis equal to 10 years). It should be noted that the low bound is by all accounts too low

to represent the regime of crises in the pre-industrial period. On the other hand, the upper bound is too low to represent the experience of some developing countries such as Bangladesh during what has elapsed of the XXth century. Thus, the range used here is quite conservative for applications to both pre-industrial societies and contemporary developing societies.

b) Definition of types of crises.

A regime of crises was characterized by two variables. The first
was the duration and the second the intensity. The duration of a crisis was arbitrarily set equal to 2 or 5 years. This was done to make results comparable with those obtained by other researchers. There was no compelling reason to select either of them although it could have been possible to estimate empirically the average durations by type of crisis and by country (region) and historical period. However, this elaboration is superfluous in the absence of a theory linking type of crisis and durations.

The intensity of a crisis depends on two correlated components: the levels of over-mortality and the levels of fertility reduction. I assumed that minor crises led to minor reactions in both fertility and mortality and that major crises led to major changes in both fertility and mortality. Post-crisis mortality was assumed to go no lower than pre-crisis levels of mortality. Thus, I disregard completely the effects of selectivity. Post-crisis

levels of fertility were characterized by a one-year rebound that took them to levels higher than during the pre-crisis period. Consistent with the discussion in Section III, the levels of mortality and fertility for the year immediately preceding the crisis are subject to a mild increase and decrease respectively. The magnitude of these changes depends on the intensity of the crisis.

Age-specific fertility is affected by the same proportionate amount so that a crisis does not bring about a change in the age-pattern of fertility. A high intensity crisis led to a decline of GRR of about 50% and a rebound of about 20% for the year following the end of the crisis. During the year before the onset of crisis levels, fertility declines gradually to 75% of its pre-crisis level, to 63% of its original value within the first year of the crisis, and is halved for a period beginning nine months after the onset of the crisis until the end of the crisis. A low intensity crisis leads to an overall decrease of 10% in the GRR and to a post-crisis rebound of 5 %.

Over-mortality was assumed to follow a pattern by age that was obtained as an average of various experiences in the pre-industrial period and during the XXth century; increases are heavier among younger children and older people. The pattern is displayed in the Appendix. A high intensity crisis was defined as one that, given the age pattern of over-mortality, would increase

the CDR by about 80% in a population with a GRR equal to 3.0 and a life expectancy equal to 50. The increase would take place starting one year before the onset of the crisis--during which the levels of mortality are increased by an amount equivalent to 15% of the CDR--and would end at the end of the duration of the crisis. A low intensity crisis leads to increases in the force of mortality that, given the age pattern of over-mortality, translates into an increase of the CDR of about 15% in a population with the same characteristics mentioned above.

The values given to the parameters in the cases of highest and lowest intensity are bracketed by those observed in the following cases: China (1950-1960), Bangladesh (1971-1975), Finland(1750-1900), Toscana (XVth century), and various other regions and countries experiences recorded by Charbonneau and Larose. The only feature that I have left out is migration which can be approximately captured by increasing the impact of mortality. This is an approximation inasmuch as the age pattern and the timing of outmigration are not necessarily the same as those of over-mortality.

c) Simulation of crises.

Two initial populations were selected. The first was nearly stationary, having a life expectancy of about 27.5 (Model East) and a GRR equal to 2.50. The second had a life expectancy equal to 50 (Model West) and a GRR equal to 3.0, with an intrinsic rate

of growth of .027. The first population resembles the Chinese farmers studied by Barclay and the second one is very similar to Bangladesh during 1976-1979. Having selected an initial population and a crisis regime a pseudo-random number generator was utilized to determine the timing of crises within a period of 200 years. In all cases, a crisis at the beginning of the projection period was imposed. Randomness entered in the determination of all crises following the first one. The population was then projected forward year by year for a period of 200 years. The simulation process was repeated 100 times and then a new crisis regime was selected and applied in analogous fashion. Altogether 800 simulations were carried out with the second initial population and 400 with the first initial population.

d) Main results.

Tables 6 and 7 summarize selected results for both initial populations. Each Table displays the expected population in four different crisis regimes, the population experiencing only one crisis and the population experiencing no crisis after 10, 50, 100, 150 and 200 from the initiation of the projection. The crisis regimes were chosen to provide the widest range of possible results. In each of the regimes dependent on random mechanisms, I have used the expected population (average over 100 simulations) even though the corresponding figures represent no particular experience. To enhance the reliability of the figures, I also show

the estimated standard deviations.

Figures 6a through 6d show the trajectory of crude rates and populations in selected regimes of crises. Expected rates and populations are shown and each series is enclosed within a band determined by one standard deviation on each side of the expected values. These figures should be compared with Figures 3,4 and 5.

The most striking feature of Tables 6 and 7 is the divergence between the trajectories of the randomized crises and the one corresponding to the one crisis, one shock regime. Notice that the combination of an initially stationary population with a relatively severe crisis regime, leads to population extinction. Here, there is no place for the calculation of time to recovery. This is a result that would simply not obtain in a one crisis, one shock regime.In other, less severe cases, the crisis regime leads to losses amounting to 32% of the size achieved at the end of 200 years by the population experiencing only one crisis and 40% of the size achieved in the absence of crisis. Naturally, the potential for recovery is much greater in the population initially growing at a fast rate (Table 7). Although severe crisis regimes do not lead here to extinction they imply massive losses in a relatively short time. Note that after 200 years the ratio of absolute sizes in the population experiencing no crisis to the populations affected by the two most serious regimes are 4 and 9 (first and second columns). Even the less severe crisis regimes

produce losses amounting to at least 10% of the final size in the society free of crisis. In the short run (first 100 years) the differentials are greater in regimes with less severe crisis and smaller in regimes with high intensity crises. Thus, even with populations growing rapidly, a randomized crisis regime inflicts losses that cannot be gauged using a one crisis model. This holds regardless of the time horizon that one selects to use for the projection exercise.

Table 8 displays information on the ratio of the expected populations in various crisis regimes to the population in the absence of crises at τ=100 and τ=200 years after the initiation of the projection. The information is represented in terms equivalent to rates of growth: the figures are the values of the difference between the rates of growth in two populations that would lead to the observed ratios after 100 and 200 years. For example, at the end of 100 years a population exposed to no crises is expected to be about 1.96 times larger than a population with frequent crises of high intensity but short durations. This is equivalent to having two populations growing at rates differing by about .0067 (or 24.2% of the initial rate of growth). Table 8 only shows results for the fast growing initial population. Note that the differences are substantial. Randomized crisis regimes lead to larger differentials than the one crisis-one shock model regardless of the type of crisis. Also, randomized crisis regimes

are strongly responsive to changes in intensity and frequency and less so to changes in duration. However, there are powerful interaction effects. Thus, shifting frequencies has a larger impact when the crises are long than when they are short, and when they are of high rather than low intensity.

All in all, these results confirm the conjecture made at the outset: provided that crises are recurrent, even a rather benign regime would end up producing substantial losses of total population. Under certain conditions, recurrent crises lead to long term extinction; under more general conditions, a pattern of population crisis can provide significant relief from excessive growth, much more than would be implied in the one crisis-one shock model.

e) Limitations of the simulations.

The assumptions of independence of successive crises, the lack of built-in correlation between duration and types of crises, the absence of combination of different types of crisis within the same projection routine, are all pitfalls that make the procedure implemented here a somewhat inaccurate and unrealistic one. However, it provides a better assessment of the quantitative impact of crises than those that rest on the assumption of only one crisis during the period over which calculations are carried out. Further refinements of this procedure are possible. Moreover, the procedure can be connected with the formal theory of

populations subjected to cyclical fluctuations (Frauenthal and Swick, 1983; Cohen, 1976; 1977).

There are, however limitations that go beyond those due to lack of acceptable measurement. In fact, the simulations are carried out in a context where neither social differentiation nor regional inequalities are taken into account. Crises do not affect equally all members of the population. A realistic representation of the effects of population crises should separately take into account and predict the differential impact on the various social strata. Insofar as social groups are endowed with different resources, the balance between demographic and non-demographic responses will vary from case to case. Only in very extreme cases will all groups be affected equally. Neglecting social heterogeneity inevitably creates distortions that are masked by the general character of population projections.

Most crises are local in that they affect certain geographic areas rather than entire nations. This effectively limits their scope and their potential impact if only because exchanges (of persons or resources) between regions will enter as an adaptive response (Caldwell, 1973). The projection exercise completely neglected this, except for the allowance made for outmigration and return migration. Thus, the projections portray an artificial state of affairs since they are only valid within a bounded (and perhaps small) region and only when the crisis is allowed to run

its course, without the beneficial influence of outside relief. Since social and geographical heterogeneity are pervasive, the results of a simulation exercise that has neglected them should only be used to gain a rough sense of the magnitudes of effects involved.

3. Other consequences of population crises.

An intriguing idea emerges from the combined studies of population crises and that of the conditions sustaining high levels of fertility (Bongaarts and Cain, 1981; Caldwell et al, 1984). The idea is that, in contrast to what has been conventionally thought, recurrent crises may lead to the development of mechanisms that support high fertility as a form of risk avoidance. This should occur whenever institutionalized systems to minimize risks, such as social security, health services, transportation networks, are absent. If this were in fact the case, the ocurrence of crises itself reinforces sustained growth in the aftermath of the crisis. This is a paradoxical consequence of the positive checks. The relation between fertility and crisis will of course vary across subgroups in the population and will depend on what the latter's position is in terms of capabilities to minimize risks.

This conjecture can be neatly connected with Sen's hypothesis about entitlements to yield predictions about what subgroups in the population are subject to higher risks and hence

to potentially higher fertility and aversive behavior toward fertility limitations. By the same token, since a crisis rarely leaves untouched the structure of families and households and since the latter are the fabric of rules of couples' behavior, the study of fertility cannot overlook the frequency and intensity of crises, quite apart from the impact that these may have on the size of a population. In fact, the study of one cannot be carried out without an understanding of the mechanisms determining the other. From this point of view the analysis of population crises is far from an esoteric exercise. As it occurs with the study of physical materials subjected to extreme conditions, the analysis of population crises should reveal the vulnerability of a society's adaptive response and of its resilience.

CONCLUSION

This paper has reviewed the state of the art in the analysis of population crises. I showed that there are models that guide the understanding of a crisis's origin and development. These models permit usto formulate typical trajectories for the most important vital phenomena. This provides an opportunity for indirect measurement insofar as hypothetical trajectories could be contrasted with observed data to infer characteristics about the crisis.

Although affected by numerous limitations, alternative

measurement techniques to quantify the intensity of crises have been developed; they should be used to create a map of their frequency and chronology. In addition, available methods allow us with variable success, to quantify the magnitude of the impact of a crisis and assess their role as checks on population growth. These methods can be designed to represent the stochastic nature of the process and preserve its recurrent character. These are essential characteristics of crisis regimes which, if neglected, can lead to misleading conclusions. Thus, I showed that a regime with randomly generated crises can lead to significant losses that would not have been discovered by more conservative modeling. Extensions of these designs may be possible if they are associated with conventional advances in modeling of populations subject to cyclical fluctuations.

The study of crises is inseparable from the study of fertility and family and household structures: not only do population crises affect family organization but the latter may be designed to minimize the expected losses in high risk situations leading to recurrent crises. Thus, population crises ought to receive more than the token attention paid to them by those who use them to create or exorcise the evils of population growth.

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Appendix 1: Basic information used to construct patterns of fertility and mortality during crises.

a) Pattern of overmortality.

The following is the pattern of overmortality expressed as ratios of increases in the age group 0-1.

Age	Used	Finland 1832-33	Finland 1867-68	Bangladesh 1971	London Plague 1603
0	1.00	1.00	1.00	1.00	1.00
1-4	3.74	3.03	2.27	2.26	4.34
5-9	3.94	4.89	3.78	12.01	15.02
10-14	3.82	3.83	3.47	1.85	16.30
15-44	1.45	2.39	2.62	1.35	6.20
45-64	1.66	1.64	2.31	1.93	1.44
65+	1.91	.39	1.00	3.09	.37

The recorded percentage increases in CDR used as references are the following:

Bangladesh	(War 1971-72)	28%
Bangladesh	(Famine 1974)	24%
China	(Famine 1959-62)	70%
Finland	(Famine 1867-68)	158%

These percentages were calculated using as baseline the overage of five years preceding the crisis (excluding observations corresponding to other crises).

The entire set of vital rates on which the construction of figure 5 is based appears at the end of this appendix. Its sources are Turpeinen (1979) for Finland; Curlin et. al (1971); Chen and Chowdhury (1977); Miranda (1982) for Bangladesh, and Charbonneau and Larose (1979) for London.

b) Pattern of decline in fertility

The average percentage decrease in TFR for China, Bangladesh and Finland during the various crisis are 48, 33 and 28 respectively. The rebound over the year following the crisis characterized by increases of 19% and 5% in China and Finland respectively. The sources for these figures are the same as in (a).

c) Frequency of crises

i) Note that in China there have been 2 documented crises over a period of seventy years and that in Bangladesh there have been 3 over the same period. If no other crises occur, these frequencies represent rates of 20 o/oo years and 30 o/oo years.

Appendix 1 (continued)

ii) Del Panta (1980) documents rates in a range of 13.3 o/oo - 100 o/oo in Italy as a whole and of 40.8 o/oo - 218 o/oo in Toscama during the XVIth century.

iii) Charbonneau and Larose (1979) document a very broad set of frequency rates for various periods and geographical regions. The average that could be established for crisis of medium intensity in 33 o/oo. However, the range of frequency rates that they can document in 24 o/oo - 230 o/oo for crises of low to high (but not highest) intensity.

Appendix 2: Data to construct Figure 5

China	CBR	CDR	TFR	Infant Mortality Rate
1955	32.6	12.3	6.3	177
56	31.9	11.4	5.9	167
57	34.0	10.8	6.4	156
58	29.2	12.0	5.7	144
59	24.8	14.6	4.3	236
60	20.9	25.4	4.0	205
61	18.0	14.2	3.3	132
62	37.0	10.0	6.0	236
63	43.4	10.0	7.5	88
64	39.1	11.5	6.2	96

Finland	CBR	CDR	TFR	Infant Mortality Rate
	N			
1863	O	29.8	4.8	211
64	T	22.6	5.3	144
65		25.9	4.8	193
66	A	33.6	4.5	217
67	V	38.1	4.5	223
68	A	77.5	3.4	391
69	I	25.2	4.5	141
70	L	18.2	4.9	136
71	A	17.9	5.0	141
72	B	19.7	4.9	173
	L			
	E			

Bangladesh	CBR	CDR	TFR	Infant Mortality Rate
1966-67	47.1	15.0	6.7	110.7
1967-68	45.4	16.6	6.3	125.4
1968-69	46.6	15.0	6.3	123.8
1969-70	45.3	14.9	6.1	127.5
1970-71	43.5	14.8	5.9	131.3
1971-72	44.5	21.0	6.0	146.6
1972-73	41.8	16.2	5.5	129.2
1973-74	45.6	14.2	6.0	128.8
1974-75	40.1	20.0	5.4	167.2
1975-76	27.6	18.2	4.3	150.4

Table 1: Classification of some recorded population crisis according to selected characteristics

Event†	Triggering Event	Losses of Entitlements vs. Food Production	Outmigration relative to nuptiality/ fertility	Nature of mortality response	Presence of feedback
China, 1958-62	Drop in Grain Output (agricultural policies)	Food Production	Nuptiality/ Fertility	Age Selective	No
Bangladesh, 1970-71	War	Food Production	Both	Age Selective	Epidemics Food Production
Bangladesh, 1974-75	Flooding	Both	Nuptiality/ Fertility	Age Selective	No
Russia, 1920-39	Agricultural Policies and War	Both	Nuptiality/ Fertility	Age Selective	?
Bengal, 1943	War	Entitlements	Both	Neutral	Epidemics Food Production
India, 1870-80	Weather Patterns (drought/ flooding)	Both	Stronger Response of Nuptiality	Neutral	Several Epidemics Food Production

†Dates correspond to time period bounding the duration of the crisis.

Table 2: Relations between timing, levels and age-sex patterns of overmortality and type of crisis

Type of Crisis	Overmortality			
	Levels	Age Pattern	Sex Pattern	Most Favored in Selection
Triggered by War with no Feedback	Variable; Immediate Increases	Concentrated in 15-50	Male Excesses	Most Frail
Triggered by Relative Food Shortages	Variable; Gradual Increases to a Maximum	Group 1-10 Most Affected	Generally Female Excesses	Least Frail
Triggered by Disease but no Feedback	Variable; Timing Follows Diffusion Curve	Variable with Disease	No Differentiation	Least Frail

Table 3: Relations between timing and levels of changes of nuptiality and marital fertility and types of crisis

		Marital Fertility		
Type of Crisis	Nuptiality	Fecundity	Intercourse	Amenorrhea
Triggered by war with no feedback	Pervasive postponement	-	Affected by separation	-
Triggered by relative food shortages	Pervasive postponement	Ovulation affected by nutrition	Affected by separation in case of outmigration and by stress	Affected by reduced breastfeeding
Triggered by disease but no feedback	Minor reduction in the absence of migration	-	Affected by conditions produced by the disease itself	Affected by reduced breastfeeding

Table 4a. Proportionate increases in fertility required to maintain net reproductive rate (one year crisis, cohort born during year preceding the crisis)

Proportionate Increases in mortality due to crisis	Values of probability of surviving the first year of life		
	.800	.850	.900
.10	1.02	1.02	1.01
.50	1.12	1.08	1.05
1.00	1.25	1.18	1.18
3.00	1.95	1.63	1.63

Table 4b: Ratios of the increase in fertility necessary to maintain precrisis net reproduction rate to the maximum possible increase (GRR = 7.5) (One-year crisis, cohort born during year preceding crisis)

Proportionate increases in mortality due to crisis	Values of probability of surviving from birth to age 1.0								
	.800			.850			.900		
	NRR*			NRR*			NRR*		
	2.36	1.77	1.00	1.36	1.77	1.00	2.36	1.77	1.00
.10	.54	.41	.23	.54	.41	.23	.54	4.0	23
.50	.59	.45	.25	.58	.43	.24	.56	.42	.24
1.00	.67	.50	.28	.63	.47	.27	.63	.47	.27
3.00	1.04	.8	.44	.87	.65	.37	.87	.65	.37

* The values of NRR correspond to values of GRR equal to 4.00, 3.00, and 1.69, respectively.

Table 5: Minimum proportional increase in mortality (values of $\hat{T}$) that would require the maximum possible increase in fertility (up to GPR=7.5) to ensure replacement (cumulated effects of crisis evaluated at age 15)

		Gross Reproduction Rate	
		4.00	1.75*
Life expectancy at birth ($^0e_0$)	35	3.25	3.26
	50	7.74	7.78

*The combination GPR=1.75 and $^0e_0$=35 corresponds to a stationary population.

Table 6. Population Sizes of Initial Population with r = .0022 in Six Different Situations

Year	Regime* 1		Regime 2		Regime 3		Regime 4		One** Crisis	No Crisis
0	1000		1000		1000		1000		1000	1000
10	891	(42.9)	831	(.000)	977	(.000)	991	(7.6)	977	1017
50	715	(71.5)	629	(110.0)	989	(24.8)	1032	(21.1)	1036	1098
100	483	(52.3)	369	(151.0)	1008	(47.6)	1080	(36.1)	1130	1203
150	317	(51.1)	184	(80.7)	997	(71.6)	1133	(50.3)	1238	1318
200	220	(40.9)	84	(40.5)	1027	(86.8)	1176	(65.2)	1357	1443

* The nature of each regime is defined by a triplet where the symbols refer (in order) Low (L) or High (H) frequency, Low (L) or High (H) intensity and Short (S) or Long (L) duration. The triplets for each regime are defined as follows:

Regime 1 H,H,S
Regime 2 H,H,L
Regime 3 L,L,L
Regime 4 L,L,S

**One crisis at the beginning of the projection period lasting 5 years and having low intensity.

The values for columns (1) through (4) correspond to expected values over 100 simulations. The figures in parentheses are the standard errors.

Table 7. Population Sizes of Initial Population with r = .027 in Six Different Situations

Year	Regime* 1		Regime 2		Regime 3		Regime 4		One** Crisis	No Crisis
0	1000		1000		1000		1000		1000	1000
10	1176	(31.9)	1094	(.0001)	1246	(.0002)	1264	(8.6)	1192	1290
50	2805	(278.6)	2357	(230.1)	3735	(73.9)	3882	(82.2)	3682	4024
100	8596	(1167.3)	5615	(1242.3)	14903	(545.0)	15918	(494.7)	15368	16858
150	25722	(3490.1)	13156	(4747.1)	59496	(3098.0)	65223	(2149.3)	64368	70619
200	73356	(11106.9)	33680	(19205.0)	240699	(13960.0)	266826	(11542.0)	269645	295836

* The nature of each regime is defined by a triplet where the symbols refer (in order) Low (L) or High (H) frequency. Low (L) or High (H) intensity and Short (S) or Long (L) duration. The triplets for each regime are defined as follows:

Regime 1	H,H,S
Regime 2	H,H,L
Regime 3	L,L,L
Regime 4	L,L,S

**One crisis at the beginning of the projection period lasting 5 years and having high intensity.

The values for columns (1) through (4) correspond to expected values over 100 simulations.
The figures in parentheses are the standard errors.

Table 8. Estimated Rates of Growth consistent with the Ratio of Population with Crises to Population without Crisis at Two Values of τ

$\tau = 100$

Frequency \ Intensity	Low Intensity		High Intensity	
	Short	Long	Short	Long
Low	.0006 (2.1)	.0012 (4.5)	.0031 (11.2)	.0048 (17.2)
High	.0013 (4.7)	.0023 (8.2)	.0067 (24.2)	.0110 (39.7)

Comparison of population with only one crisis to population with no crisis:

Duration \ Intensity	High	Low
Short	.0005 (1.7)	.0001 (.4)
Long	.0009 (3.3)	.0002 (.7)

$\tau = 200$

Frequency \ Intensity	Low Intensity		High Intensity	
	Short	Long	Short	Long
Low	.0005 (1.8)	.0010 (3.7)	.0027 (10.0)	.0046 (16.6)
High	.0013 (4.7)	.0022 (7.9)	.0070 (25.3)	.0109 (39.4)

Comparison of population with only one crisis to population with no crisis:

Duration \ Intensity	High	Low
Short	.0005 (1.7)	.0001 (.4)
Long	.0009 (3.3)	.0002 (.7)

The percentage differences relative to the original rate of growth appear in parentheses (.0277).

FIGURE 1 : RELATIONS BETWEEN MAIN VARIABLES IN POPULATION CRISES

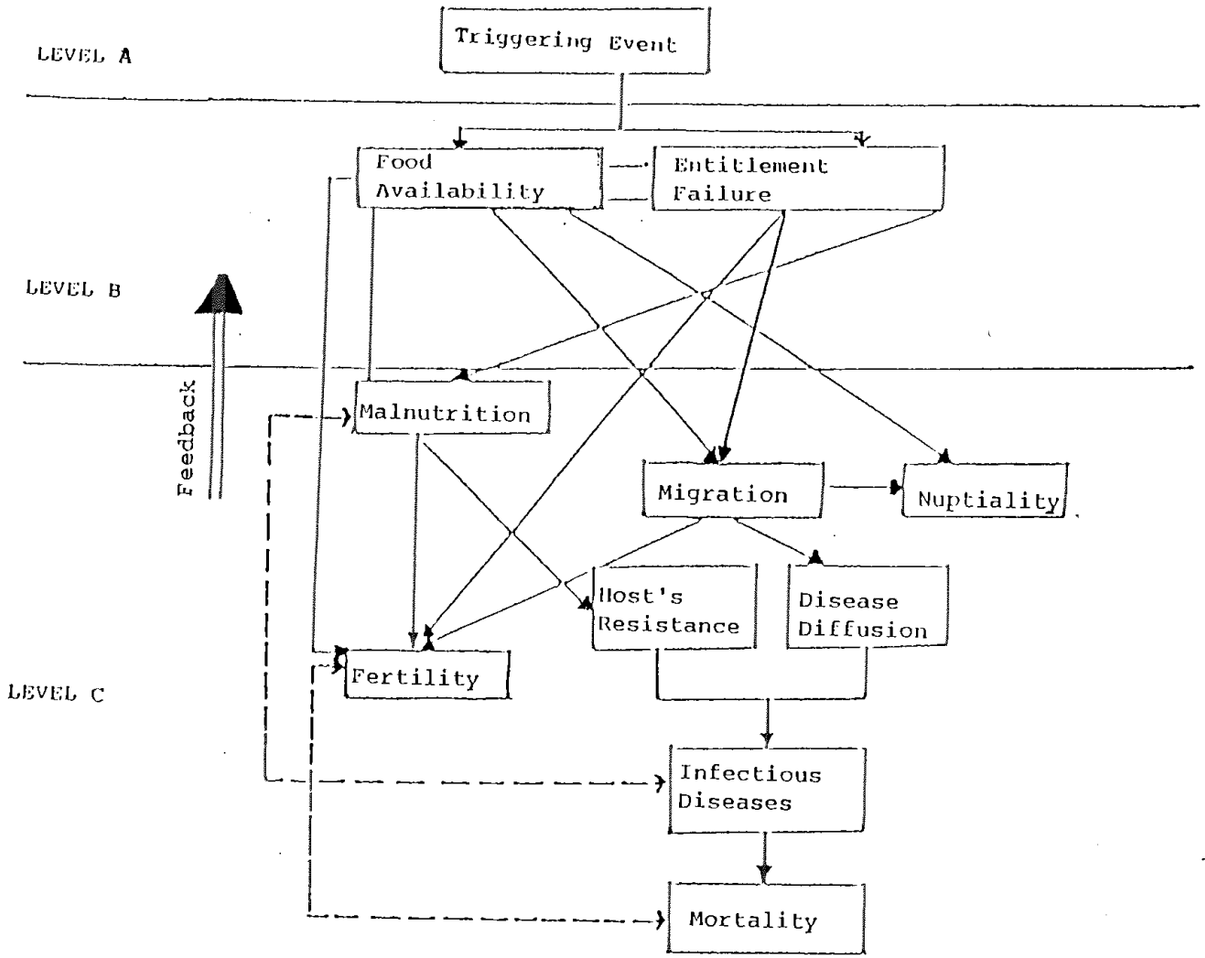
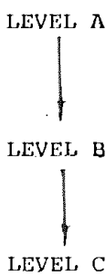


FIGURE 2 : ALTERNATIVE SEQUENCE OF EVENTS IN A CRISIS

CASE 1 : CRISIS INVOLVING NO FEEDBACK MECHANISM



CASE 2 : CRISIS INVOLVING FEEDBACK MECHANISM
ORIGIN : INFECTIOUS DISEASE

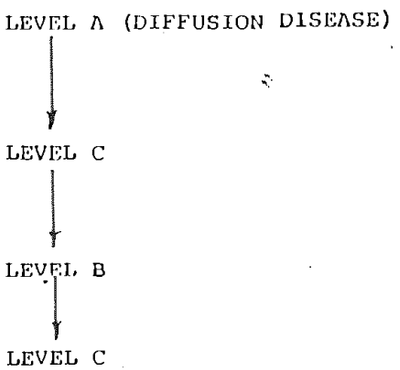


FIGURE 3 : RATIOS OF MORTALITY MEASURES DURING CRISES TO MORTALITY MEASURES DURING NORMAL PERIODS : CDR (CRUDE DEATH RATE), Q(5) (PROBABILITY OF DYING BEFORE AGE 5).

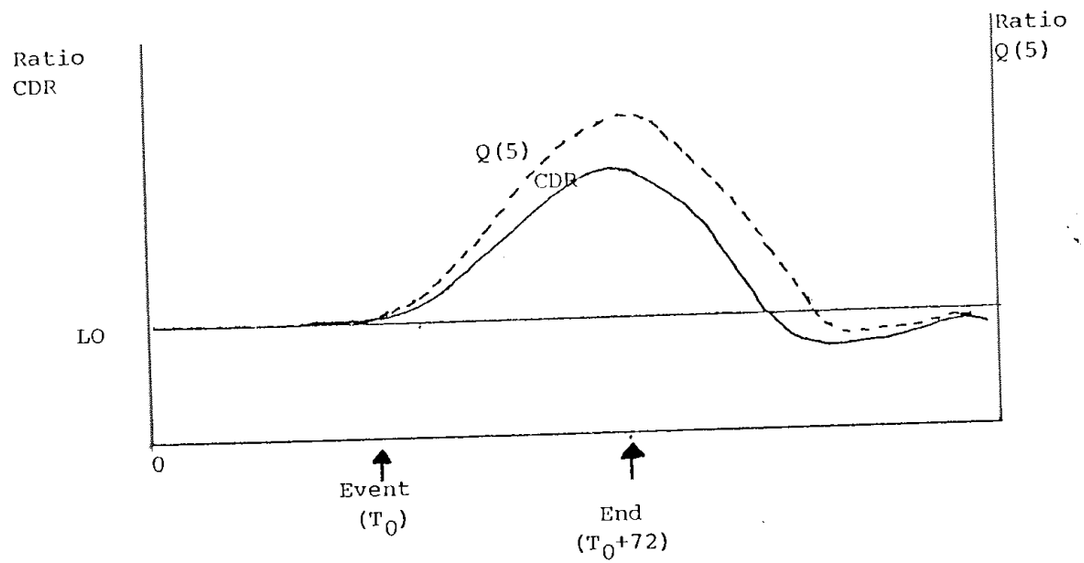


FIGURE 4 : RATIOS OF FERTILITY MEASURES DURING CRISES TO MORTALITY MEASURES DURING NORMAL PERIODS: CBR(CRUDE BIRTH RATE), TFR(TOTAL FERTILITY RATE).

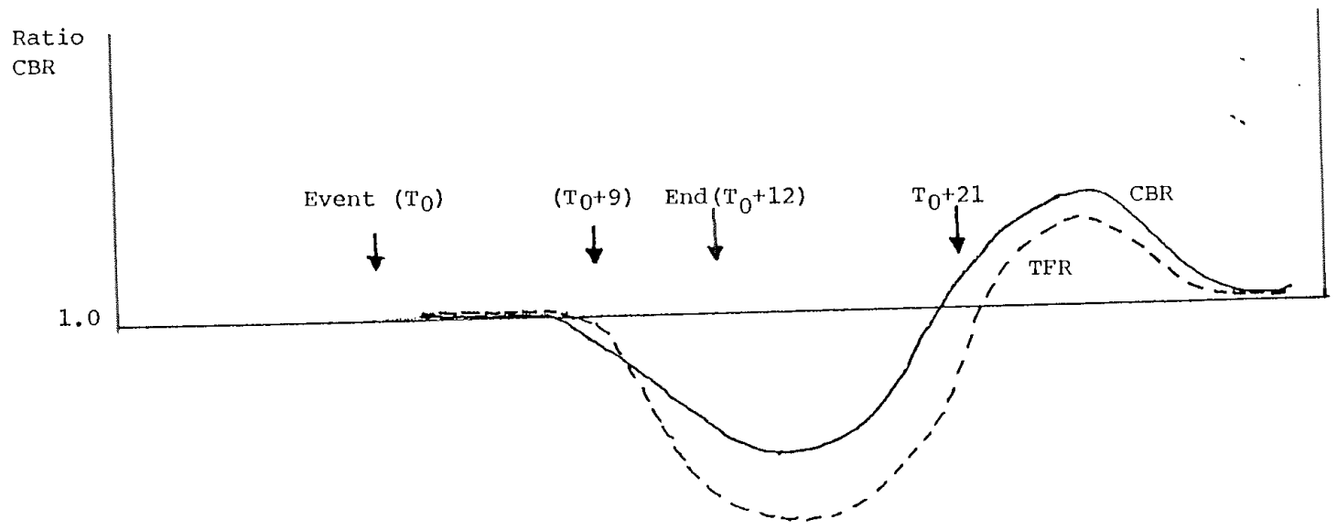
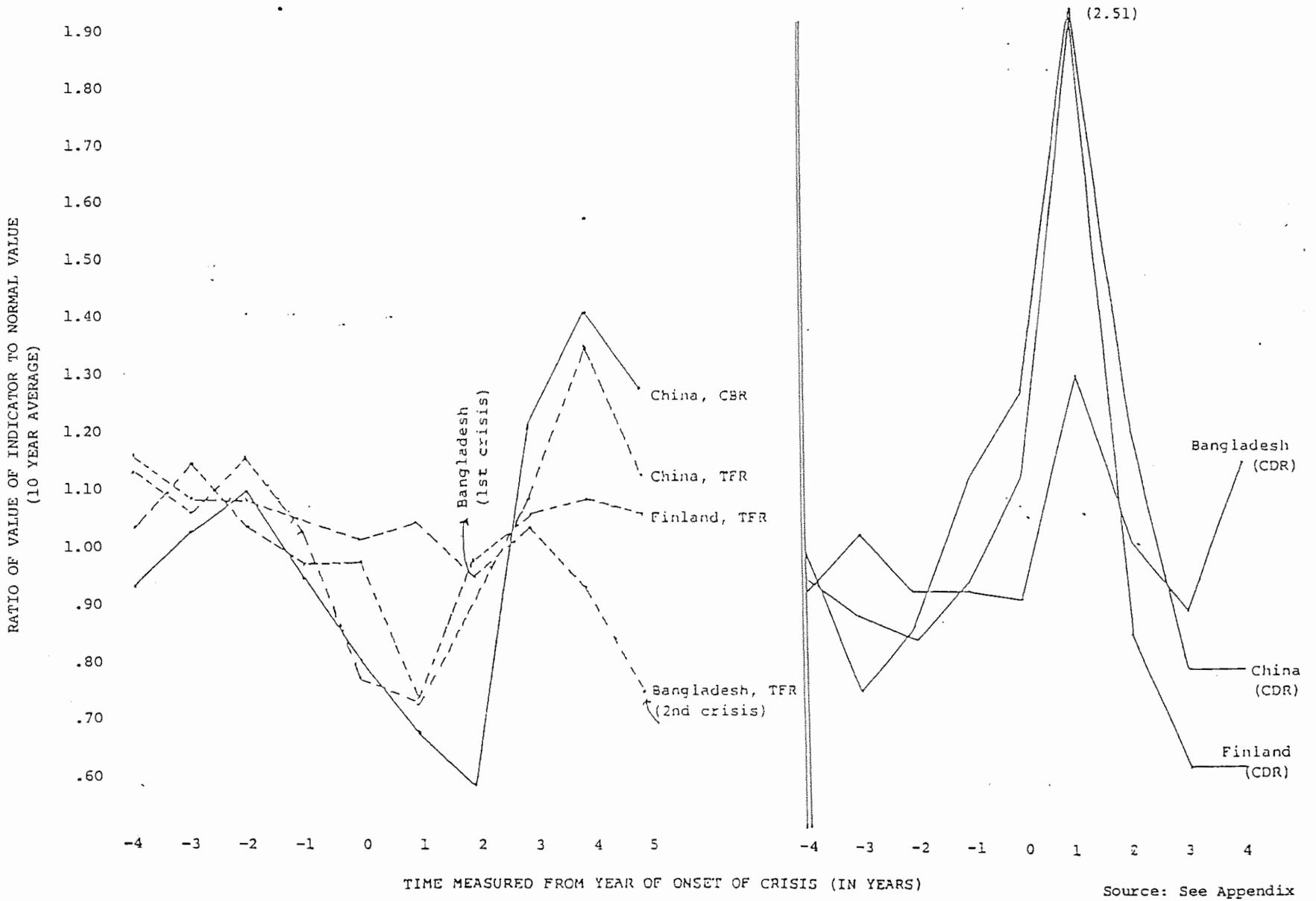
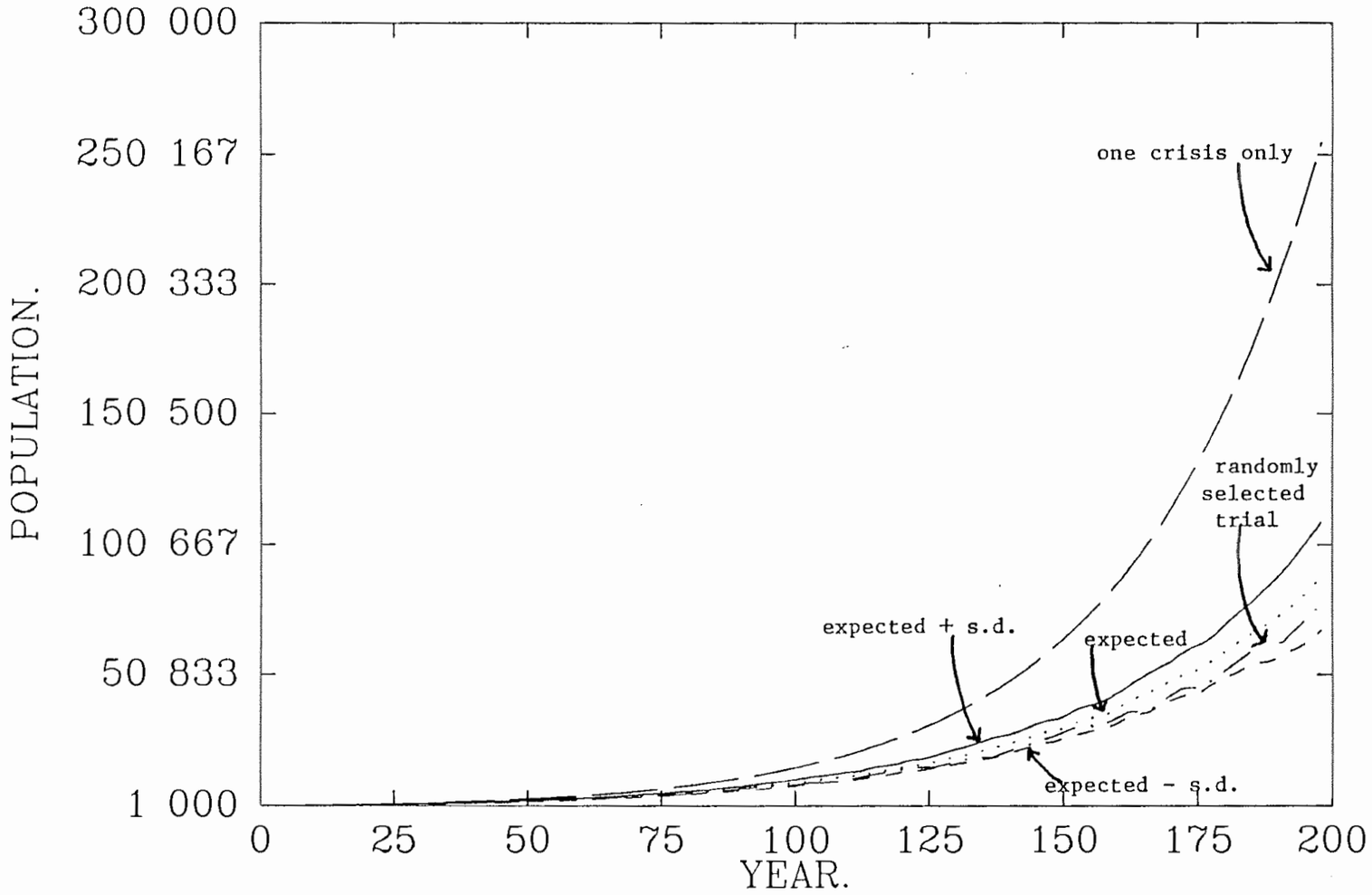


FIGURE 5 : TRAJECTORY OF DEMOGRAPHIC INDICATORS IN THREE SELECTED COUNTIES



Source: See Appendix

FIGURE 6A. PROJECTED POPULATIONS. CRISIS REGIME=HHS.
YEARS 1–200.



(Initial Population: growing at r = .027)

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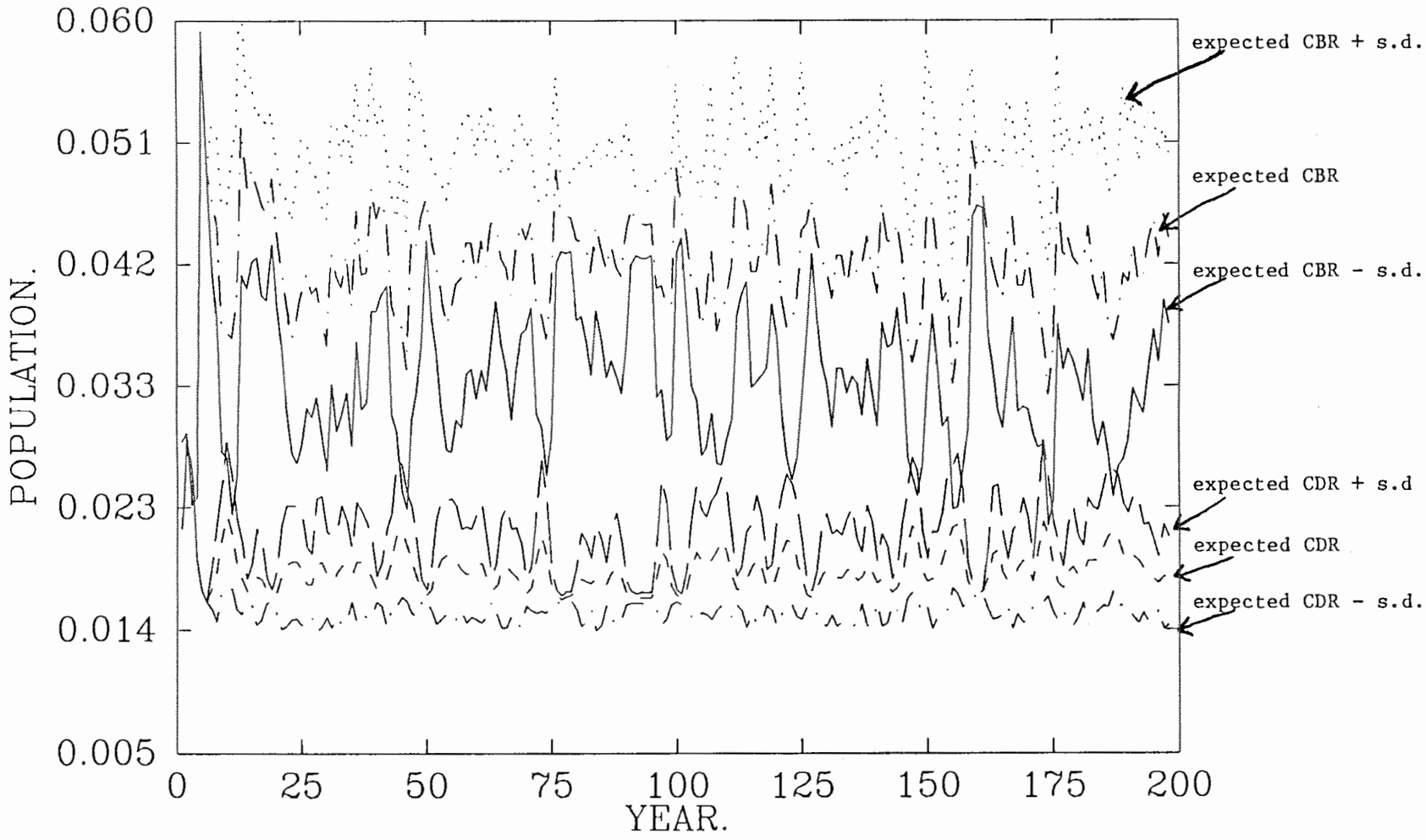
FIGURE 6B. VITAL RATES. CRISIS REGIME=HHS. YEARS 1–200.



(Initial Population: growing at r = .027)

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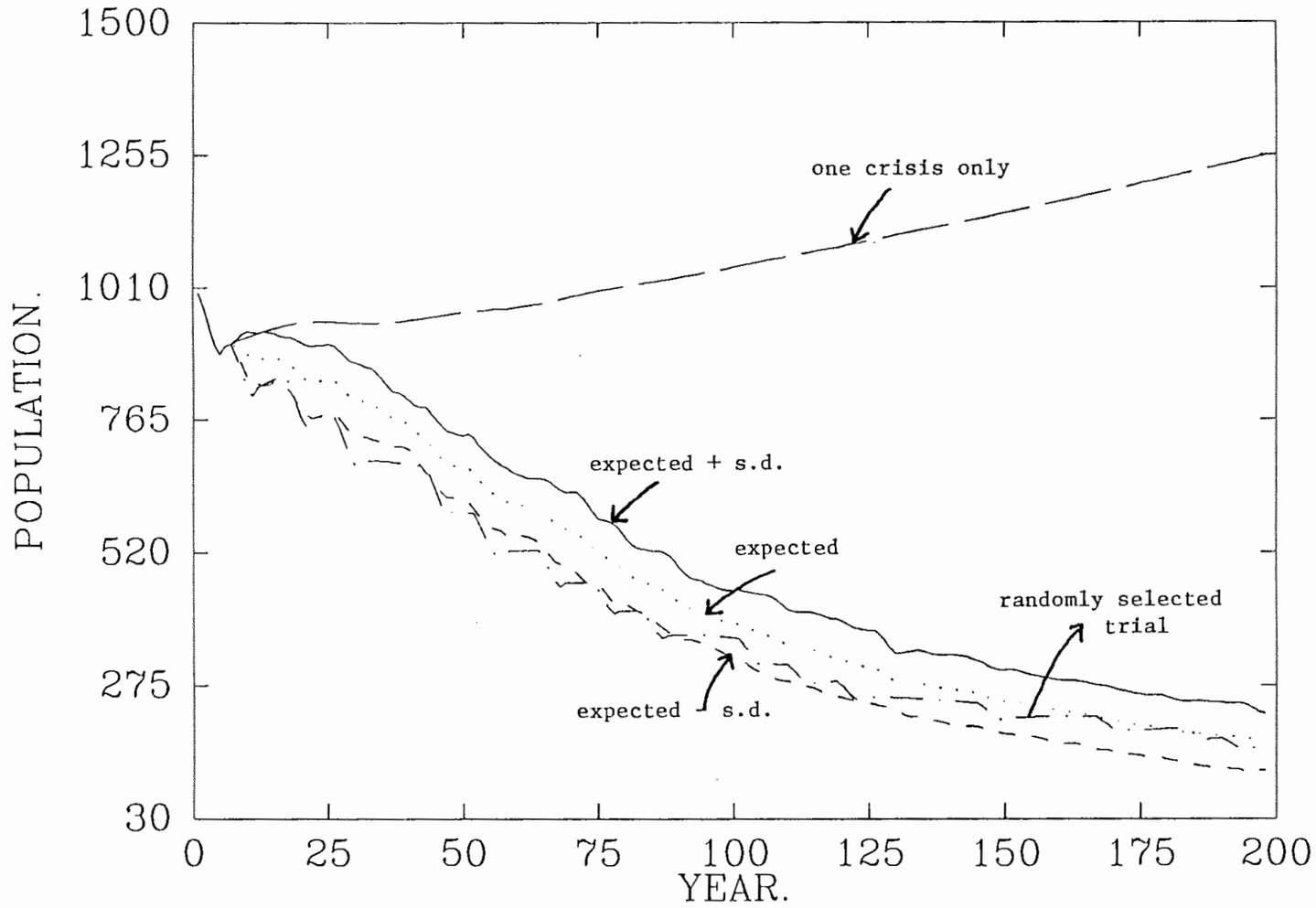
FIGURE 6C. PROJECTED POPULATIONS. CRISIS REGIME=HHS.
YEARS 1-200.



(Initial Population: nearly stationary)

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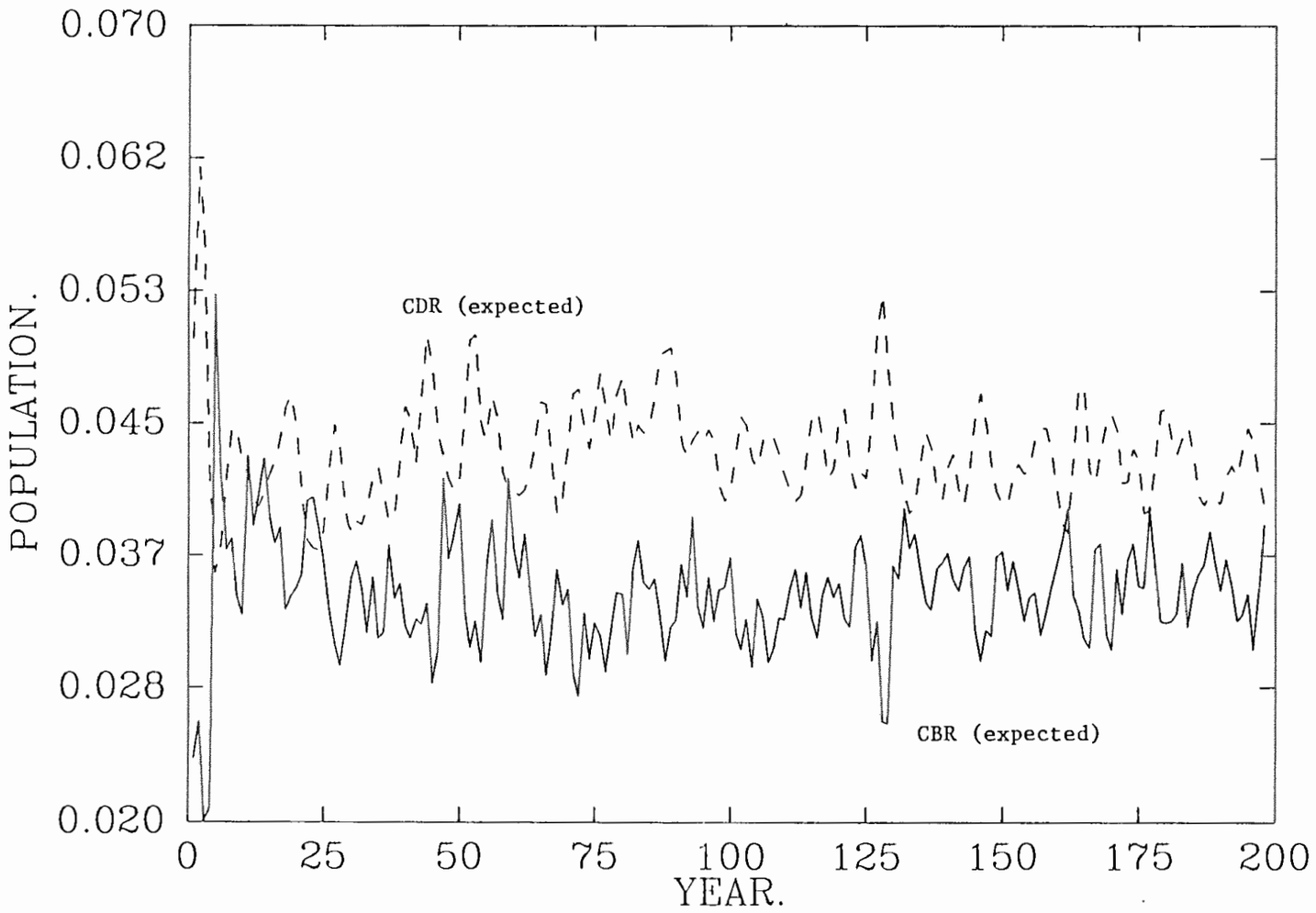
FIGURE 6D. EXPECTED CBR AND CDR. CRISIS REGIME=HHS.
YEARS 1-200.



(Initial Population: nearly stationary)

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