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TIME SERIES CAUSALITY BETWEEN INFANT MORTALITY AND FERTILITY IN LESS DEVELOPED COUNTRIES

City University of New York PH.D. 1982

TIME SERIES CAUSALITY BETWEEN INFANT MORTALITY AND FERTILITY IN LESS DEVELOPED COUNTRIES

BY

RATNA KARMAKAR

A DISSERTATION SUBMITTED TO THE GRADUATE FACULTY OF THE DEPARTMENT OF ECONOMICS OF THE CITY UNIVERSITY OF NEW YORK IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

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Professor Linda N. Edwards

Supervisory Committee

The City University of New York

TO MY PARENTS

AND

MY FAMILY

Abstract

TIME-SERIES CAUSALITY BETWEEN
INFANT MORTALITY AND
FERTILITY IN LESS DEVELOPED
COUNTRIES

by

Ratna Karmakar

Advisor: Professor Michael Grossman

Causality tests between infant mortality and fertility are performed for eight LDCs. The countries are: Egypt, Mauritius, India, Srilanka, Puerto Rico, Jamaica, Venezuela, and Columbia. Aggregate annual time series data are employed to test causation based on Granger-Sims definitions using leads and lags. The Box-Jenkins technique is used to whiten the series to run Sims' test. Both tests use first differenced series.

According to Granger's test, infant mortality affects fertility in six countries. The exceptions are India and Columbia. Fertility affects infant mortality in Egypt, Srilanka, India, Jamaica and Columbia. Sims' test shows causality running from infant mortality to fertility in four countries: Egypt, Mauritius, India and Columbia. Fertility affects infant mortality in only two countries:

Mauritius and Puerto Rico. An additional test was performed on Granger causality using a linear trend term and without first differencing. This test shows only one way causality from fertility to infant mortality in the following five countries: Mauritius, India, Srilanka, Venezuela and Columbia.

FOREWORD

This thesis is predominantly an empirical econometric study of causal relationships between socioeconomic variables. I have tried to present the material as simply as I could.

I owe debts to many persons in preparing this thesis. The firstname that comes in the array is my dissertation supervisor Prof. Michael Grossman. Since my joining the Graduate School, I have been blessed by his kind help and valuable suggestions on many occasions. I was delighted to think that I am one who received special treatment from him. Now that I know him and the department more intimately, I am sure that everyone feels the same way about him. He is such a special person who could most genuinely afford a special treatment to everyone he associated with.

Prof. Harvey Gram, a member of my supervisory committee, is a rare combination of a good man and a good professor. His comments in an initial draft helped gain clarity and cogency. Thanks are also due to Prof. Linda Edwards for her criticisms and suggestions.

Discussions with my friends, Dr. Tadashi Yamada and Mr. Michael Massourakis, helped clarifying many issues.

The Department Secretary, Mrs. Ann Holzman, was extremely nice and helpful. She allowed me to use her own typewriter in expediting the preparation of the first draft. Ms. Vicki L. D'Andrea did the final typing. She did an excellent job.

My husband helped me in so many ways and bore with me all the agonizing moments. Without his help this thesis would be an impossibility. Yet I have chosen to keep all my appreciations for him private. I do remember my parents at this moment with respect and gratitude. They have always inspired me for higher studies and academic attainment and had given me opportunities beyond their financial limitations. They are probably the happiest ones to know about my getting the Ph.D. Degree. Last but not the least, our 4 year old son, Arnab, deserves mention for his cooperation in my academic pursuit. He is very attached to his mother, yet he never stood in her way to study.

All the names could not be mentioned. But there are many others who directly and indirectly helped in completing the thesis. Their contribution is no less important and is gratefully acknowledged. However, all errors and responsibilities are unshared by anyone except myself for most rational reason.

February 1982 Ratna Karmakar

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CHAPTER I

INTRODUCTION

The elucidation of a causal relationship between fertility and infant mortality in the less developed countries is the major goal of this empirical econometric research. Economic theory [Preston, (1978), Ben Porath (1978), Schultz (1969, 1975), O'Hara (1975)] tells us that infant mortality affects fertility. The correlation could be positive or negative.

Parents usually have a family size goal. Higher infant mortality requires more births to attain that goal. Lower infant mortality results in fewer births that are necessary to reach the targeted family size. "Hoarding" and "replacement" strategies help us explain the reactions to infant mortality. A hoarding strategy implies that parents will try to protect themselves against expected infant mortality by having more children than they would otherwise consider optimal. A replacement strategy refers to any reaction to an experienced child death. These are discussed in detail in Chapter II.

Infant mortality, ex post, can also affect fertility through the "interval effect" that operates via lactation. For the mothers who breast feed their children the next

birth is usually speeded up if the nursing baby dies. This is caused by a faster return of ovulation.

However, it is also possible that as infant mortality goes up fertility goes down. This may be because the unpleasant experience of child death may cause parents to refrain from exposure to the same risk again. Infant mortality can also bring about a revision in the preferred number of surviving children. This occurs when the price elasticity of demand for children exceeds one so that an increase in infant mortality rate brings about a reduction in the number of births. The negative correlation is explained in Chapter II.

On the other hand it may also be argued that an increase in fertility raises infant mortality. High fertility is associated with repeated and closely spaced pregnancies which result in low birth weights, damaging child's health and thereby increasing infant mortality rate. Further, in families with many children there is likely to be a higher incidence of malnutrition, poor sanitation and untreated illness. All these result in high infant mortality rate. Thus there exists a positive correlation from fertility to infant mortality.

High correlation between two variables does not in any sense guarantee that the variables are causally related. Very frequently variables may be functionally related but

not correlated, or they may be correlated yet not functionally related. According to David A. Pierce (1977): "The former effect arises because correlation is a measure of linear association only, the latter because of common association of both with a third factor." Even if there exists correlation between two variables the question remains whether the variables are causally related, and if so, what is the direction of causality. In this dissertation an effort is made to answer the following two questions: Does there exist any unidirectional causality from fertility to infant mortality or from infant mortality to fertility? Do we get any feedback relationship between the two variables such that infant mortality affects fertility and is in turn affected by fertility?

To test the relative strengths of these various hypotheses we use lagged independent variables. Instantaneous causality, i.e., current fertility affecting current infant mortality and vice versa, is ruled out as an impossibility.

Of the relevant statistical techniques, the most popular and widely used are those developed by C. W. J. Granger (1969) and Christopher Sims (1972) using time series data. Sims (1972), Mehra (1977), Sargent (1976), Hsiao (1979) and others have used the techniques to detect causality between macroeconomic variables. There is also

one pioneering research effort in the field of Labor Economics [Robert Michael (1977)] to detect causality among fertility, divorce and labor force participation rate of women. This work has inspired the present study which addresses the issue of causality between fertility and infant mortality in eight less developed countries: Egypt and Mauritius - Island of Maurice in Africa, India and Srilanka in Asia, Jamaica and Puerto Rico in North America and Columbia and Venezuela in South America.

Aggregate annual time series data are taken from the Demographic Yearbook published by the United Nations. In addition data from several years for India have been collected from publications of the Vital Statistics Division, Office of the Registrar General and Census Commissioner, India and also from the U. S. Bureau of Census. The beginning year is 1932 for each country, but the final year varies from 1968 to 1977 depending on the availability of data. The data are discussed in detail in Chapter IV. However, the data for most of the countries are unreliable to some degree. These drawbacks seriously preclude any firm conclusion to be drawn on the basis of the empirical findings. Nonetheless, they could serve as an approximation in the absence of any better alternative.

The tests that are used in this thesis can be described briefly. According to Granger's test, in a bivariate analysis, a variable X is said to cause another variable Y if the lagged values of X improve the predictability of Y relative to the prediction based on Y's own past. According to Sims' test, X causes Y if, in a regression of X on current, past and future Y it is found that the future or lead values of Y are significant as a group.

Previous studies based mostly on a single cross section do not give unbiased estimates of causal relations because most economic and demographic aspects of the household are jointly dependent on prior conditions that have influenced fertility and infant mortality. The neglect of intermediary causal relations might give an exaggerated and distorted effect of fertility on infant mortality and vice versa.

Again, reproductive behavior occurs sequentially. A distributed lag model seems to be more appropriate to allow time for reproductive behavior to respond to any change in the independent variable. In view of these considerations, empirical analyses of aggregate and individual time series, to study causal relationship are warranted.

Furthermore, this paper focuses on the causal issue which is a very important aspect of the LDCs, yet which economists have studied the least.

The plan of the work is as follows: Chapter II discusses the theoretical premises; Chapter III develops and explains statistical techniques and methodology; Chapter IV presents the detailed empirical findings and Chapter V summarizes the results.

CHAPTER II

THEORETICAL PREMISES

2.1: Factors Affecting Fertility

Individual tastes, religion, culture, social norms all play major roles in determining fertility. Yet differences in fertility can also be explained by differences in economic and social environment (Schultz, 1974). Parents receive pleasure from their children, but there is a cost involved in deriving pleasure from offspring. Time and money have to be spent in bringing up children. In this sense children are purely time intensive consumption goods. Children can also be considered as an investment in future consumption of goods and services if they work during childhood or support parents in disability or old age. Since children are a source of satisfaction one might expect richer parents to demand more of them. Yet richer parents usually have fewer children. This paradox can be resolved if we distinguish between quantity and quality of children and treat them as two separate variables in a model that determines reproductive behavior of parents. This is the approach of modern theory of fertility developed by Becker (1960), Becker and Lewis (1975) and Willis (1973). The basic tenet is that parents

choose an optimal number of children as well as optimal level of well-being (quality) of children. Quality is assumed to be a substitute for quantity, so that families would reduce the quantity of children if they raise the quality of children, ceteris paribus. The utility function of parents appears as

$$U = U(n,q,c) \quad \ldots \qquad (2.1.1)$$

where, n = number of surviving children, q = quality per child and c = parental consumption or an index of parents' standard of living. Both quantity and quality of children are objects of choice and they compete with each other as well as with parents' own consumption in the sense that parents have to allocate scarce resources among three "goods": n, q and c. With given income and time, q can be raised only by lowering c and/or n and vice versa.

In LDCs poverty and high fertility go hand in hand [Burdick (1959), Chandrashekhar (1946), Coale (1958)]. Children of poor parents work at home and outside home at an early age. For poor parents child labor is vital for family welfare and the parents usually prefer more children (number) to less. Moreover, if children help to support their parents in old age, the current cost of raising a child is like an insurance premium, they are willing to

pay. Also in these countries since mothers command only low wages, the difference between childrens' and mothers' earnings may be small. Workdays lost by mother during a child's infancy may easily be recouped by the child later on. Sometimes older children can look after the younger ones and save mothers' time to work at home and outside the home. Finally, much of women's traditional work in these countries (such as household labor, cutting fodder, feeding cattles, etc.) can be combined with looking after children.

The link between poverty and high fertility is further reinforced by high rates of infant and child mortality in these countries. The effect of infant mortality on fertility has been extensively discussed in the writings of Ben Porath (1978), Schultz (1969, 1974), Donald O'Hara (1975), Freedman (1979), Heer (1966), Heer and Smith (1968), Preston (1978), Potter (1965), Easterlin, Pollak and Wachter (1980. The economic arguments for this relationship are reviewed in the following section.

2.2: Infant Mortality Affecting Fertility

Let us maximize the above utility function (2.1.1) subject to the income constraint:

$$I = P_c\, c + n\, q\, \pi \qquad (2.2.1)$$

where,

I = full income (including foregone earnings)

$n\, q\, \pi$ = total outlays on children

P_c = price per unit of c (=\$1)

For simplicity let us assume that index of q is one, i.e., there is no variation in q. Then the relevant Lagrangian function would be

$$L = u(n,c) + \lambda\, (I - P_c c - \pi\, b) \qquad (2.2.2)$$

π is the cost of a birth whether the child survives or not. So πb (and not πn) measures total outlays on children. Let, $b = \frac{n}{p}$ i.e., birth rate is a ratio of number of surviving children (n) and the probability of survival (p). Let us suppose (1-p) infant mortality rate is 3/8. Then (P) the probability of survival is 5/8. In which case, it is clear that a family that wants 5 surviving kids, should have 8 births (because n=5, $p=\frac{5}{8}$, so $b = 5\ (\frac{8}{5})$ or 8). Let us now substitute (n/p) for b in (2.2.2) and get

$$L = u(n,c) + \lambda\, (I - P_c c - \frac{\pi}{p}\, n) \ \ldots \qquad (2.2.3)$$

The solution to this function yields

$$\frac{u_n}{u_c} = \frac{\pi}{p} \quad \cdots \qquad (2.2.4)$$

which is the first order condition for maximization. $\frac{\pi}{p}$ is the relative price of n. Let us denote $\frac{\pi}{p}$ by π^*. If p goes up, relative price of children goes down and the quantity of surviving children demanded would go up too. On the other hand if P goes down (which is the same thing as (1-p) infant mortality going up) relative price of children goes up and the quantity of surviving children demanded decreases.

We shall now consider the effect of infant mortality on birth rate (b). First we consider the target model where families desire a fixed number of surviving children [n]. That number is chosen once and is not altered by actual outcome of births. Hence, infant deaths are fully offset by additional births. Let us start from the definition of birth rate, b. Since $b = \frac{n}{p}$,

$$\partial \ln b = \partial \ln n - \partial \ln P \quad \cdots \qquad (2.2.5)$$

and $$\frac{\partial \ln b}{\partial \ln P} = -1 \quad \cdots \quad (\because n \text{ is fixed } \frac{\partial \ln n}{\partial \ln p} = 0) \qquad (2.2.6)$$

Also, $$\frac{\partial \ln b}{\partial \ln (1-p)} = \frac{\partial \ln b}{\partial \ln p} \frac{\partial \ln P}{\partial \ln (1-p)} \quad \ldots \qquad (2.2.7)$$

$$= - \frac{\partial \ln P}{\partial \ln (1-P)} = - \frac{(\frac{1}{P})}{-(\frac{1}{1-P})} \qquad (2.2.8)$$

$$= \frac{1-P}{P} \quad \ldots \qquad (2.2.9)$$

Equation (2.2.6) shows that if the rate of probability of survival goes up, birth rate goes down at the same rate in a target model. Equation (2.2.9) shows that infant mortality rate and birth rate move in the same direction always in a target model. Thus

$$\frac{\partial F_{t+1}}{\partial M_t} > 0 \quad \ldots \qquad (2.2.10)$$

Where M_t is infant mortality rate at the current period and F_{t+1} is the fertility rate one period ahead. Infant mortality raises the number of births necessary to achieve the desired family size goal. If infant mortality rate goes down, the family size goal is attained with fewer births. Hence fertility rate goes down.

In this theory the predicted positive relationship is based on the assumption, that all other things remaining the same, all parents desire the same number of surviving children, regardless of the infant mortality

levels they expect. This assumption holds good if the demand for children is perfectly inelastic or if the cost of an infant death is zero. In reality neither of these conditions holds. More realistically, whether an increase in infant mortality lowers or raises desired number of children depends partly on the cost associated with infant deaths and partly on the elasticity of demand for surviving children.

Let us now consider the static neo-classical model where families choose an optimal number of surviving children. Families do not revise their demand for optimal number of children based on actual experience, but they take account of expected survival probability in picking the optimal number of survivors. In such a model,

$$\ln b = \ln n - \ln P$$

$$\frac{\partial \ln b}{\partial \ln p} = \frac{\partial \ln n}{\partial \ln p} - 1 \quad \ldots \qquad (2.2.11)$$

$$= \frac{\partial \ln n}{\partial \ln \pi^*} \frac{\partial \ln \pi^*}{\partial \ln p} - 1 \quad \ldots \qquad (2.2.12)$$

$$= -\epsilon \frac{\partial \ln \pi^*}{\partial \ln p} - 1 \quad \ldots \qquad (2.2.13)$$

Since $\pi^* = \frac{\pi}{p}$, $\ln \pi^* = \ln \pi - \ln p$ and $\frac{\partial \ln \pi^*}{\partial \ln p} = -1$ substituting this value in (2.2.13).

$$\frac{\partial \ln b}{\partial \ln p} = \varepsilon - 1 \lesseqgtr 0, \text{ as } \varepsilon \lesseqgtr 1 \quad \ldots \qquad (2.2.14)$$

$$\text{and } \frac{\partial \ln b}{\partial \ln (1-p)} = \frac{1-p}{p}(1-\varepsilon) \quad \ldots \qquad (2.2.15)$$

where ε is gross own price elasticity. If $\varepsilon<1$, it implies that there should exist in equilibrium a positive association between birth rate and anticipated or experienced infant death rate.

Finally, let us consider the sequential (dynamic) model. Infant mortality could as well bring about a revision in the preferred number of surviving children. A decline in infant mortality will increase the desired family size goal if the elasticity of demand for children exceeds one. Different economists have developed different models about the effect of infant mortality on fertility. A synopsis of these models follows next.

Ben Porath (1978) mentions about two types of reactions to infant mortality: "hoarding" and "replacement." Hoarding refers to response of fertility to expected infant mortality whereas replacement refers to such response due to an experienced child death. Hoarding involves

deviations from optimum life cycle if expected infant mortality deviates from actual infant mortality significantly. It also necessitates a large number of births and requires supporting them to adulthood. Hence, hoarding is a comparatively costly strategy if infant mortality goes down unexpectedly.

If sequential replacement is pursued, parents will give birth to an additional child if they lose one. Thus fewer births would be necessary to achieve the family size goal and hence it is less costly. In this sense replacement is a superior strategy to hoarding. Generally parents have time and ability to replace an infant's death. However, replacement strategy is more risky than hoarding strategy because sometimes replacement might not be feasible or desirable because of the loss of fecundity, health hazards or for psychological reasons.

According to Schultz (1969) parents try to compensate for the average incidence of infant death by seeking the number of births that will give them the desired number of surviving children. This can be done in two ways. First, parents incorporate expected incidence of infant death into their lifetime reproductive behavior. Second, parents may make an added effort to have an additional child only if they lose one of their children. Ben Porath's distinction above, is very similar to Schultz's distinction.

In addition, Schultz emphasizes that birth rates may also be affected by the uncertainty in the family formation process where births and deaths cannot be predicted with any degree of accuracy. If parents were to value highly having no less than the desired number of children, they would try to hedge against any uncertainty arising out of infant deaths by increasing the number of live births. Hence, effective birth rates would exceed the desired birth rate.

On the other hand, if parents regard additional offspring as a burden to their time and resource constraints, they would rely on the replacement strategy to fulfill the desired family size goal.

Knodel's insurance effect is also very similar to hoarding strategy. Knodel (1978) speaks about the "insurance" effect whereby couples adjust fertility to anticipate possible future child loss, thus insuring the survival of at least the minimum number of offspring they desire. The insurance effect is based on the couple's awareness of the community level of child mortality independent of their own experience. If infant mortality rate in the society goes down it pulls the birth rate in the same direction.

According to Mattheissen and McCann[1] lower infant mortality results in larger cohorts surviving to adulthood. This in turn implies even scarcer resources and means of support. The response to this pressure manifests itself in late marriages and fewer number of children. This idea is related to Malthusian notion of "positive check."

Donald J. O'Hara (1975) analyzes the effect of a mortality decline on parental decision concerning both quality and number of children. He assumes that a mortality decline affects both costs and rewards associated with a given fertility behavior. A mortality decline generates a systematic shift in relative costs, reducing both expected cost of quantity and quality of children relative to other goods and also the expected cost of quality of children relative to quantity. Using Von-Neumann Morgenstern utility theory he shows that parents woulc choose a smaller quantity of children following a decline in infant mortality if they substitute quality for quantity and if this substitution effect is strong enough to outweigh the tendency to substitute quantity for other goods.

Preston (1978) speaks about the interval effect operating through lactation and affecting fertility. Since breast feeding stops if a nursing child dies, the death

may speed up the return of ovulation and make the arrival of next birth closer. Hence infant mortality could be expected to result in more births over the course of a woman's lifetime. Since breast feeding is a common practice in the LDCs, this is a very influential relationship in these countries and operate without deliberate choice being made by the parents to have additional children. However, such effect cannot result in complete replacement. Preston says that 50 per cent is the maximum replacement of fertility to changes in infant mortality resulting from interval effect. If infant mortality goes down the interval between any two births would be widened, thereby lowering fertility.

Ronald Freedman (1979) argued that "known low infant mortality is one of the necessary conditions for an effective social policy for reducing fertility." In a cross-country regression analysis, Heer (1966) showed that infant mortality is one of the strongest and most consistent predictors of fertility levels. Heer and Smith (1968) showed that a decline in infant mortality could produce a more than compensating fertility decline if we consider the long run effects.

It is also possible to find negative correlation between infant mortality and fertility. The unpleasant experience of infant mortality makes couples flinch from

the same risk and painful experience in future and they might not want to give birth to an additional child. From strictly economic point of view, a child death may increase the imputed cost of raising a surviving child and thus produce a substitution effect away from expenditure on children. Thus, if the price elasticity of demand for children exceeds one, infant mortality would induce fertility in opposite direction.

All the above models emphasize either replacement or hoarding strategy. The models based on replacement strategy do not consider the fact that replacement of a child will be much more risky and also more expensive if replacement leads to high order birth. If replacement is associated with old age of the mother (say about 30) the probability of survival of the new born would be much less. This would increase both pecuninary and psychic cost of an additional child to parents. The same reasoning will render hoarding as a risky strategy too. Hence, before predicting that replacement or hoarding would lead to additional birth to compensate for an infant's death, actual or anticipated, economists should really make an explicit cost-benefit analysis. Parents would be rational to replace a lost child or hoard children in anticipation of future losses, only if the present value of benefit exceeds or at least equals the present value of cost.

Second, neither of these two strategies does recognize differences in sex. In a society where there is a strong preference for a male child, the death of a female child would have no impact on the reproductive behavior of parents. Only the death of a male child will be replaced. But again what is the guarantee that the next birth will be of the same sex that parents want to replace? In the absence of any such guarantee rational behavior would lend little support to replacement strategy. However, if parents are indifferent to a boy or a girl, these strategies are justified.

Third, the hoarding strategy implies that parents want to insure the survival of at least a certain number of their children. This is important if children are considered as old age security. Since old age security or benefit is becoming an integral part of public policy in industrialized societies, the investment criterion is starting to lose its significance. As a result hoarding strategy is becoming weaker and weaker as an explanation of having children. Even in agrarian societies many children are no more considered as boon because of mechanized argiculture which has rendered many hands absolutely unnecessary in farmland. Also, because of revised land legislation in many of these countries, allotment of land to an individual follows a quota system.

This pattern of allocation of land to a farmer has curtailed the demand for children as a source of support in agricultural work.

Finally, if we think of children as consumption goods, then replacement strategy seems to be superior to hoarding strategy. This is because hoarding has the risk of exceeding the optimal number of children if actual infant mortality falls short of expected infant mortality. This will lead to "crowding out" effect. Too many children will interfere with parents' utility function and cause disutility.

2.3: Fertility Affecting Infant Mortality

In 1798, Thomas Robert Malthus first postulated the positive correlation between fertility and infant mortality. But in his model increase in infant mortality rate lags behind increase in fertility -

$$\frac{\partial\ IM_{t+1}}{\partial\ F_t} > 0. \qquad (2.3.1)$$

According to Malthus plagues and sickly seasons occur after a period of rapid increase in the population. The number of inhabitants exceed the food supply and the accommodations necessary to keep them healthy. A greater number of masses would be crowded together in one house. These natural

causes would produce a sickness even though the country might not be crowded with populous in any absolute sense. The standard of living and food might have unfavorable effect on the health of the common people. The redundant population is eventually carried off by nature's disorders which become almost evanescent. As fertility goes down infant mortality goes down too basically due to improvements in the level of living, particularly better sanitation and personel hygiene.

According to modern theory of population, medical and public health facilities could be extended to more families as fertility rate declines thus lowering infant mortality rate. Infant mortality caused by congenital malformations, birth injuries, postnatal asyphexia, atelectasis account for about 50 per cent of infant death in the developed countries. In the LDCs diseases peculiar to early infancy include preventable death due to accidents at birth, infections of the new born, malnutrition, epidemics like malaria, diarrhea, gastrointestinal illness, etc.. The infant mortality rate responds whenever a community has sufficient resources to ensure hospital delivery, improved sanitation and public hygiene and impart a modicum of health education to the public, especially the pregnant mothers. The lower the fertility rate the more will these facilities be extended to its members.

Consequently, infant mortality rate will slow down. To borrow Chandrashekhar's terminology[2], ..."a low infant mortality rate is a purchasable commodity ... in an underdeveloped country." In families with many children there are more problems with malnutrition and illness of children than in small families. These result in higher mortality rates among younger children; slower physical growth and less intellectual development. Excessive "crowding" of children is equally harmful as excessive number of children. In LDCs infant and child mortality are much higher in larger families compared to small families. According to a report of the National Academy of Sciences[3], during 1955-58, 206 out of 1000 infants died in families in which mother had given birth to seven or more children. The infant mortality was considerably lower in families where mothers had given birth to only two children, at 116 out of 1000. The same proportionate differences in mortality rates were evident in New York City, although the levels of mortality are significantly lower here, (National Academy of Sciences, 1971). As fertility goes down parents can give greater amount of care to protect children from the common causes of death, diarrhea, gastrointestinal illness etc.. This is endogenous element of decline in child mortality.

The PAHO[4] study of child mortality in thirteen Latin American projects had disclosed that malnutrition was an associated or underlying cause of 57 per cent of the deaths before age five. Per capita food production in the developing regions, taken as a whole, failed to stay abreast of the growth of population during 1959-61 and 1965-67. However, in several countries with large populations this trend continued for most of the sixties. India was one of the countries. This caused actual famines or fears of famines increasing the mortality rate in general and infant mortality rate in particular. FAO projected an average 3.9 percent per year increase in demand for food in the low income countries up to 1985. The "population effect" accounts for 71 per cent of this demand, while "income effect" accounts for only 29 per cent. Thus fertility is expected to affect infant mortality in the same direction.

Moreover, when high fertility is associated with repeated and closely spaced pregnancies, mothers' health can suffer: the results - low birth weight and early weaning - thus damaging child's health and increasing the probability of infant death.

Thus, when we consider the effect of fertility on infant mortality, both Malthusian theory and modern theory of population envisage a positive correlation between fertility and infant mortality.

FOOTNOTES TO CHAPTER II

1. Mattheissen, Paul C. and McCann, James C., "The Role of Mortality in the European Fertility Transition: Aggregate Level Relations." In Preston, 1978.

2. Chandrashekhar, Sripaty, Population Conference, 1965, Vol. II, In Fertility, Family Planning, Mortality. The United Nations, New York, 1967.

3. Rapid Population Growth - Consequences and Policy Implications. Office of the Foreign Secretary. National Academy of Sciences. The John-Hopkins Press, 1971.

4. Preston, Samuel H., 1980, P. 301.

CHAPTER III

DEFINITIONS OF CAUSALITY AND METHODOLOGY

3.1: Granger's Test of Causality

This definition of causality is in fact based on 'Weiner-Granger Causality' which states: a variable X causes another variable Y wish respect to a given universe or information set that includes X and Y, if present Y can be better predicted by using past values of X than by using the past values of Y only. To start with let us fit a bivariate model to each series:

$$F_t = \alpha_o + \sum_j a_j F_{t-j} + \sum_j b_j IM_{t-j} + \varepsilon_t \quad \ldots \qquad (3.1.1)$$

$$IM_t = \beta_o + \sum_j c_j IM_{t-j} + \sum_j d_j F_{t-j} + n_t \quad \ldots \qquad (3.1.2)$$

where, F_t is fertility rate and IM_t is infant mortality rate. α_o and β_o are the intercept terms. F_t and IM_t are two stationary stochastic time series with constant mean and variances. ε_t and n_t are two uncorrelated white noise series, i.e., $E(\varepsilon_t \varepsilon_s) = E(n_t n_s) = 0$, $s \neq t$ and $E(\varepsilon_t \varepsilon_s) = E(n_t n_s) \neq 0$ for all $t=s$. Theoretically j can take any value up to infinity. But for all practical purposes j must be finite. This is a necessary condition for the

finite length of the data. In order to be meaningful j is in fact shorter than the given time series.

Let the bar over each variable represent past series of the variable and let the vertical line | mean given.

Let $\bar{U}_t$ be all the information in the universe accumulated since time t-1. Let $\overline{U_t - IM_t}$ denote all this information apart from the specifies series $\overline{IM_t}$. Also let $\sigma^2 (F_t|\bar{U}_t)$ be the minimum predictive error variance given information set $\bar{U}_t$ and $\sigma^2 (F_t|\bar{U}_t - \overline{IM_t})$ be the minimum predictive error variance using information set $\bar{U}_t$ less $\overline{IM_t}$. If $\sigma^2 (F_t|\bar{U}_t) < \sigma^2 (F_t|\bar{U}_t - \overline{IM_t})$, we say that infant mortality is causing fertility. In order words, if we are better able to predict fertility using information on past infant mortality than not using it, we conclude that infant mortality is causing fertility.

However, the most unrealistic part of the above definition is the use of the series U_t, a large majority of which would be quite irrelevant in analyzing causal relation between fertility and infant mortality. A straight forward and logical way is to use the vector set of the time series which are directly relevant for our model. Hence only two vector sets F_t and IM_t are included in the model. All other relevant variables that affect fertility or infant mortality, are already incorporated in past F_t and IM_t.

Let $\sigma^2 (F|\bar{F})$ be the minimum predictive error variance of F_t using only past F_t. Let $\sigma^2 (F|\bar{F},\overline{IM})$ be the minimum predictive error variance if both past F_t and past IM_t are used to predict F_t. Then IM_t is said to cause F_t ($IM_t \rightarrow F_t$) if $\sigma^2 (F|\bar{F},\overline{IM}) < \sigma^2 (F|\bar{F})$. By summetry, F_t is said to cause IM_t if $\sigma^2 (IM|\overline{IM},\bar{F}) < \sigma^2 (IM|\overline{IM})$. If both these are true there is said to be feedback relationship between fertility and infant mortality. The best linear predictor of F_t (IM_t) using past F_t (IM_t) and past Imt (F_t) will take the following form:

$$P_t (F|(\bar{F},\overline{IM}) = \alpha_o + \sum_j a_j F_{t-j} + \sum_j b_j IM_{t-j} + \epsilon_t \quad (3.1.3)$$

$$P_t (IM|\overline{IM},\bar{F}) = \beta_o + \sum_j c_j IM_{t-j} + \sum d_j F_{t-j} + n_t \quad (3.1.4)$$

Thus the definition of causality is based entirely on the predictability of any series F_t (IM_t). If some other series $IM_t(F_t)$ contains information in past terms that improve the prediction of the first series, that is not contained in past terms of any other series used in the predictor, then we could say that $F_t(IM_t)$ is causing $IM_t(F_t)$. Thus the flow of time is crucial in Granger causality.

In equation (3.1.3) IM_t will cause F_t if some $b_j \neq 0$. Similarly, in equation (3.1.4) if some $d_j \neq 0$, F_t is said to cause IM_t. If both of these events occur there is said to be a feedback relationship between IM_t and F_t. However, to test the hypotheses $\sum_j b_j \neq 0$ and $\sum_j d_j \neq 0$, we have to have two more equations:

$$Pt(F|\bar{F}) = \alpha_o + \sum_j a_j F_{t-j} + \epsilon_t' \ldots \quad (3.1.5)$$

$$Pt(IM|\overline{IM}) = \beta_o + \sum_j c_j IM_{t-j} + n_t' \ldots \quad (3.1.6)$$

Let us label equations (3.1.3) and (3.1.4) as unconstrained equations and equations (3.1.5) and (3.1.6) as constrained equations. When we run both the unconstrained and the constrained equations we have information in past terms of the other series (crossed lags). We can now use that information to run the F-tests to find out the significance of the crossed lags. F_A is a test of the null hypothesis that the lagged values of infant mortality do not improve the forecast of fertility than can be made on the basis of the own lagged values of fertility. If F_A is significant at 10 per cent we reject the null hypothesis and conclude that infant mortality is causing fertility (IM → F). F_B is a test of the null hypothesis that the lagged values of fertility do not

improve the forecast of infant mortality than can be made on the basis of its own past. If we fail to reject the null hypothesis we conclude that there is no causal link from fertility to infant mortality. ($F_t \not\rightarrow IM_t$). If F_B is significant we conclude that fertility affects infant mortality ($F_t \rightarrow IM_t$). The partial F-tests are calculated as follows:

$$F_{(k-1,\ n-k)} = \frac{\dfrac{(ESS_u - ESS_c)}{(k-1)}}{\dfrac{(USS_u)}{n-k}} \quad \ldots (3.1.7)$$

where,

ESS_u = Explained sum of squares of the unconstrained equation.

ESS_c = Explained sum of squares of the constrained equation.

USS_u = Unexplained sum of squares of the unconstrained equation.

k-1 = Degrees of freedom in the constrained equation less one.

n-k = Total number of observations less the degrees of freedom in the unconstrained equation.

The above formula ultimately reduces to:

$$= \frac{(R_u^2 - R_c^2) \ / \ (k-1)}{(1-R_u^2) \ / \ (n-k)} \quad \ldots \qquad (3.1.8)$$

The subscripts u and c denote unconstrained and constrained equations respectively.[1]

The main caution with this approach is that there be no serial correlation in the residuals. To get rid of this problem one should enter a sufficient number of lagged values. Omitting relevant lagged values could produce "spurious regression phenomenon." A reasonable specification is to enter all lagged coefficients which are significantly different from zero. However, caution should also be practiced as one introduces own lagged terms, because mutlicollinearity might be a problem and it would not be possible to get any estimate. To get rid of this problem one might introduce few own lagged terms and more cross lagged terms.

The original time series have been transformed to stationary series by taking the first differences of the logs. The logarithmic transformations are causality preserving in the same way as are the original data.

There is also an advantage of working with logs. When the mean is increasing over time, the magnitude of first differencing also increases over time indicating lack of homogeneity in variance. In such a situation homogeneity would be induced by taking natural logarithms of the raw data and taking differences of the logs.

There are two problems associated with Granger's Test:

1. If relevant data had not been included in this set then spurious causality could arise. For example, if the series D consists only of two series, but in fact there was a third series say, per capita income (I_t) which was causing both F_t and IM_t within the enlarged set $D' = (F_t, IM_t, I_t)$, then for the original set D, spurious causality between fertility and infant mortality could be found.
2. If F_t is causing IM_t with lag one unit but the series is sampled every two time units, then this would also suggest causality although none really exists. This is because some relevant informations have not been taken into account. This contradiction could be eliminated if the economic variables are recorded at more frequent time intervals.

3.2: Sims' Test of Causality

Sims (1972) makes use of a direct test of unidirectional causality. This test requires estimating two sided lag distributions between the variables. Causation can be inferred by regressing a suitably transformed stationary series of fertility (infant mortality) on past, current and future values of a stationary transformed series of infant mortality (fertility). Let us consider the following two equations:

$$F(t) = a_o + \sum_{s=-e_1}^{e_2} a(s)\ IM(t-s) + e(t) \quad \ldots \qquad (3.2.1)$$

$$IM(t) = b_o + \sum_{s=-e_1}^{e_2} b(s)\ F(t-s) + n(t) \quad \ldots \qquad (3.2.2)$$

$t = 1, \ldots, T.$

where, F(t) and IM(t) are the filtered series of fertility and infant mortality respectively. e_1 and e_2 are the lengths of the lag distribution on leading and lagging values of the independent variables. $\varepsilon(t)$ and n(t) are white noise processes.

According to Sims' test, fertility causes infant mortality (F → IM) if the leading values of IM, as a group, have regression coefficients significantly different from zero. Similarly, infant mortality is said to cause fertility (IM → F) if leading values of F have regression

coefficients which are significantly different from zero. In other words,

$F \rightarrow IM$, if $\Sigma a(s) \neq 0$, for $s<0$, and

$IM \rightarrow F$, if $\Sigma b(s) \neq 0$, for $s<0$.

To find out the significance of the future values of the coefficients we need two additional sets of equations:

$$F(t) = a_o + \sum_{s=0}^{e_1} a(s)\ IM\ (t-s) + e'(t) \quad \ldots \qquad (3.2.3)$$

$$IM(t) = b_o + \sum_{s=0}^{e_2} b(s)\ F\ (t-s) + n'(t) \quad \ldots \qquad (3.2.4)$$

The partial F-tests are run following the procedure described in Section (3.1). The constrained equations (3.2.3) and (3.2.4) include only past and current lags. The unconstrained equations include past, current and future lags. The F-values are calculated to determine the significance of future values of F and IM. If future values of F(IM) are significantly different from zero, we conclude that infant mortality (fertility) affects fertility (infant mortality). This feedback causation is based on the logic that future values of a variable cannot cause a change in the current value of another variable.

Sims mentions that, in order for the causality test to be meaningful, the time series should be detrended, jointly covariance stationary with no serial correlation in the regressions' residuals. Therefore, it is imperative that the assumption of serially uncorrelated residuals be "approximately accurate" in order for the F-test to be precise. This is crucial because the F-tests are highly sensitive to the presence of serial correlation in the residuals.

Sims has taken the natural logs of the variables and prefiltered them using a quasi-second differencing method to correct for the serial correlations. He used the filter $\ln X(t) - 2K \ln X(t-1) + k^2 \ln X(t-2)$; with the ad hoc value of $k = .75$. Sims conjectures that most economic time series will be uncorrelated with this prefiltering and the regression residuals will be very nearly white noise.

If, however, serial correlation remains in the residuals after filtering them, the least squares estimates will be consistent, but the estimates of the variances will be biased. Very often the bias is downward producing inflated F-statistic and R^2 and values.[2] Mehra (1977) suggested that the naive application of Sims' filter to correct for serial correlation might not always work. He started with some ad hoc filter to prefilter the variables and applied

OLS to the filtered variables to estimate the lag profile; then ran autoregressions of these residuals and evaluated their autoregressive properties. If the autoregression showed purely uncorrelated error structure, he retained them. Otherwise, the whole process was repeated with a new value of k. He thought that this process should be continued until an uncorrelated autoregressive error structure could be found.

Pierce and Haugh (1977) remarked that it is possible to find causality in the application of Sims' test although none in fact exists if there are substantial autocorrelations in the ad hoc filtered series. They have suggested to relate the prefiltering to the characteristic of the particular series being studied. This method is fillowed in this analysis. Each series is prefiltered by empirically determined filters to generate white noise residuals and then OLS regressions are run on the filtered series. This empirically filtered series is supposed to remove serial correlation adequately and to give "approximately accurate" F-values because the estimates of the variances will be unbiased. The Box-Jenkins Forecasting technique is used to model the time-series and to whiten it. There are two advantages of this technique over the traditional methods like three parameters

Exponential Smoothing, Brown's Discrete Least Squares, Regressions etc..

1. Using Box-Jenkins the analyst eliminates inappropriate models until the most suitable one is traced out.
2. Box-Jenkins presents a rational structure approach to the determination of a model. However, experiment and judgment still play an important role in identifying the model.

The Box-Jenkins Forecasting methodology is used here to model a time series with ARIMA (Autoregressive Integrated Moving Average) structure that reduces observed data to random noise.

3.3: Autoregressive Integrated Moving Average (ARIMA) Model

An approach to the modeling of ARIMA structure involves iterative use of the three stage processes of identification, estimation and diagnostic checking. Estimation of the autocorrelation and the partial auto-correlation function will be the primary aid in model identification. The computer program PDQ computes sample autocorrelations. Here P refers to AR parameters, D denotes differencing and q denotes MA parameters. For example, if P=1, D=1 and Q=1, the model (1,1,1) is referred to as a model with first AR parameter, first differenced series, and first MA parameter. One needs only

identify the lowest level of differencing for which a stationary model is apparent. Nothing is gained by further differencing. Generally, most economic time series are rendered stationary with first differencing.

The sample autocorrelations show the relationship between a current observation and previous observations. The sample autocorrelation function is used to determine which model is appropriate for a particular time series. The identification of a moving average model can be obtained from the sample autocorrelation. Partial autocorrelation model is useful in identifying the AR process. When a mixed (ARIMA) model is present both the sample autocorrelation and partial autocorrelation have a decaying pattern. Pure AR models tend to have exponential decaying patterns. But these are not absolute guidelines. The analyst must use considerable judgment. The general rule to identify a model is described below:

> We look for a test of the statistical significance of the sample autocorrelations. Bartlett's approximation[1] suggests such a test. Since the distribution of the autocorrelation coefficient r_j for $j>q$ is approximately normal, we may regard a sample autocorrelation larger in absolute value than 1.95 standard deviations as being significantly different from zero at the .05 level. In practice,

we use sample estimates of r_j. As a rule of thumb, any autocorrelation coefficient greater than two times its standard error is considered significant. Thus a large value of partial correlation coefficient, ϕ_1 (greater than two times its standard error) might indicate that a first order autoregression ought to be added in the model, while a large value of sample autocorrelation θ_1 (greater than two times its standard error) indicates a first order moving average ought to be added in the model. If, however, ϕ_1 and ϕ_4 are significant but ϕ_2 and ϕ_3 are insignificant, we should suppress (ϕ_2) AR_2 and (ϕ_3) AR_3 and the model might have a second order autoregression. This rule applies equally to moving average parameters.

The next step is estimation. In the process of estimation initial values of AR parameters (Ø) and MA parameters (θ) are provided. The sample autocorrelations are used to obtain preliminary estimates of parameters. These estimates provide starting values for the iterative procedure used in computing the maximum likelihood estimates of parameters.

The computer then calculates the appropriate model coefficient values which minimize the sum of squared residuals. The general expression is to minimize

$$S(\hat{\phi}, \hat{\theta}) = \sum_{t=1}^{T} (Z_t - \hat{Z}_t)^2 \quad \ldots \qquad (3.3.1)$$

where $S(\hat{\phi}, \hat{\theta})$ is the sum of square function. Z_t is the true parameter value of a set of observations generated by the identified model and $\hat{Z}_t$ is the estimate. $\hat{\phi}$, $\hat{\theta}$ are estimated AR and MA coefficients respectively. Alternatively (3.2.5) can be represented as:

$$S(\hat{\phi}, \hat{\theta}) = \sum_{t=1}^{T} \hat{u}(\hat{\phi}, \hat{\theta})\ t^2 \qquad (3.3.2)$$

where, $\hat{u}$ represents residuals.

The next and final step is diagnostic checking to see that the model adequately represents the observed time series. Diagnostic checking of the model involves examining the sample autocorrelations of the residual series, $\hat{u}_t = (Z_t - \hat{Z}_t)$. If it is independently and randomly distributed around zero, the model is adequate. Nelson (1973), P114) suggested the obvious way to check is overfitting and testing the hypotheses that the added parameter is equal to zero. But he also warned that one must be on the lookout for the simplest adequate model and try to avoid redundancies resulting from overfitting. Box and Pierce have suggested a Chi-square test to evaluate whether the residual sample autocorrelations Σr_j^2 exhibit any systematic error. This is the Q-statistic given by

$$Q = n \sum_{j=1}^{K} r_j^2$$

with (k-p-q) degrees of freedom.

where,

Q is computed Chi-square statistic

n = total number of observations minus the maximum back order

r = residual sample autocorrelation values that have been calculated

and p and q represent the number of estimated AR and MA coefficients in the model under consideration. The test statistic (Q) is evaluated against a χ^2 distribution with (k-p-q) degrees of freedom, whereby K = number of lags, P = AR parameters, Q = MA parameters (estimated).

The purpose of the Q-statistic is to examine the autocorrelations of the residuals from the fitted model. On the hypothesis that r_j is white noise, these quantities should be approximately normally distributed with zero means and standard deviations $n^{-1/2}$, where n is the number of residuals. An overall test on the size of these quantities is to compare the Q-statistic with tabulated values of χ^2.

If the Q-statistic is not greater than χ^2 distribution then the evidence does not contradict the hypothesis of white noise behavior in the residuals and the model is adequate. If, however, Q is greater than χ^2, this indicates that the model is inadequate.

3.4: A Comparative Evaluation of Granger Causality and Sims' Causality

Econometricians generally agree about an appropriate definition of causality, but until present time there has been no consensus about the operational procedure to detect causal link. According to Sims (1972), Granger and Sims Causality are equivalent. Therefore, the present study applied both techniques and compared the results.

Recent studies (Zellner (1978), Hsiao (1979), Jacobs (1979)) have criticized both Granger and Sims' tests as inadequate measure for detecting causal links.

Sargent (1977) has criticized Granger's definition on the ground that it is not invariant with respect to interventions in the form of imposed changes in the process governing the causing variables.

According to Zellner (1978), both the tests are based on the assumption that future cannot cause the past. Also in both of these tests causality can be determined for a group of stochastic processes. It is however, not possible to detect causality between two deterministic processes.

These limitations definitely restrict the range of applicability of the concepts.

Third, both these definitions are non-operational definition involving consideration of predictability in a very special setting. These definitions are not based on economic laws.

Another disadvantage of these tests are that they essentially restrict investigation of causality relative to the information set consisting only of two variables. Also these analyses have not addressed the question of measurement error or added noise.

Further, problem of "spurious causal orderings" can occur if independent variable can be controlled or influenced by someone who does so on the basis of movements in a dependent variable. The "reaction function" can very well dominate the causal findings. Sims (1972) showed that these two tests are equivalent. They are equivalent only in the population, but not in the sample. In a small sample series the two tests might give quite different results. Granger's test might have significant F, while Sims' test can give insignificant F for the same data set. This is because there is a loss of one observation due to the addition of each lag and lead values. In Sims' test the addition of future values consumes too many degrees of freedom which might be the

cause for insignificant F. This is a point worth considering in a small sample. However, Granger's test is intuitively more appealing and straight forward in application. Also, Sims' test (using Box-Jenkins filter) contains a bias in favor of the null hypothesis (Michael, 1978). This is because the filtering estimates the $\emptyset$'s first and then in second stage it estimates the b's yielding biased estimates of b's in general. Granger's test is free from this drawback.

Sims' test has one advantage over Granger's test. In Sims' test multicollinearity could never be a problem as it is in Granger's test. This is because there is no autoregressive parameter in Sims' model. As a result 'contemporaneous causality' can never be a problem in Sims' test. But this could be a problem in Granger's test.

Footnotes to Chapter III

1. Kmenta (1971), p. 367.

2. Gujrati (1978), Pp. 210.

3. According to Bartlett's approximation, standard error for r_j is given by

$$SE\ (r_j) = \frac{1}{\sqrt{T}}\ (1 + 2 \sum_{j=1}^{q} r_j^2)^{1/2},\ j > q \qquad (1)$$

and the rough criterion

$$|r_j| > 2\ \frac{1}{\sqrt{T}}\ (1 + 2 \sum_{j=1}^{q} r_j^2)^{1/2},\ j > q \qquad (2)$$

signifies that q is significant.

CHAPTER IV

EMPIRICAL FINDINGS

4.1: Limitations of Data

The causality tests are run for eight countries, Egypt and Mauritius - Island of Maurice in Africa, India and Srilanka in Asia, Jamaica and Puerto Rico in North America and Columbia and Venezuela in South America. The starting year is 1932 for each country. The final years are however different. The end year is 1968 for Srilanka. For Egypt it is 1974. For Venezuela, Jamaica, India and Columbia the final year is 1976. For Puerto Rico and Mauritius the final year goes upto 1977. Data from 1932 to 1947 are collected from the Demographic Yearbook, 1950, published by the United Nations, New York. Data from 1948 onwards are collected from Demographic Yearbook - Special Issue; Historical Supplement, 1978, the United Nations, New York. However, the Demographic Yearbook does not have data for India for all the years. It has data on India only upto 1964. Indian data for 1964-1970 are collected from Fertility Tables published by the Vital Statistics Division, Office of the Registrar General and Census Commissioner, India. The data for 1971 to 1977 are derived at the U.S. Bureau of Census and published in International Population

Dynamics, 1981. The U.S. Bureau has inflated the reported crude birth rate from the Sample Registration System of India by 5 per cent. The 1973-74 results from the Sample Registration System may have been affected by the postponement of the second half yearly survey of 1973 to the first half yearly survey of 1974, and the suspension of supervisory work during this time period.

The selection of these countries is dictated completely by the availability of data over the entire period under study. The basic characteristic of all these countries is one - they are economically less developed. A comparison among them should tell whether they have the same fertility and infant mortality pattern or not. The data for this study are plotted in Figures B.2.1 through B.2.8 in Appendix B. They illustrate the relatively stable period of crude birth rate and high and unstable period of infant death rate. In all these countries birth rate is more or less stable or decreases very slowly, while the decline in infant mortality rate has been faster compared to birth rate. In general fertility and infant mortality trends follow identical pattern in all countries, as evident from the graphs.

However, the crude nature of these data does not allow for much confidence to be attached to the conclusions derived from the tests performed. They could only serve

as a rough approximation. The demographic yearbook discusses the problems that usually blur the collection of data from the LDCs. Under registration is a serious problem with demographic data of LDCs in general.

There is one additional problem with the data. No data on general fertility rate was available before 1948. Even after 1948 no data on general fertility rate was available for all the years and for all the countries. This deficiency of data was overcome by replacing data on general fertility rate by data on crude birth rate. If the female population of child bearing age remains stable as a ratio of total population, crude birth rate should act as a good proxy for fertility rate. General fertility rate is the number of live births per 1000 female population between ages 15 - 49, whereas crude birth rate is the number of live births per thousand population:

$$GFR = \frac{\text{number of live births}}{\text{number of female population}} \times 1{,}000 \qquad (4.1.1)$$

$$CBR = \frac{\text{number of live births}}{\text{number of female population of child bearing age}}$$

$$\frac{\text{number of female population of child bearing age}}{\text{number of total population}} \times 1{,}000 \qquad (4.1.2)$$

Birth rate trends are affected by factors such as the composition of population by age and sex, the number of married persons, the proportion married at various ages, and the distribution of married population according to number of years married, number of children born previously, and the time elapsed since the last birth. The trend in gross reproduction rate is independent of the age and sex composition of the population. However, in most countries they followed closely the trend in birth rates, showing that the increase in the birth rate was not caused by changes in the age and sex composition (for details see tables on Gross Reproduction Rate and Fertility Rate in Demographic Yearbook, the United Nations). It is possible that a decreasing proportion of women in the reproductive ages to total population would tend to give an exaggerated picture of the fertility decline as measured by birth rate. Table B.1.2 in Appendix B shows female population of child bearing age as a ratio of total population for selected years. This ratio is very stable giving confidence to use crude birth rate as a first overall view of variations in fertility rate. Infant mortality rate is the number of deaths between 0 - 1 year per thousand live births.

$$IMR = \frac{\text{number of deaths between 0-1 year}}{\text{number of live births}} \times 1{,}000 \qquad (4.1.3)$$

4.2: Empirical Results from Granger's Test

Ad hoc lag structure is used in this analysis of lead-lag relationship. The general criterion is to keep adding lagged variables (both own and cross) until the lagged variables become insignificant in terms of the partial F-test. This method was applied to all the countries to determine the relevant lag structure for each equation. But the method failed to produce any significant lag structure that would be acceptable on both statistical and economic grounds. For example, when infant mortality was regressed on lagged infant mortality, none of the lagged values was significant for Egypt, Mauritius, India, Srilanka and Puerto Rico. While regressing infant mortality on lagged fertility, only one or two of the lagged values turned out to be significant. Similar was the result when fertility was regressed on lagged value of itself and lagged infant mortality. Because of the small number of sample size, only upto fifth lag was examined in each case. As they failed to give a significant lag structure, ad hoc lag structure was selected.

Different regressions, using different lags were run for each variable. Out of them the most promising ones, i.e., those regressions which gave significant F-values, were selected and reported here.

Only one country's case is illustrated. This is not identical but typical of the problems with that of all other countries. Let us take the case of Puerto Rico. When fertility was regressed on lagged fertility and lagged infant mortality second, third and fourth own lags were significant, but no cross lag was significant as measured by partial F-values. In case of regression of infant mortality on lagged fertility and lagged infant mortality, however, no own lag was significant and only third and fourth cross lags were significant.

According to Schultz, current reproductive behavior in developing countries, is significantly conditioned by past accumulated reproductive behavior. Hence, it is justified to include own lags although they are not significant. In terms of the econometric method that is pursued here, it is crucial that lagged values of both variables be included. Hence, I have decided on ad hoc lag structure as a prelude to the empirical test.

This procedure of choosing the lag structure may also be supported by Prof. G. B. Wetherill's comment on the paper by Mr. Gomme, Mr. Coen and Dr. Kendall (p. 159, 1969), "... in particular, an equation which made sense physically may be preferred, even if it were not the best."

I have started each regression with two own lags and five cross lags. Two own lags are chosen in preference to only one own lag because information about changes in infant mortality and fertility patterns in any society take time (at least more than a year). Also parents take some time to adjust their reproductive behavior on the basis of the information they receive. In view of these considerations distant lags are more important than immediate past.

Then I added own lags sequentially. Each time I have tested for the significance of the joint values of cross lags (F-test). And if the F-test turned out to be significant, I picked that as the relevant lag structure. If the addition of own lags failed to give significant F, I started to run new tests by altering the cross lags sequentially. So, the method is basically that of trial and error process that is pursued here.

Sometimes combinations of different lags gave significant F-values. In such situations, I have chosen the one with the maximum lags.

The lag structure for each country and each equation is presented below. The relevant lag structure for each country and for each equation is presented in Tables 4.2.1 and 4.2.2. For Egypt fertility rate has been regressed on third and fourth own lags and third, fourth and fifth cross lags, while the infant mortality rate has been regressed on fourth and fifth own lags and second, third, fourth and fifth cross lags. In case of Mauritius, both fertility and infant mortality regressions include first three own lags and first five cross lags. For Srilanka fertility equation has second, third and fourth own lags and upto first four cross lags. Infant mortality equation has first two own lags and first five cross lags. For India the fertility equation has first four own lags and first five cross lags, while the infant mortality equation has second, third, fourth and fifth own and cross lags. In case of Jamaica the fertility equation has first three own lags and first five cross lags and infant mortality equation has second and third own lags and first, second and third cross lags. For Puerto Rico, fertility equation has two own lags (first and second) and upto five cross lags. Infant mortality equation has first, second and third own lags and first five cross lags. Venezuela has a lag structure which includes only first and second own lags and first five cross lags in both fertility and

Table 4.2.1: The Lag Structure for Fertility Equation[1] For Each Country

Countries	Own Lags	Cross Lags
Egypt	(3-4)	(3-5)
Mauritius	(1-3)	(1-5)
Srilanka	(2-4)	(1-4)
India	(1-4)	(1-5)
Jamaica	(1-3)	(1-5)
Puerto Rico	(1-2)	(1-5)
Venezuela	(1-2)	(1-5)
Columbia	(1-4)	(1-5)

1. $F_t = \sum_j a_j F_{t-j} + \sum_j b_j IM_{t-j} + \epsilon_t$

Table 4.2.2: The Lag Structure For Infant Mortality Equation[2] For Each Country

Countries	Own Lag	Cross Lags
Egypt	(4-5)	(2-5)
Mauritius	(1-3)	(1-5)
Srilanka	(1-2)	(1-5)
India	(2-5)	(2-5)
Jamaica	(2-3)	(1-3)
Puerto Rico	(1-3)	(1-5)
Venezuela	(1-2)	(1-5)
Columbia	(1-3)	(1-5)

2. $$IM_t = \sum_j c_j \, IM_{t-j} + \sum_j d_j \, F_{t-j} + n_t$$

infant mortality equations. Lastly, Columbia has first four own lags and first five cross lags in fertility equation and first three own lags and first five cross lags in infant mortality equation.

In all these equations fewer own lags are chosen compared to cross lags. That should be alright in view of the multicollinearity problem which shows up if more own lags are chosen. The tables of coefficients are presented in Appendix A, tables A.4.2.1 to A.4.2.16. Tables 4.2.3 and 4.2.4 show results of F-tests in Granger causality. In Table 4.2.3 F_A is a test of the null hypothesis that infant mortality does not affect fertility ($IM \not\rightarrow F$). The null hypothesis has been rejected for six countries. Hence we conclude that in these countries infant mortality affects fertility. The countries are Egypt, Mauritius, Srilanka, Jamaica, Puerto Rico and Venezuela. There is no causality from infant mortality to fertility in India and Columbia. Table 4.2.4 shows the F-values when fertility affects infant mortality. F_B is a test of the null hypothesis that fertility does not affect infant mortality ($F_t \not\rightarrow IM_t$). This has been rejected for five countries. On the basis of significant F-tests we conclude that fertility affects infant mortality in the following five countries viz, Egypt, Srilanka, India, Jamaica and Columbia.

Table 4.2.3: F-Values in Granger's Test of Causality

Countries	FA (IM → F)	Degrees of Freedom
Egypt	3.19*	(3,21)
Mauritius	2.38*	(3,32)
Srilanka	4.16*	(3,24)
India	0.52	(4,30)
Jamaica	2.29*	(3,30)
Puerto Rico	2.92*	(2,32)
Venezuela	2.40*	(2,31)
Columbia	1.84	(4,28)

* implies the F-tests are significant at 10%.

Note: F_A is a test of the null hypothesis that the parameters of lagged infant mortality in equation (3.1.1) are zero, as a group.

Table 4.2.4: F-Values in Granger's Test of Causality

Countries	FB (F → IM)	Degrees of Freedom
Egypt	3.82*	(2,30)
Mauritius	1.82	(3,30)
Srilanka	2.85*	(2,23)
India	3.73*	(4,30)
Jamaica	2.94*	(2,35)
Puerto Rico	0.84	(3,31)
Venezuela	1.16	(2,31)
Columbia	3.32*	(3,29)

* implies the F-tests are significant at 10%.

Note: F_B is a test of the null hypothesis that the parameters of lagged fertility rate in equation (3.1.2) are zero, as a group.

When both way causality could be found the situation is known as feedback relationship. In this analysis feedback relationship could be found for three countries which are Egypt, Srilanka, and Jamaica.

Although infant mortality affects fertility in six countries, the economic explanation would not be the same. For Asian and African countries, where breast feeding is still prevalent it may be the interval effect which is at work benind the causality from infant mortality to fertility. However, in Latin American countries resort to formula feeding has become more and more common. In these countries the effect may be said to operate via replacement and/or hoarding.

When we observe causality running from fertility to infant mortality, the countries that have significant F-values are the ones that have lowest per capita income in rank (See Appendix B, Table B.1.1 for estimates of per capita income for selected years). This may be explained either as a direct corrective to excessive births or as an indirect consequence of the pressure on real income, nutrition and health. The countries where fertility does not affect infant mortality are Puerto Rico, Venezuela and Mauritius. Of these countries the per capita income is much above that of LDC average in Puerto Rico and Venezuela. This might prevent the death toll among

infants from being high due to high fertility.

4.3: Sign of Direction of Causality

Let us consider equations (3.1.1) and (3.1.2) once again ignoring the intercept terms.

$$F_t = \Sigma\ a_j\ F_{t-j} + \Sigma\ b_j\ IM_{t-j} + E_t \qquad (3.1.1)$$

$$IM_t = \Sigma\ c_j\ IM_{t-j} + \Sigma\ d_j\ F_{t-j} + n_t \qquad (3.1.2)$$

Let us suppose $IM_{t-1} = IM_{t-2} = \ldots = IM^*$. This may be viewed as the long run value of IM. Also let $F_{t-1} = F_{t-2} = \ldots = F^*$. Then,

$$F_t = \Sigma\ a_j\ F_{t-j} + (\Sigma\ b_j)\ IM^* \qquad (4.3.1)$$

$$IM_t = \Sigma\ c_j\ IM_{t-j} + (\Sigma\ d_j)\ F^* \qquad (4.3.2)$$

Equation (4.3.1) provides a rationale for summing the coefficients ($\Sigma\ b_j$) to determine the sign and magnitude of causality. ($\Sigma\ b_j$) shows the effect on F_t of a one-unit increase in the long run value of IM* with F_{t-j} held constant. Similarly, if we assume $F_{t-1} = F_{t-2} = \ldots = F^*$ ($\Sigma\ d_j$) shows the effect of a one-unit increase in the long run value of F* with IM_{t-j} held constant. The t-values are then calculated to find out the significance of the summed coefficients. To determine the t-values we need

to know the standard deviation. The standard error is calculated by taking the squre root of the variance which is obtained from the variance - covariance matrix of the coefficients computed by the computer.

One problem[2] with summing the coefficients is that it assumes ΣF_{t-j} in equation (4.3.1) to be fixed. But they will vary as IM* varies. Therefore, let $F^* = F_t = F_{t-1} = F_{t-2} = , \ldots,$ be the long run value of F. Substituting F* into equation (4.3.1) one obtains:

$$F^* = \frac{\Sigma b_j}{(1-\Sigma a_j)} \quad IM^* \quad \ldots \qquad (4.3.3)$$

Let, $\frac{\Sigma b_j}{(1-\Sigma a_j)} = r_j$. Hence, r_j is a more comprehensive measure of the sign and magnitude of direction of causality. When applied to equation (4.3.2), $r_j = \frac{\Sigma c_j}{(1-\Sigma d_j)}$. Hence, the sign and magnitude of the direction of causality depends on the t-tests derived on the basis of r_j. I have computed the t-values for both the summed coefficients (Σb_j and Σd_j) and the r_j's to determine the sign and direction of causality[3]. The results are reported in Tables 4.3.1 through 4.2.4 Tables 4.3.1 report the t-statistics for r_j when IM → F. Table 4.3.2 shows the t-values for r_j

Table 4.3.1 Signs of Direction of Causality in Granger's Test For The Countries With Significant F-Values

Causality From Infant Mortality To Fertility (IM → F)

Countries	r_j	Standard Error	T-Statistics	Degrees of Freedom
Egypt	-0.282	0.163	-1.84*	31
Mauritius	2.112	4.682	0.45	32
Srilanka	-0.68	1.12	-0.605	24
Jamaica	1.01	3.36	0.29	30
Puerto Rico	0.012	0.22	0.08	32
Venezuela	-0.26	0.25	-1.32	31

* significant at 5% for one-tailed test

Table 4.3.2: Signs of Direction of Causality In Granger's Test For The Countries With Significant F-Values.

Causality From Fertility To Infant Mortality (F → IM)

Countries	r_j	Standard Error	T-Statistic	Degrees of Freedom
Egypt	0.184	0.26	0.70	30
India	0.317	0.186	1.7*	31
Srilanka	0.006	0.33	0.20	25
Jamaica	0.146	0.13	1.07	35
Columbia	0.981	0.974	1.006	29

* significant at 5% for one-tailed test.

Table 4.3.3 Signs of Direction of Causality by the Simple Method of Summing Up The Coefficients

Causality From Infant Mortality To Fertility (IM → F)

Countries	β_j	Standard Deviation	T-Statistics	Degrees of Freedom
Egypt	-0.448	0.78	-0.574	31
Mauritius	2.668	1.84	1.45*	32
Srilanka	-0.681	1.24	-0.55	24
Jamaica	2.709	5.14	0.53	30
Puerto Rico	0.014	0.181	0.08	32
Venezuela	-0.29	0.24	-1.21	31

* significant at 5% level in a one-tailed test run to determine whether the T-statistic is greater than zero or not.

Table 4.3.4: Signs of Direction of Causality by the Simple Method of Summing Up The Coefficients.

Causality From Fertility To Infant Mortality (F → IM)

Countries	Σd_j	Standard Error	T-Statistics	Degrees of Freedom
Egypt	0.208	0.184	0.247	30
India	0.486	0.182	2.67*	31
Srilanka	0.005	0.199	0.02	25
Jamaica	0.146	0.067	2.18*	35
Columbia	1.563	1.04	1.50*	29

* significant at 5% level in a one tailed test.

when F→IM. Table 4.3.3 shows the T-values for $\Sigma \beta_j$ when IM → F and finally Table 4.3.4 shows the T-values for Σd_j when F → IM.

In Tables 4.3.1 and 4.3.2, the T-test is a test of the null hypothesis that r_j is not significantly different from zero. The alternative hypothesis is that r_j is greater than zero. If the T-test is rejected we conclude that the sign of direction of causality is significant. In Table 4.3.3 the null hypothesis is that $\Sigma \beta_j$ is not significantly different from zero, while in Table 4.3.4, the null hypothesis is that $\Sigma d_j = 0$.

However, I have calculated the signs of the directions of causality for only those countries in which F-values are significant.

As revealed in Table 4.3.1, when IM → F, the signs of direction of causality are positive in cases of Mauritius, Jamaica and Puerto Rico and the signs are negative for the countries of Egypt, Srilanka and Venezuela. But the t-statistic is significant for only one country, i.e., Egypt. Both positive and negative signs fit a priori expectations. Parents whose children die often try to replace them; and where infant mortality is common, social norms tend to encourage "insurance" against the expected loss of children. Similarly, if infant mortality goes down, that decline may itself cause fertility decline in LDCs. Since the motives for having large families would be satisfied at lower levels of fertility. A smaller number of births would provide an adequate supply of child labor during seasons of peak requirements of agricultural labor. If the probability of each child's survival increases, a smaller number is required to insure support for parents in old age. Hence, we would expect a positive sign. However, the sign can also be negative if causality runs from infant mortality to fertility. This is possible if quality is substituted for quantity or if the price elasticity of demand for children exceeds unity. When we summed up the cross lagged

coefficients only and performed the same test, it shows (Table 4.3.3) Srilanka and Venezuela have negative signs. All other countries have positive signs.

When causality goes from fertility to infant mortality, the signs are positive for all five countries by both tests fitting with a priori theoretical arguments. The results are shown in Tables 4.3.2 and 4.3.2. High fertility contributes to high infant mortality because many births, specifically if they are close together, can weaken both mother and child thereby increasing the risk of infant deaths. Also the low per capita income causes malnutrition and health hazards which are strong reasons for high infant deaths in LDCs. But although the sign is positive, it is significant only for India by the first test ($r_j = 0$). While the second test ($\Sigma \beta_j = 0$) shows the signs are positive for three out of the five countries, viz; Jamaica, Columbia and India.

Again these findings could be justified on the basis of the characteristics of the LDCs. The majority of the population still have very limited access to modern and simple means of contraceptions. The contraceptives are often expensive in relation to the income of the poor. In some circumstances the psychological and financial costs of avoiding pregnancy may exceed the cost of having another child. Hence, although infant mortality affects fertility, it could not exert any significant impact so

far as the direction of causality is concerned. High fertility rather has significant impact on infant mortality because the per capita income in most of these countries is very low. This acts as a hinderance to proper medical care, nutrition, etc. This exerts a significant damage to infant's health and causes high infant mortality.

In this context it might also be mentioned that the finding in Puerto Rico is in line with the Nerlove and Schultz[1] (1970) study of Puerto Rico during 1950 - 1960. In their study they found that in Puerto Rico infant mortality affects fertility, but fertility has no impact on infant mortality.

4.4: Empirical Findings From Sims' Test

In order to run Sims' test the original time series for both fertility rate and infant mortality rate have been transformed to white noise series through ARIMA filters. The Box-Jenkins forecasting methodology, described in Chapter 3, is applied to form the ARIMA model. The estimated ARIMA structures are reported in Tables A.4.3.1 through A.4.3.8 in Appendix A. In Panel A, sample autocorrelation and partial autocorrelation coefficients are given. In Panel B, the figures within the parentheses give the T-ratios for the coefficients. Each ARIMA structure is chosen on the basis of Q-statistics. Repeated diagnostic checking through over fitting could not improve the results.

The whitened series thus generated are expected to be jointly covariance stationary. These series are then estimated to detect causality between infant mortality and fertility and vice versa. In the estimation process two sided distributed lags are chosen for both equations.

Once again the Lead-lag structures are ad hoc. For Egypt and Srilanka two lagged values (2,3) and three lead values (1,2,) are chosen when causality is tested from fertility to infant mortality. For Mauritius two lagged values (3,4) and four lead values (1,2,3,4) are relevant. For India and Jamacia fourth and fifth lagged values and five lead values are chosen. For Puerto Rico three lagged values (2,3,4) and five lead values (1 to 5) are selected. For Columbia and Venezuela lagged values are the same (3,4), while the lead values run from one to five and one to four respectively.

When we consider causality from infant mortality to fertility the lag structure runs as follows:

For Egypt two lagged values (2,3) and three lead values (1-3) are chosen. For Mauritius two lagged values (3,4) and four lead values (1-4) are selected. For India the same lag structure is estimated as in the case of Mauritius. For Srilanka, Jamaica and Columbia, the lag structure is the same. In each of these countries two lagged values (4,5) and five lead

values are chosen. For Venezuela, two lagged values (1-4) are selected. Lastly, for Puerto Rico two lagged values (1,2) and five lead values (1-5) are chosen.

Tables 4.4.1 and 4.4.2 give the F-tests for Sims' Causality. In Table 4.4.1, when causality from infant mortality to fertility is considered, the null hypothesis of no causality is rejected for four countries. F_A is a test of the null hypothesis that the lead coefficients of fertility are zero as a group. The countries for which F_A turned out to be significant are: Egypt, Mauritius, India and Columbia. However, no such causality could be detected in Jamaica, Puerto Rico, Srilanka and Venezuela.

These results are at variance with Granger's test using first differenced model for six countries, viz; India, Srilanka, Puerto Rico, Jamaica, Venezuela and Columbia. In Granger's test infant mortality caused fertility both in Jamaica, Puerto Rico and Venezuela but no such causality could be established on the basis of Sims' test. The reverse is true for India and Columbia. In these countries, Sims' test shows causality running from infant mortality to fertility, whereas no such causality was found on the basis of Granger's test.

Table 4.4.1: The F-Values in Sims' Test of Causality

Countries	F_A^1 (IM → F)	Degrees of Freedom
Egypt	2.65*	(3,26)
Mauritius	2.47*	(3,25)
India	2.21*	(3,23)
Srilanka	1.66	(3,17)
Jamaica	1.74	(4,24)
Puerto Rico	0.92	(4,26)
Venezuela	1.72	(3,28)
Columbia	2.9*	(3,25)

*significant at 10%.

1. $IM_t = b_o + \sum_{s=-e_1}^{e_2} b(s)\, F_{t-s} + n_t$

Table 4.4.2: The F-Values in Sims' Test of Causality

Countries	F_B^2 (F → IM)	Degrees of Freedom
Egypt	1.06	(4, 22)
Mauritius	2.96*	(3, 26)
Srilanka	1.75	(3, 17)
India	1.21	(3, 22)
Puerto Rico	3.36*	(2, 27)
Jamaica	1.54	(4, 24)
Venezuela	0.60	(3, 28)
Columbia	1.73	(3, 25)

*significant at 5%.

2. $F_t = a_o + \Sigma\, a(s)\ IM_{t-s} + e_t$

Let us now discuss the causality from fertility to infant mortality. The F-tests are reported in Table 4.4.2. F_B is a test of the null hypothesis that the future coefficients of infant mortality are not significantly different from zero as a group. Where the null hypothesis is rejected we conclude that fertility affects infant mortality.

The F's are significant only in Mauritius and Puerto Rico. Once again the results are at variance with Granger's test where no such causality was evident for either of the two countries. In none of the five countries viz; Egypt, Srilanka, India, Jamaica, and Columbia does Sims' test give significant F-statistics, whereas significant causality was detected in Granger's test. The tables of coefficients are presented in Appendix A, tables A.4.3.9 through A.4.3.24.

4.5: Signs of Direction of Causality in Sims' Test

To determine the sign and magnitude of direction of causality[3] in Sims' test, we sum up the lagged values of infant mortality, when causality runs from infant mortality to fertility. Similarly, if fertility affects infant mortality, the lagged values of fertility are summed up. When feedback relationship exists (IM $\overleftarrow{\rightarrow}$ F), the lead values of the coefficients in equation (3.2.1) are summed up to yield proper sign from fertility to infant mortality. And when infant mortality causes fertility the

lead values of the coefficients in equation (3.2.2) are summed up. The standard error for each summed coefficient is calculated from the variance-covariance matrix of the coefficients. The resulting statistics are reported in Table 4.5.1. The null hypothesis is that the sum of the coefficients is zero. If the null hypothesis is rejected, the sign is significant.

When causality runs from infant mortality to fertility, the sign of the sum of the coefficients is positive for all four countries, viz; Egypt, India, Mauritius and Columbia. But the t-statistic is significant for only one country, i.e., Columbia as shown in Panel A.

Next, let us consider causality from fertility to infant mortality. The sign turns out to be negative for each country. This is a disturbing result in the sense that fertility is said to affect infant mortality in the same direction, a priori. However, none of these t-values is significant as presented in Panel B.

Table 4.5.1: Signs of Direction of Causality in Sims' Test

Panel A: (IM → F)

Countries	Summed Coefficient	Standard Error	T-Statistics
India	0.219	0.496	0.44
Mauritius	3.124	2.45	1.27
Egypt	0.923	1.174	0.786
Columbia	1.193	0.736	1.62*

Panel B: (F → IM)

Countries	Summed Coefficient	Standard Error	T-Statistics
Mauritius	-0.028	0.046	-0.599
Puerto Rico	-0.004	0.106	-0.037

4.6: Empirical Findings From Granger's Test With A Linear Trend Only

As an alternative method I have run Granger's causality using the logarithms of original data and with a linear trend term only, but no first differencing. The results of the F-tests are reported in Table 4.6.1.

I have used uniform lag structure for each country and each equation. Each equation consists of three own lags and five cross lags.

As revealed from Table 4.6.1, there is no causality from infant mortality to fertility. Only one way causality is found from fertility to infant mortality. This one way causality is true for India, Venezuela, Columbia, Srilanka, and Mauritius. This result supports the Malthusian notion which states excess fertility results in high mortality. Once again, the sign and magnitude of direction is insignificant (not shown).

Table 4.6.1: The F-Values in Granger Causality With A Linear Trend Only.

Countries	F_A[1] (IM → F)	F_B[2] (F → IM)	Degrees of Freedom
Egypt	1.42	0.79	(4, 28)
Mauritius	1.14	3.04*	(4, 31)
India	0.39	3.71**	(4, 30)
Srilanka	1.61	2.39*	(4,22)
Puerto Rico	0.82	0.44	(4, 31)
Jamaica	1.50	1.94	(4, 30)
Venezuela	1.04	2.14*	(4, 30)
Columbia	0.74	2.79**	(4, 29)

* significant at 10%

**significant at 5%

$$1.\quad F_t = \alpha_o + \sum_{j=1}^{3} a_j F_{t-j} + \sum_{j=1}^{5} b_j IM_{t-j} + L_t + e_t$$

$$2.\quad IM_t = \beta_o + \sum_{j=1}^{3} c_j IM_{t-j} + \sum_{j=1}^{5} d_j F_{t-j} + L_t + n_t$$

4.7: A Comparison With Causal Patterns In The Developed Countries

Tadashi Yamada (1981) has conducted a test of causality between infant mortality and fertility in seventeen industrialized countries of the world. A comparison of his findings with the present study is warranted both to compare how does fertility determination in the developed countries differ from that in the underdeveloped countries and also how do we account for the difference.

In his study, Yamada included the following countries: Israel, Canada, the United States, Japan, Taiwan, Australia, New Zealand and ten countries in Western Europe. In his analysis, significant causality from infant mortality to fertility was evident in Denmark, Finland, France, Italy, the Netherlands, Portugal, Sweden, Switzerland, the United Kingdom, Israel, the U.S.A. and Australia. The signs of direction of causality were positive for France, Italy, the Netherlands, Portugal and Sweden. For the rest of the countries the sign appeared to be negative.

When causality was run from fertility to infant mortality, the F-test was significant for Finland, France, the Netherlands, Norway, Switzerland, the United Kingdom, Israel, Canada, Japan, Taiwan, Australia and New Zealand. Feedback relationship was found for Finland, France, the Netherlands, Switzerland, the United Kingdom, Israel and Australia.

On the basis of these results Yamada concluded that infant mortality should be considered as an endogenous variable in the determination of a general model for fertility. However, the results are not basically different from that of the LDCs where infant mortality affected fertility in six out of eight countries by first differencing method and was also affected by it in five countries. Hence, we could conclude that economic theory explaining fertility behavior in the developed countries, is equally applicable to the LDCs. But as it now stands, economic role of the household in explaining fertility is probably more important in the LDCs than in the developed countries in view of the changes that are taking place in the LDCs. However, in the case of LDCs Malthusian theory might be said to be exerting stronger influence. This is more so when we consider causality with a linear trend term without first differencing.

Footnotes to Chapter IV

1. Schultz, T. W., ed. (1974): P. 6.

2. This point was suggested by my dissertation supervisor Prof. Michael Grossman.

3. For an exposition, see Kmenta (1974): p. 444.

4. I am indebted to my friend Dr. Tadashi Yamada for this method.

CHAPTER V

CONCLUDING REMARKS

The chief emphasis in this research was to determine the sign and direction of causality between fertility and infant mortality in the LDCs. The techniques used were Granger and Sims causality. The major contribution of this thesis is that it has focused on an issue which is very important for the LDCs, yet the least studied. Also, the study uses time series data which are much more relevant in analyzing cause and effect relation of the kind explained in preceding chapters. So far little attention has been given to economic theory and statistical techniques that explain models of dynamic behavior. However, to test the existing theories meaningfully longitudinal data would be much preferred. In the absence of any such data, aggregate time series data are used.

This study has revealed that in the less developed countries infant mortality is a strong determinant of fertility behavior of parents. In Granger's test, using first difference model, infant mortality has been found to cause fertility in six out of the eight countries studied. The two countries are India and Columbia where

infant mortality has no significant effect on fertility. Sims' test lends support to first difference model of Granger's test for Egypt and Mauritius. But for Puerto Rico, Venezuela, Jamaica, Columbia and India the findings of Sims' test are in contradiction with Granger's test. In Puerto Rico, Jamaica and Venezuela, there is no causal relationship from infant mortality to fertility in Sims' test, whereas in India and Columbia we find causality running from infant mortality to fertility. As regards the signs of direction of causality, Granger's test gives positive signs for Mauritius, Jamaica, Puerto Rico and Columbia, while negative signs are observed for Venezuela, Srilanka and Egypt. As regards Sims' test the direction of causality are positive for all four countries.

Both positive and negative signs fit a priori expectations. Usually with higher (lower) infant mortality fertility would go up (down) because hoarding and replacement strategies require parents to reach their family size goal with more (fewer) births. However, as infant mortality goes down the cost of each child decreases too. Hence parents might have a tendency to substitute children (number) for other goods that enter their utility function. In this case we might observe a negative relationship (i.e., as IM $\downarrow$, F $\uparrow$), if price elasticity of demand for children is greater than one. Also, if

infant mortality goes up parents might revise their family size goal downwards if they do not want to experience the loss of children again. In other words, the psychic cost of birth rises with an increase in infant mortality rate. This increase in psychic cost might lower the birth rate as well.

As regards causality running in the reverse order, say, from fertility to infant mortality, Granger's test using first difference model, shows significant results in five countries viz; Egypt, India, Srilanka, Jamaica and Columbia. And the signs of direction of causality are positive for all these countries once again fitting a priori expectations. The causality from fertility to infant mortality is not significant in Venezuela, Puerto Rico and Mauritius.

All these countries have a high fertility rate per woman, but per capita income has a very different range. Puerto Rico and Venezuela have per capita income much above that of the LDC average (see Appendix Table B.1.1). Probably due to high per capita income the nutritional level is high and this makes possible higher medical facilities and better sanitation which could prevent fertility from exerting its influence on infant mortality. Although per capita income in Mauritius is not as high, fertility does not affect infant mortality in Mauritius either.

As an alternative method to test Granger causality we have used a model with only a linear trend term and no differencing. The result shows there is no causality from infant mortality to fertility in any of these countries. But it shows a definite causal link from fertility to infant mortality in five countries. The countries are: Mauritius, India, Srilanka, Venezuela and Columbia.

So far the sign of direction of causality is concerned it appears to be insignificant by either test for most of the countries. This may be accounted for the presence of multicollinearity or fairly short time series or even misspecified lag structure.

When we consider the direction of causality from fertility to infant mortality, the first difference model of Granger's test gives significant result for three out of five countries. This is in line with other empirical research. Various studies have already shown that the reasons for infant deaths in LDCs are malnutrition, poor health of mothers, unhygienic living conditions, poor sanitation, etc.. All of these are results of too many births as was first conceded two centuries ago by Thomas Robert Malthus.

The economic frustrations experienced by many LDCs are simply due to ever increasing population. The perception that economic progress attained was not keeping pace with population directed concern toward the population control program. But before any step is taken to control population it is essential to determine what are the factors that affect fertility. This study completed a project that is directly relevant toward this goal. It has shown that infant mortality affects fertility in the LDCs. It does in no sense claim that it is the only determinant of fertility nor that it is the most important determinant of fertility.

On the other hand infant mortality is affected by fertility. Infant mortality rate itself is the indicator of economic growth. The less developed a country is, the higher is the infant mortality rate. Fertility control should result in immediate and remarkable decline in infant mortality rate. The implication of this empirical finding is of particular importance for policy parameters. It follows that in spite of the improved health measures in the last two decades in the LDCs, a more significant result is yet to be achieved by population control policy.

As as been pointed out earlier, the scope of this particular research is limited to one aspect of the population problem of the LDCs. Research could also be directed to find out the differential fertility and infant mortality rates in different regions or sub-regions of the LDCs. There have been some studies on Puerto Rico and Jamaica in that direction. But for most of the countries systematic regional data are not available as yet and hence research in that direction is halted. Yet there is an urgent need for much experimentation and statistical work to explore the intricate areas of human behavior in the LDCs striving for economic development.

APPENDIX A

General Notes to Appendix A: Tables of Coefficients.

1. The 'B' associated with the variables in Granger's Test, denotes the lag operator. The Superscripts denote the degree of the lag operators.
2. F in the tables of coefficients in Granger's Test stands for first differences of the logarithms of the original series on birth rate. IM is the first differences of the logarithms of the original series on infant mortality rate.
3. In the tables of coefficients for Sims' Test, Lag and Led refer to lagged and leading operators respectively. The subscripts refer to the degree of lag and lead operators respectively. In these equations BR and IM are white noise series of fertility and infant mortality respectively.
4. In tables for ARIMA models,
   ARSUP = Suppressed Autoregressive Parameters
   MASUP = Suppressed Moving Average Parameters
5. The relevant equation for each country and each regression appears right below each table.
6. All figures are rounded to three decimal points or the nearest possible point after that. That might

cause some discrepancies with the reported T-statistics calculated by the computer more accurately (upto 8 decimal points).

7. The general form of the ARIMA structure is:

$$\Delta^d x_t = \alpha + \phi_1 \Delta^d x_{t-1} + \ldots + \phi_p \Delta^d x_{t-p} - \theta_1 u_{t-1} \ldots$$

$$\theta_q u_{t-q} + u_t$$

The ARIMA structure in each table defines the values of the indices, d.

Table A.4.2.1: Tables of Coefficients for Egypt. Granger's Test

Independent Variables	Estimated Coefficients	Standard Error	T-Statistics
FB^3	-0.28	0.177	-1.589
FB^4	-0.306	0.179	-1.715
IMB^3	-0.099	0.099	-1.00
IMB^4	-0.272	0.108	-2.507*
IMB^5	-0.077	0.09	-0.853

$$F_t = \alpha_o + \sum_{j=3}^{4} a_j F_{t-j} + \sum_{j=3}^{5} b_j IM_{t-j} + E_t$$

Table A.4.2.2: Tables of Coefficients for Egypt: Granger's Test

Independent Variables	Estimated Coefficients	Standard Error	T-Statistics
IMB^4	0.236	0.222	1.062
IMB^5	-0.368	0.217	-1.697
FB^2	-0.239	0.342	-0.698
FB^3	0.708	0.341	2.074*
FB^4	0.397	0.387	1.027
FB^5	-0.658	0.394	-1.669

$$IM_t = \beta_o + \sum_{j=4}^{5} c_j \, IM_{t-j} + \sum_{j=2}^{5} d_j \, F_{t-j} + n_t$$

Table A.4.2.3: Tables of Coefficients for Mauritius. Granger's Test

Independent Variables	Estimated Coefficients	Standard Error	T-Statistics
FB^1	-1.09	1.15	-0.947
FB^2	-0.203	1.201	-0.169
FB^3	1.035	1.218	0.849
IMB^1	0.679	0.567	1.197
IMB^2	-0.268	0.63	-0.425
IMB^4	0.839	0.628	1.334
IMB^5	1.113	0.519	2.146*

$$F_t = \alpha_o \sum_{j=1}^{3} a_j F_{t-j} + \sum_{j=1}^{5} b_j IM_{t-j} + E_t$$

Table A.4.2.4: Tables of Coefficients for Mauritius. Granger's Test

Independent Variables	Estimated Coefficients	Standard Error	T-Statistics
IMB^1	0.729	0.639	1.14
IMB^2	-0.439	0.674	-0.65
IMB^3	0.319	0.637	0.499
FB^1	-0.51	1.312	-0.389
FB^2	-1.092	1.371	-0.796
FB^3	0.771	1.362	0.566
FB^4	0.867	1.36	0.638
FB^5	-2.085	1.112	-0.187

$$IM_t = \beta_o + \sum_{j=1}^{3} c_j \, IM_{t-j} + \sum_{j=1}^{5} d_j \, F_{t-1} + n_t$$

Table A.4.2.5: Tables of Coefficients for Srilanka. Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
FB^2	-0.0001	0.208	-0.0007
FB^3	0.244	0.226	1.076
FB^4	0.318	0.182	1.748
IMB^1	-0.153	0.073	-2.088*
IMB^2	0.067	0.065	1.028
IMB^3	-0.0002	0.072	-0.003
IMB^4	0.091	0.064	1.412

$$F_t = \alpha_o + \sum_{j=2}^{4} a_j F_{t-j} + \sum_{j=1}^{4} b_j IM_{t-j} + E_t$$

Table A.4.2.6: Tables of Coefficients for Srilanka. Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
IMB^1	0.078	0.204	0.381
IMB^2	-0.197	0.166	-1.185
FB^1	-0.479	0.475	-1.007
FB^2	0.058	0.439	0.132
FB^3	0.282	0.408	0.69
FB^4	-0.548	0.399	-1.37
FB^5	0.006	0.398	0.014

$$IM_t = \beta_o + \sum_{j=1}^{2} C_j \, IM_{t-j} + \sum_{j=1}^{5} d_j \, F_{t-j} + n_t$$

Table A.4.2.7: Table of Coefficients for India.
Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
FB^3	-0.082	0.161	-0.509
FB^4	-0.234	0.173	-1.351
FB^5	0.456	0.169	2.692*
IMB^2	0.291	0.451	0.648
IMB^3	-0.422	0.454	-0.928
IMB^4	-0.263	0.428	-0.615
IMB^5	-0.276	0.432	-0.637

$$F_t = \alpha_o + \sum_{j=3}^{5} a_j F_{t-j} + \sum_{j=2}^{5} b_j IM_{t-j} + E_t$$

Table A.4.2.8: Tables of Coefficients for India. Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
IMB^2	-0.233	0.176	-1.323
IMB^3	0.0006	0.171	0.003
IMB^4	0.073	0.161	0.454
IMB^5	-0.079	0.162	2.759*
FB^2	0.096	0.065	1.472
FB^3	0.096	0.065	1.472
FB^4	0.104	0.069	1.519
FB^5	0.116	0.065	1.777

$$IM_t = \beta_o + \sum_{j=2}^{5} C_j \, IM_{t-j} + \sum_{j=2}^{5} d_j \, F_{t-j} + n_t$$

Table A.4.2.9: Tables of Coefficients for Puerto Rico: Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
FB^1	0.171	0.169	1.009
FB^2	0.026	0.174	0.152
IMB^1	0.070	0.065	1.075
IMB^2	0.06	0.062	0.978
IMB^3	-0.093	0.062	-1.489
IMB^4	-0.016	0.062	-0.252
IMB^5	-0.007	0.058	-0.128

$$F_t = \alpha_o + \sum_{j=1}^{2} a_j F_{t-j} + \sum_{j=1}^{5} b_j IM_{t-j} + E_t$$

Table A.4.2.10: Tables of Coefficients for Puerto Rico. Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
IMB^1	-0.193	0.167	-1.157
IMB^2	-0.209	0.159	-1.312
IMB^3	-0.237	0.162	-1.457
FB^1	0.516	0.443	1.164
FB^2	0.042	0.451	0.093
FB^3	-0.323	0.443	-0.728
FB^4	-0.084	0.419	-0.20
FB^5	-0.226	0.387	-0.584

$$IM_t = \beta_o + \sum_{j=1}^{2} c_j \, IM_{t-j} + \sum_{j=1}^{5} d_j \, F_{t-j} + n_t$$

Table A.4.2.11: Tables of Coefficients for Jamaica: Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
FB^1	-1.001	0.213	-4.701*
FB^2	-0.565	0.264	-2.140*
FB^3	-0.142	0.203	-0.701
IMB^1	-2.441	1.499	-1.62
IMB^2	-0.235	1.791	-0.13
IMB^3	2.131	1.749	1.22
IMB^4	2.201	1.482	1.480
IMB^5	1.053	1.408	0.75

$$F_t = \alpha_o + \sum_{j=1}^{3} a_j F_{t-j} + \sum_{j=1}^{5} b_j IM_{t-j} + E_t$$

Table A.4.2.12: Tables of Coefficients for Jamaica. Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
IMB^2	-0.087	0.202	-0.432
IMB^3	0.079	0.199	0.399
FB^1	0.059	0.025	2.40*
FB^2	0.054	0.034	1.56
FB^3	0.033	0.028	1.19

$$IM_t = \beta_o + \sum_{j=2}^{3} c_j \, IM_{t-j} + \sum_{j=1}^{3} d_j \, F_{t-j} + n_t$$

Table A.4.2.13: Tables of Coefficients for Venezuela: Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
FB^1	-0.248	0.165	-1.503
FB^2	0.179	0.138	1.301
IMB^1	-0.152	0.088	-1.729
IMB^2	0.025	0.093	0.272
IMB^3	0.008	0.091	0.084
IMB^4	-0.057	0.092	-0.617
IMB^5	-0.112	0.091	-0.241

$$F_t = \alpha_o + \sum_{j=1}^{2} a_j F_{t-j} + \sum_{j=1}^{5} b_j IM_{t-j} + E_t$$

Table A.4.2.14: Tables of Coefficients for Venezuela. Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
IMB^1	-0.242	0.188	-1.306
IMB^2	0.077	0.191	0.404
FB^1	-0.087	0.348	-0.251
FB^2	0.048	0.279	0.171
FB^3	-0.307	0.289	-1.061
FB^4	-0.247	0.286	-0.864
FB^5	0.112	0.289	0.387

$$IM_t = \beta_o + \sum_{j=1}^{2} c_j \, IM_{t-j} + \sum_{j=1}^{5} d_j \, F_{t-j} + n_t$$

Table A.4.2.15: Tables of Coefficients for Columbia: Granger's Test

Independent Variables	Estimated Coefficient	Standard Error	T-Statistics
FB^1	-0.629	0.171	-3.687*
FB^2	-0.332	0.144	-1.715
FB^3	-0.356	0.202	-1.765
FB^4	-0.371	0.175	-2.125*
IMB^1	-0.004	0.099	-0.036
IMB^2	-0.142	0.134	-1.061
IMB^3	0.166	0.176	0.944
IMB^4	0.200	0.169	1.183
IMB^5	0.235	0.148	1.586

$$F_t = \alpha_o + \sum_{j=1}^{4} a_j F_{t-j} + \sum_{j=1}^{5} d_j IM_{t-j} + E_t$$

Table A.4.2.16: Tables of Coefficients for Colombia. Granger's Test

Independent Variables	Estimated Coefficients	Standard Error	T-Statistics
IMB^1	-0.338	0.158	-2.138*
IMB^2	-0.341	0.213	-1.601
IMB^3	-0.506	0.269	-1.877
FB^1	0.089	0.290	0.309
FB^2	0.093	0.321	0.289
FB^3	0.401	0.377	1.122
FB^4	0.258	0.332	0.777
FB^5	0.848	0.299	2.838*

$$IM_t = \beta_o + \sum_{j=1}^{3} c_j \, IM_{t-j} + \sum_{j=1}^{5} d_j \, F_{t-j} + n_t$$

Table A.4.3.1: Estimated ARIMA Structure for Egypt; 1932 - 1974.

Panel A: Autocorrelation and Partial Autocorrelation; Lags 1 - 5 Years

	Autocorrelation					Partial Autocorrelation				
Variable	1	2	3	4	5	1	2	3	4	5
Fertility	-0.17	0.17	-0.12	-0.10	-0.00	-0.17	-0.15	0.15	-0.07	-0.16
Infant Mortality	-0.50*	0.00	-0.00	0.00	-0.00	-0.50*	0.38*	-0.25	-0.20	-0.17

* Implies correlation greater than two times its standard error (estimated).

Panel B: ARIMA Structure

Variable	ARIMA Structure	C	ϕ_1	ϕ_2	θ_1	θ_2	σ^2	χ^2+
Fertility	212	-1.83 (-97.8)	-0.99 (-99.7)	-1.75 (-38.01)	-0.99 (-26.3)	-0.25 (-0.29)	2.514	6.7
Infant	111	-1.039 (-3.434)	0.342 (2.238)	-	0.963 (39.94)	-	79.13	9.3

+ The null hypothesis (no serial correlation) is accepted at $\alpha = 0.05$.

Table A.4.3.2: Estimated ARIMA Structure for Mauritius: 1932 - 1975

Panel A: Autocorrelation and Partial Autocorrelation; Lags 1 - 5 Years

	Autocorrelation					Partial Autocorrelation				
	1	2	3	4	5	1	2	3	4	5
Fertility	-0.21	0.01	0.33*	-0.33*	0.21	-0.21	-0.04	0.34*	-0.22	0.12
Infant Mortality	-0.43*	-0.18	0.43*	-0.30	-0.00	-0.43*	-0.45*	0.19	-0.11	-0.06

*Implies correlation greater than two times its standard error (estimated).

Panel B: ARIMA Structure

Variable	ARIMA Structure	C	ϕ_1	ϕ_2	θ_1	θ_2	σ^2	χ^2+	ARSUP	MASUP
Fertility	012	4.107	-	-	-0.44	0.38	10.68	6.1	2	2
Infant Mortality	011	-2.29	-	-	0.58	-	426.1	10.8	1	

+ The null hypothesis is accepted at $\alpha = 0.05$.

Table 4.3.3: Arima Structure for India: 1932 - 1976.

Panel A: Autocorrelations and Partial Autocorrelations; Lags 1 - 5 Years

Variable	Autocorrelation					Partial Autocorrelation				
	1	2	3	4	5	1	2	3	4	5
Fertility	-0.48*	0.02	0.00	-0.34	0.61*	-0.48*	-0.26	-0.15	-0.58*	0.20
Infant Mortality	-0.21	-0.01	0.14	0.05	0.12	-0.21	-0.05	-0.13	0.12	0.18

*Implies correlation greater than two times its standard error (estimated)

Panel B: Arima Structures

Variable	ARIMA	C	ϕ_1	ϕ_2	σ_1	σ_2	υ^2	χ^2+	ARSUP	ARSUP
Fertility	(212)	5.666	-0.32 (-2.426)*	0.48 (3.71)*	1.277 (294.8)*	-0.284 (-7.352)*	47.02	6.9	2	2
Infant Mortality	(010)	(0.544)								

+The null hypothesis is accepted at $\alpha = 0.05$.

Table A.4.3.4: Estimated ARIMA Structures for Srilanka; 1932 - 1973

Panel A: Autocorrelations and Partial Autocorrelations; Lags 1 - 5 Years

Variable	Autocorrelation					Partial Autocorrelation				
	1	2	3	4	5	1	2	3	4	5
Fertility	-0.27	-0.15	-0.04	0.22	-0.09	-0.27	-0.24	-0.18	0.13	-0.01
Infant Mortality	-0.31	-0.17	-0.03	0.06	0.03	-0.31	-0.29	-0.22	-0.1	-0.04

*Implies correlation greater than two times its standard error (estimated).

Panel B: ARIMA Structures

Variable	ARIMA	C	ϕ_1	ϕ_2	θ_1	θ_2	θ^2	χ^2
Fertility	(212)	-0.179 (-0.788)	0.544 (9.472)*	-0.917 (16.53)*	0.938 (14.36)*	-0.931 (-23.55)*	1.877	9.3
Infant Mortality	(010)	-3.11 (-0.66)					79.33	6.5

Table A.4.3.5: Estimated ARIMA Structures for Puerto Rico; 1932 - 1978

Panel A: Autocorrelations and Partial Autocorrelations; Lags 1 - 5 Years

Variable	Autocorrelation					Partial Autocorrelation				
	1	2	3	4	5	1	2	3	4	5
Fertility	0.00	-0.03*	0.05	-0.07	0.05	0.00	-0.03	0.05	-0.07	0.05
Infant Mortality	-0.13	-0.36*	-0.13	0.39*	-0.02	-0.13	-0.39*	-0.30*	0.20	-0.05

*Implies correlation greater than two times its standard error (estimated).

Panel B: ARIMA Structures

Variable	ARIMA Structure	C	ϕ_1	ϕ_2	θ_1	θ_2	σ^2	χ^2	ARSUP	MASUP
Fertility	010	-0.397 (-2.49)*	-	-	-	-	1.146	2.5+	-	-
Infant Mortality	111	-4.67 (-3.44)*	-0.994 (-121.8)	-0.653 (-6.66)*	-	-	45.57	8.0+	1	2

+The null hypothesis is accepted at $\alpha = 0.05$.

Table A.4.3.6: Estimated ARIMA Structures for Jamaica; 1932 - 1978

Panel A: Autocorrelations and Partial Autocorrelations; Lags 1 - 5 Years

Variable	Autocorrelations					Partial Autocorrelations				
	1	2	3	4	5	1	2	3	4	5
Infant Mortality	-0.35*	-0.21	0.17	-0.01	-0.07	-0.35*	0.30	-0.08	-0.06	-0.06
Fertility	-0.13	0.03	0.24	-0.09	0.19	-0.13	0.01	0.25	-0.03	0.17

*Implies correlation greater than two times its standard error (estimated)

Panel B: ARIMA Structures

Variable	ARIMA Structure	C	ϕ_1	ϕ_2	θ_1	θ_2	σ^2	χ^2+
Fertility	010	-0.065 (-0.3169)	-	-	-	-	0.19	15.3+
Infant Mortality	111	-2.534 (-3.317)*	0.098 (0.389)	-	0.65 (3.356)*	-	37.3	4.9

+ The null hypothesis is accepted at $\alpha = 0.05$.

Table A.4.3.7: Estimated ARIMA Structure for Venezuela: 1932 - 1976.

Panel A: Autocorrelations and Partial Autocorrelations; Lags 1 - 5 Years

Variable	Autocorrelation					Partial Autocorrelation				
	1	2	3	4	5	1	2	3	4	5
Fertility	-0.15	0.22	0.33	0.15	0.01	-0.15	0.21	0.09	0.12	0.03
Infant Mortality	-0.27	0.08	-0.02	-0.07	0.12	-0.27	0.01	0.00	-0.08	0.09

Panel B: ARIMA Structures

Variable	ARIMA Structure	C	ϕ_1	ϕ_2	θ_1	θ_2	σ^2	χ^2+
Fertility	010	0.18 (0.768)	-	-	-	-	2.463	6.1
Infant Mortality	010	-2.729 (-3.141)*	-	-	-	-	33.23	13.9

+ The null hypothesis is accepted at $\alpha = 0.05$.

Table A.4.3.8: Estimated ARIMA Structures for Columbia; 1932 - 1978.

Panel A: Autocorrelations and Partial Autocorrelations; Lags 1 - 5 Years

Variable	Autocorrelation					Partial Autocorrelation				
	1	2	3	4	5	1	2	3	4	5
Fertility	-0.43*	0.06	-0.00	-0.22	0.54*	-0.43*	-0.16	-0.05	-0.3	0.42*
Infant Mortality	-0.41*	0.13	0.01	0.02	-0.04	-0.41	-0.05*	0.06	0.07	-0.02

*Implies correlation greater than two times its standard error (estimated).

Panel B: ARIMA Structures

Variable	ARIMA Structure	C	ϕ_1	ϕ_1	θ_1	θ_2	σ^2	AR SUP	MA SUP
Fertility	212	1.321 (0.055)	-6.42 (-0.312)	0.497 (2.458)*	0.498 (2.495)*	-0.17 (-0.821)	6.271	1.2	3
Infant Mortality	011	-1.68 (-1.548)	-	-	0.439 (3.539)*	-	16.03	3.5	

+ The null hypothesis is accepted at $\alpha = 0.05$.

Table A.4.3.9: Tables of Coefficients for Egypt: Sims' Test

Independent Variable	Estimated Coefficient	Standard Error	T-Statistic
Lag IM_3	-0.023	0.024	-0.992
Lag IM_4	-0.017	0.024	-0.708
Lag IM_5	0.024	0.025	0.972
Lag IM_0	-0.112	0.021	-5.206*
Led IM_1	-0.023	0.021	-1.096
Led IM_2	0.004	0.022	0.196
Led IM_3	0.012	0.022	0.57
Led IM_4	0.021	0.021	1.012
Led IM_5	-0.026	0.023	-1.143

Table A.4.3.10: Tables of Coefficients for Egypt: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
Lag BR_3	0.140	0.812	0.173
Lag BR_4	0.922	0.810	1.138
Lag BR_0	-4.432	0.797	-5.557
Led BR_1	0.079	0.801	0.098
Led BR_2	-0.642	0.806	-0.796
Led BR_3	-0.272	0.790	-0.345
Led BR_4	-0.072	0.794	-2.610

Table A.4.3.11: Tables of Coefficients Mauritius: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistics
Lag IM_3	0.029	0.023	1.276
Lag IM_4	0.020	0.022	0.917
Lag IM_0	-0.015	0.025	-0.609
Led IM_1	0.017	0.024	0.733
Led IM_2	-0.026	0.022	-0.186
Led IM_3	-0.042	0.024	-1.779
Led IM_4	0.024	0.025	0.973

Table A.4.3.12: Tables of Coefficients Mauritius: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistics
$LAGBR_3$	-3.327	1.190	-2.795
$LAGBR_4$	2.708	1.102	2.457
$LAGBR_0$	-2.442	1.127	-2.167
$LEDBR_1$	-1.578	1.159	-1.361
$LEDBR_2$	1.290	1.172	1.100
$LEDBR_3$	0.652	1.138	0.573
$LEDBR_4$	2.759	1.199	2.300

Table A.4.3.13 Tables of Coefficients for India: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGIM_4$	0.075	0.184	0.047
$LAGIM_5$	0.205	0.182	1.125
$LAGIM_0$	0.045	0.145	0.309
$LEDIM_1$	0.200	0.147	1.360
$LEDIM_2$	-0.162	0.144	-1.121
$LEDIM_3$	0.029	0.144	-1.121
$LEDIM_4$	-0.002	0.136	-0.013
$LEDIM_5$	0.099	0.135	0.739

Table A.4.3.14: Tables of Coefficients for India: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGBR_3$	0.063	0.307	0.205
$LAGBR_4$	-0.057	0.308	-0.185
$LAGBR_0$	0.227	0.277	0.821
$LEDBR_1$	0.437	0.277	1.576
$LEDBR_2$	0.668	0.287	2.324
$LEDBR_3$	0.356	0.271	1.311
$LEDBR_4$	0.116	0.270	0.431

Table A.4.3.15: Tables of Coefficients for Srilanka: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGIM_4$	0.009	0.008	1.049
$LAGIM_5$	0.008	0.010	0.849
$LAGIM_0$	-0.012	0.016	-0.754
$LEDIM_1$	-0.003	0.018	-0.198
$LEDIM_2$	0.003	0.018	0.146
$LEDIM_3$	0.020	0.018	1.129
$LEDIM_4$	-0.004	0.018	-0.223
$LEDIM_5$	0.022	0.016	1.356

Table A.4.3.16: Tables of Coefficients for Srilanka: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistics
$LAGBR_2$	0.763	4.232	0.180
$LAGBR_3$	0.975	4.119	1.450
$LAGBR_0$	-3.732	3.937	-0.948
$LEDBR_1$	-3.472	3.731	-0.931
$LEDBR_2$	5.402	3.988	1.355
$LEDBR_3$	-4.216	4.181	-1.008

Table A.4.2.17: Tables of Coefficients for Puerto Rico: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGIM_1$	-0.009	0.031	-0.319
$LAGIM_2$	0.012	0.032	0.391
$LAGIM_0$	-0.002	0.032	-0.068
$LEDIM_1$	-0.004	0.032	-0.114
$LEDIM_2$	-0.064	0.034	-2.134
$LEDIM_3$	-0.076	0.034	-2.206
$LEDIM_4$	-0.016	0.034	-0.487
$LEDIM_5$	-0.045	0.034	-1.333

Table A.4.3.18: Tables of Coefficients for Puerto Rico: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGBR_2$	-1.392	1.123	-1.228
$LAGBR_3$	-2.029	1.228	-1.652
$LAGBR_4$	1.037	1.04	0.997
$LAGBR_0$	0.789	1.095	0.721
$LEDBR_1$	-1.082	1.165	-0.928
$LEDBR_2$	-0.823	1.092	-0.753
$LEDBR_3$	0.803	1.135	0.707
$LEDBR_4$	-0.365	1.262	-0.289
$LEDBR_5$	-1.632	1.171	-1.393

Table A.4.3.19: Tables of Coefficients for Jamaica: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGIM_1$	-0.128	0.064	-1.486
$LAGIM_2$	-0.084	0.066	-1.271
$LAGIM_3$	0.036	0.066	0.651
$LAGIM_0$	0.083	0.067	1.247
$LEDIM_1$	-0.069	0.068	-1.025
$LEDIM_2$	-0.018	0.047	-0.395
$LEDIM_3$	0.080	0.044	1.814
$LEDIM_4$	-0.034	0.045	-0.759
$LEDIM_5$	0.039	0.045	0.874

Table A.4.3.20: Tables of Coefficients for Jamaica: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGBR_1$	-0.554	0.511	-1.084
$LAGBR_2$	0.021	0.532	0.039
$LAGBR_3$	0.287	0.527	0.545
$LAGBR_0$	-0.088	0.516	-0.171
$LEDBR_1$	-1.122	0.518	-2.167
$LEDBR_2$	-0.462	0.489	-0.944
$LEDBR_3$	0.170	0.491	0.347
$LEDBR_4$	0.529	0.480	1.103
$LEDBR_5$	0.547	0.478	1.146

Table A.4.3.21: Tables of Coefficients for Columbia: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGIM_3$	0.009	0.057	0.173
$LAGIM_4$	-0.034	0.051	-0.669
$LAGIM_0$	0.052	0.073	0.718
$LEDIM_1$	-0.113	0.074	-1.537
$LEDIM_2$	0.009	0.050	0.197
$LEDIM_3$	0.067	0.05	1.368
$LEDIM_1$	-0.044	0.050	-0.894
$LEDIM_5$	0.030	0.050	0.632

Table A.4.3.22: Tables of Coefficients for Columbia: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGBR_4$	0.420	0.475	0.884
$LAGBR_5$	0.878	0.476	1.846
$LAGBR_0$	0.021	0.467	0.044
$LEDBR_1$	-1.072	0.467	-2.278
$LEDBR_2$	-0.496	0.470	-1.092
$LEDBR_3$	0.271	0.455	0.597
$LEDBR_4$	0.569	0.440	1.293
$LEDBR_5$	0.518	0.437	1.185

Table A.4.3.23: Tables of Coefficients for Venezuela: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGIM_2$	0.038	0.054	0.712
$LAGIM_3$	-0.046	0.047	-0.984
$LAGIM_0$	-0.082	0.052	-1.574
$LEDIM_1$	-0.063	0.054	-1.153
$LEDIM_2$	-0.042	0.057	-0.733
$LEDIM_3$	-0.013	0.058	-1.774
$LEDIM_4$	-0.049	0.056	-0.878

Table A.4.3.24: Tables of Coefficients for Venezuela: Sims' Test

Independent Variable	Estimated Coefficient	Estimated Standard Error	T-Statistic
$LAGBR_2$	0.225	0.577	0.389
$LAGBR_3$	-0.923	0.564	-1.636
$LAGBR_0$	-0.999	0.654	-1.529
$LEDBR_1$	-0.404	0.654	-0.613
$LEDBR_2$	0.391	0.643	0.609
$LEDBR_3$	-0.325	0.651	-0.499
$LEDBR_4$	-0.088	0.641	-0.137

APPENDIX B

Appendix Table B.1.1.: Estimates of Per Capita Income (In U. S. $). For Selected Years.

Countries	1960	1963	1970	1974	1975	1976
Egypt	123	147	202	263	...	...
Mauritius	202	288	225	632	629	...
India	69	84	94	136	...	...
Srilanka	134	145	164	228	236	214
Puerto Rico	718	925	1784	2169	2190	2259
Jamaica	363	412	657	1090	1265	1296
Colombia	224	241	310	477	513	...
Venezuela	859	715	943	2046	2128	2357

Source: Statistical Yearbook, 1977. The United Nations, New York. Table 194.

Note: ... means not available.

Appendix Table B.1.2.: Female Population (15-49 Years)

As A Ratio of Total Population For Selected Years.

Country	Year	Total Population (1)	Female Population (15-49 Years) (2)	Ratio
Egypt	1960	25,984,101	5,883,428	0.23
Mauritius	1952	516,556	103,818	0.20
	1962	701,016	144,770	0.21
	1972	851,334	195,164	0.23
Srilanka	1953	8,097,895	1,848,753	0.23
	1963	10,582,064	2,347,786	0.22
	1971	12,689,897	3,015,198	0.24
India	1951	356,798,700	86,773,900	0.23
	1961	438,774,729	100,240,936	0.22
	1971	547,949,800	121,385,900	0.24
Puerto Rico	1950	2,210,703	505,255	0.23
	1960	2,349,544	533,501	0.23
	1970	2,712,033	661,170	0.24
Jamaica	1953	1,476,923	390,089	0.26
	1960	1,609,814	386,784	0.24
	1970	1,813,594	366,034	0.20
Venezuela	1950	5,034,838	1,192,416	0.24
	1961	7,523,999	1,638,172	0.22
	1971	10,721,522	2,445,443	0.23
Colombia	1951	11,228,509	2,711,146	0.24
	1964	17,484,508	3,963,061	0.23
	1973	22,847,055	5,421,668	0.24

Note: Total Female Population of child bearing age and the ratio are calculated from data on population by age, sex, etc.

Source: Table 3. Demographic Yearbook; Special Issue. Historical Supplement, 1978.

Sources:

1. Demographic Yearbook, New York, 1950, the United Nations.

2. Demographic Yearbook: Special Issue; Historical Supplement, 1978, The United Nations, New York.

3. Annual Reports and Fertility Tables - The Vital Statistics Division, Office of the Registrar General.

4. International Population Dynamics 1950 - 1979. U.S. Department of Commerce, Bureau of the Census.

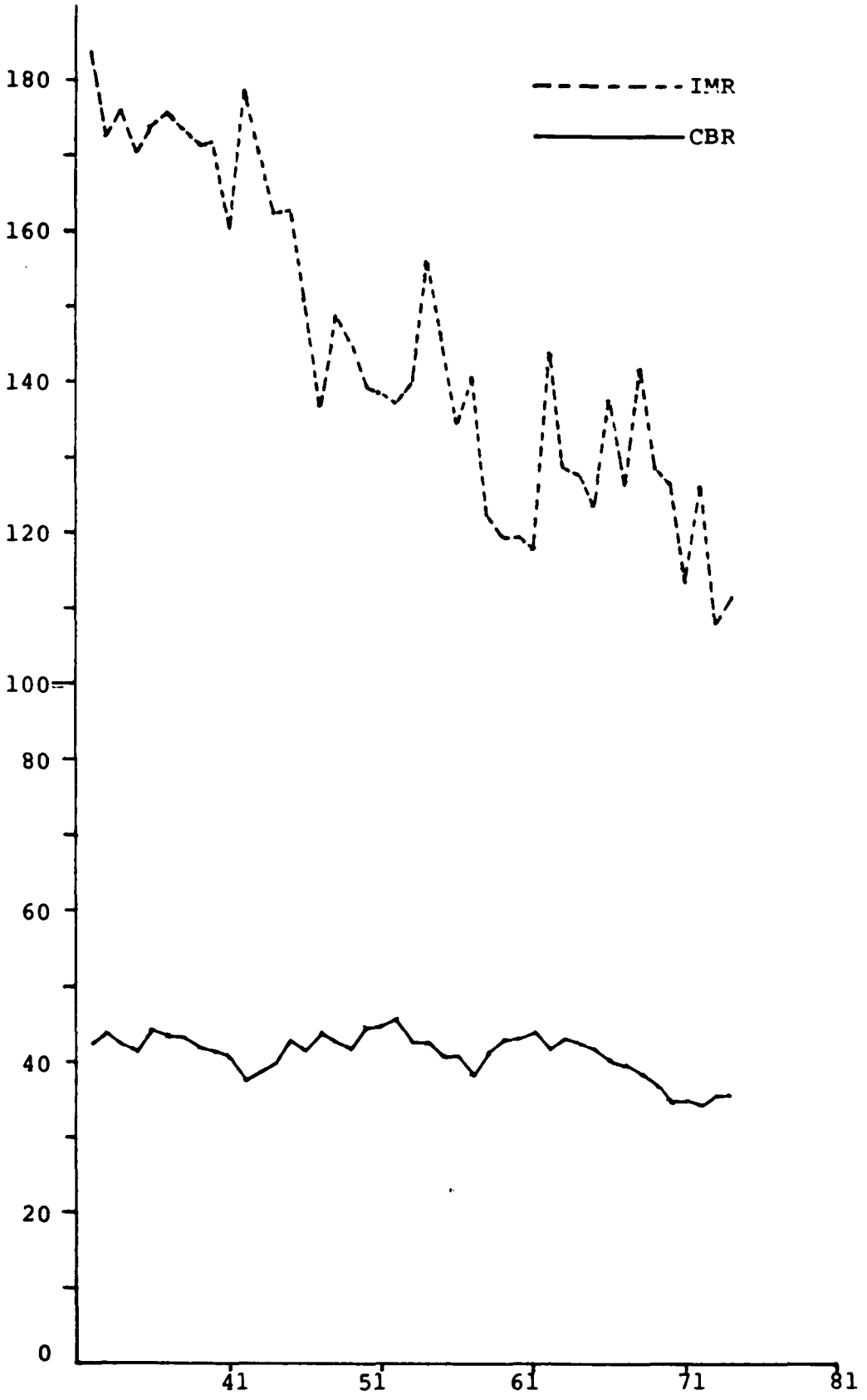

Figure B.2.1: Infant Mortality Rate and Crude Birth Rate in Egypt: 1932-1974

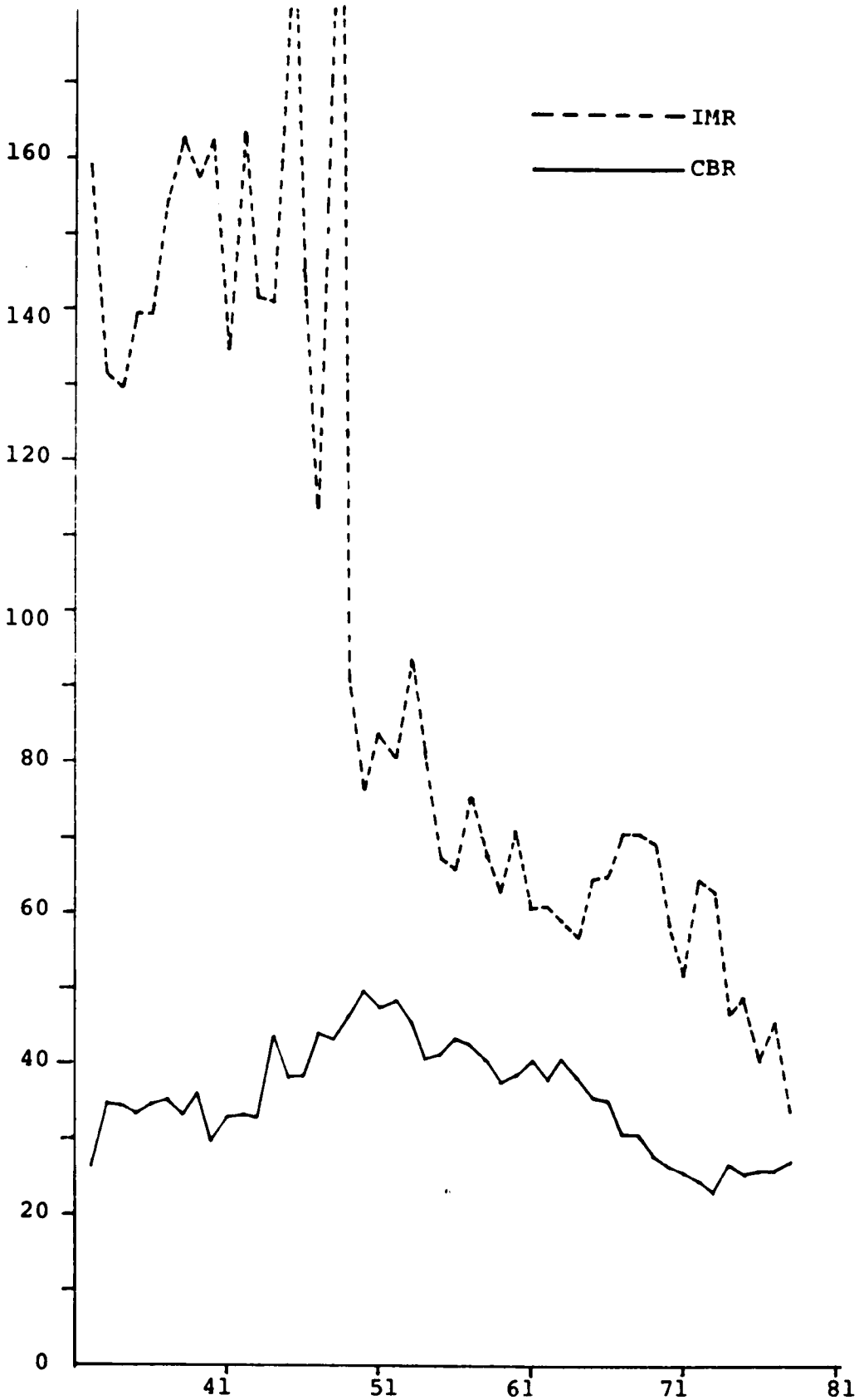

Figure B.2.2: Infant Mortality Rate and Crude Birth Rate in Mauritius: 1932-1977

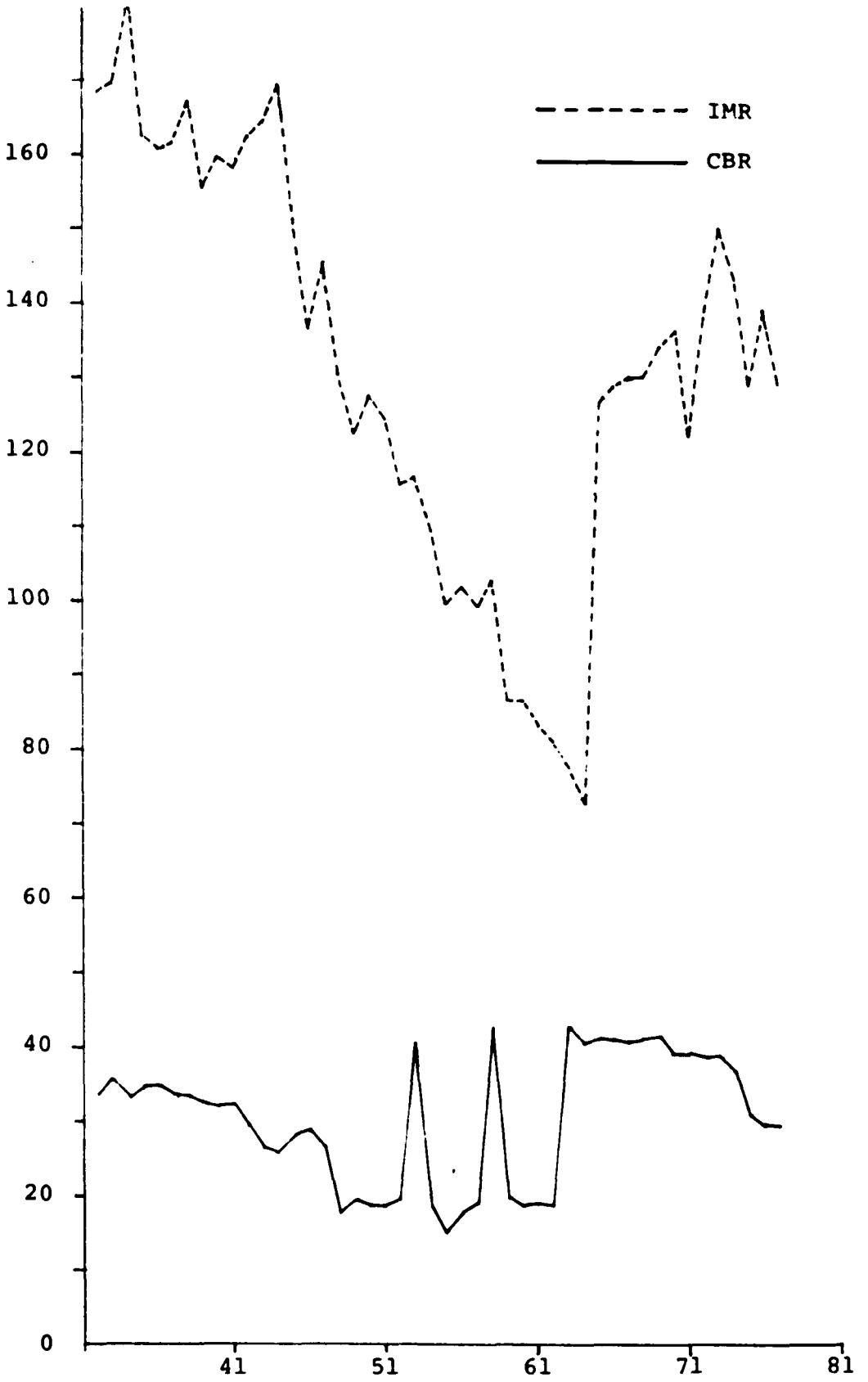

Figure B.2.3: Infant Mortality Rate and Crude Birth Rate in India: 1932-1976

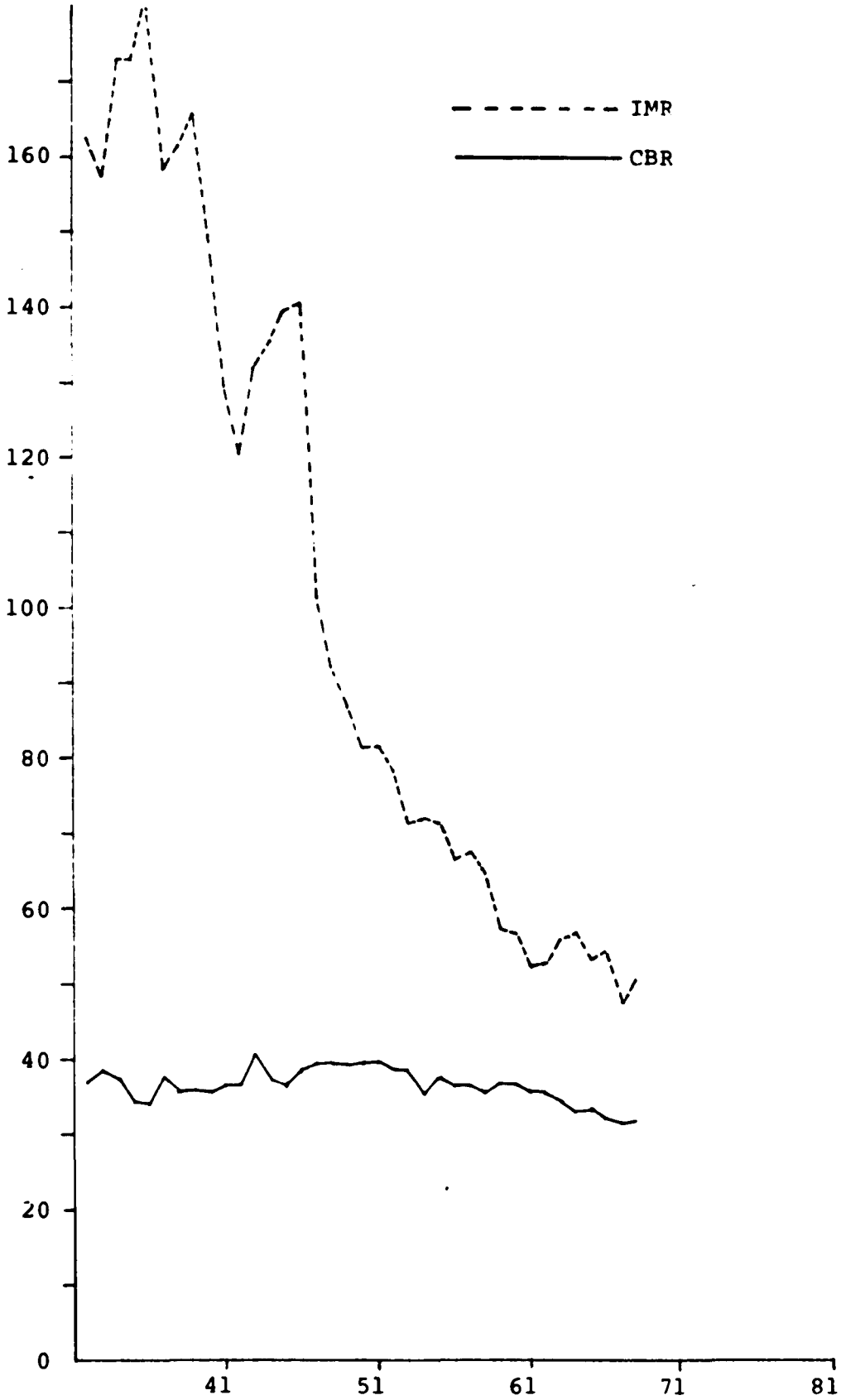

Figure B.2.4: Infant Mortality Rate and Crude Birth Rate in Srilanka: 1932-1968

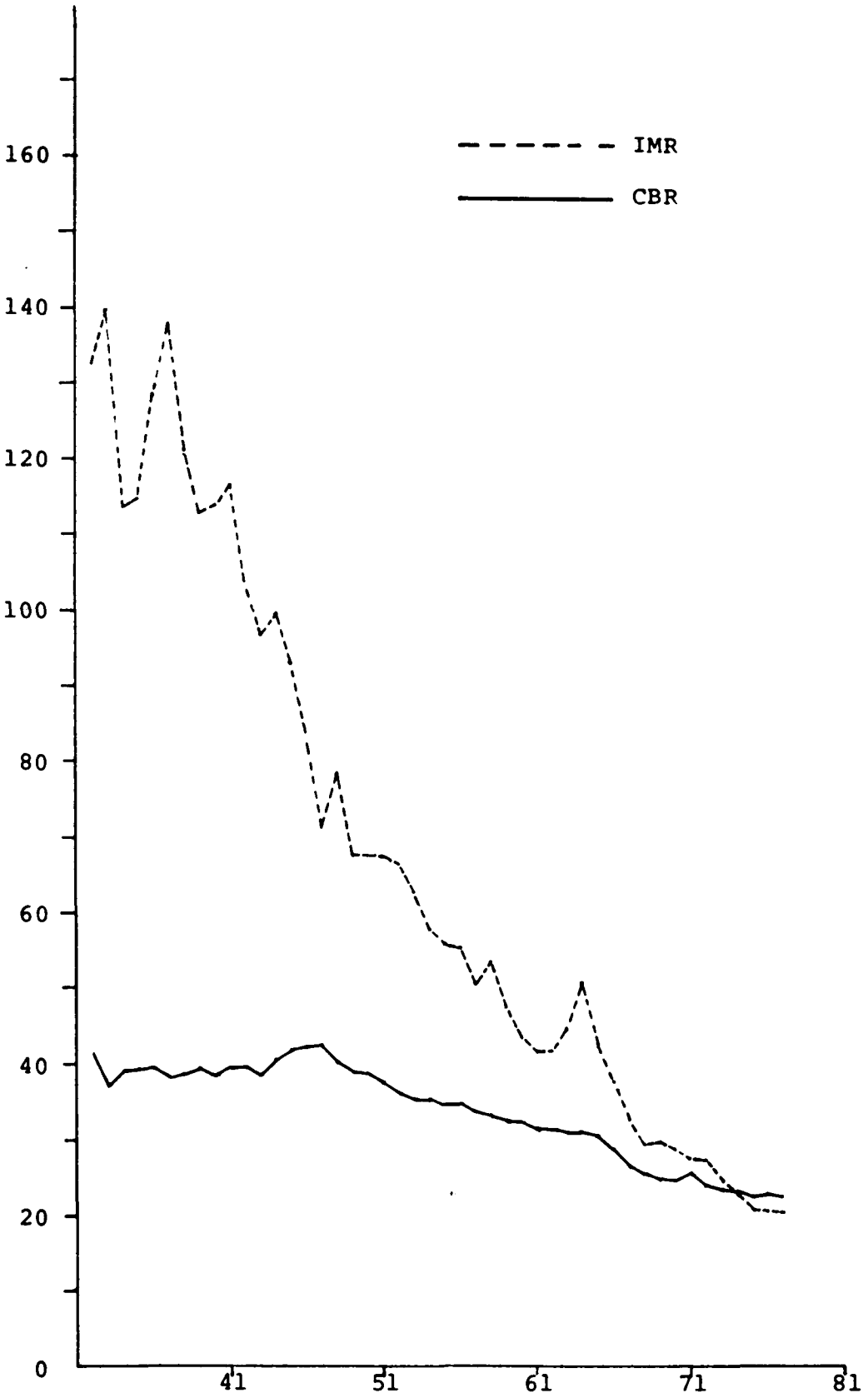

Figure B.2.5: Infant Mortality Rate and Crude Birth Rate in Puerto Rico: 1932-1977

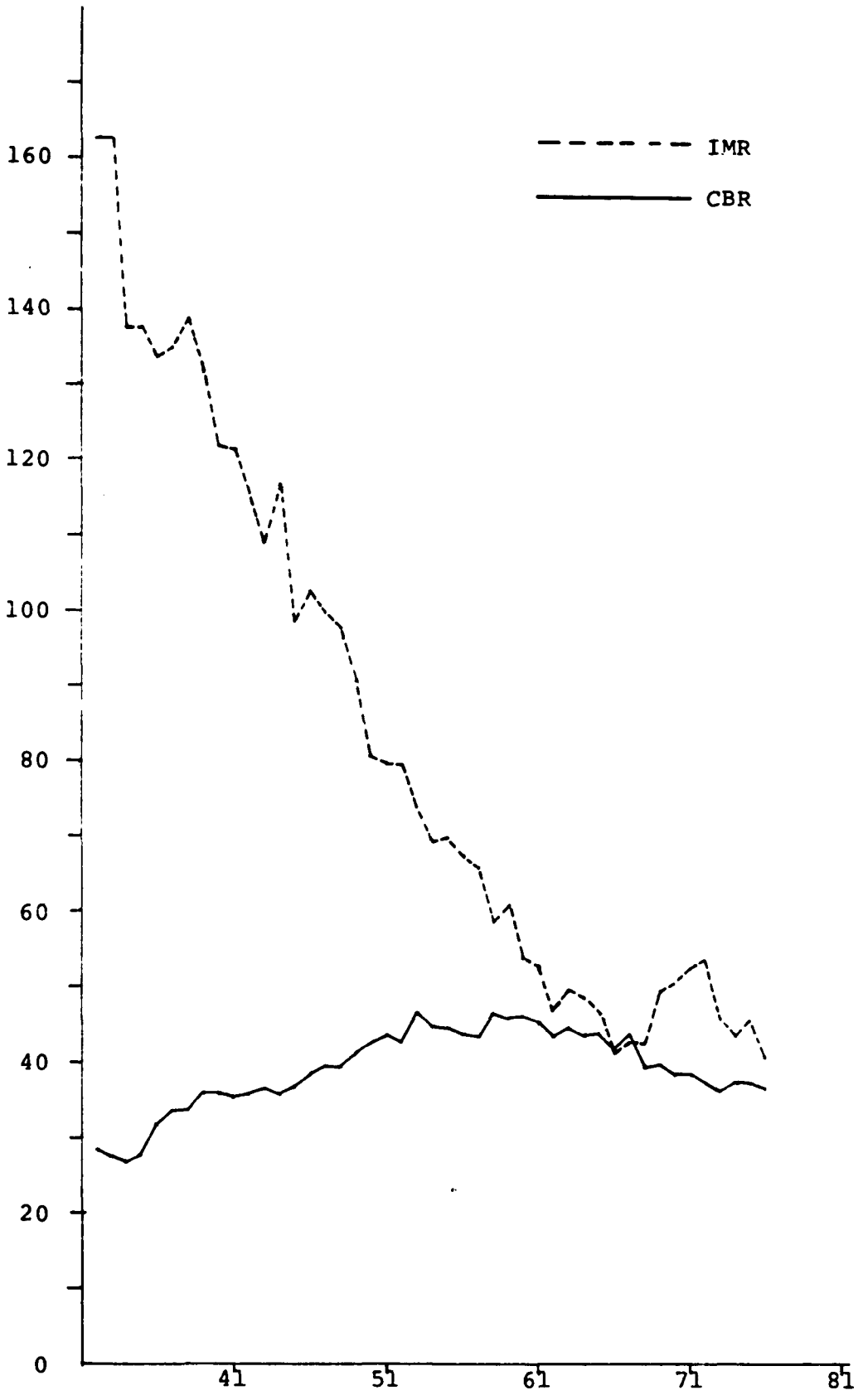

Figure B.2.6: Infant Mortality Rate and Crude Birth Rate in Jamaica : 1932-1976

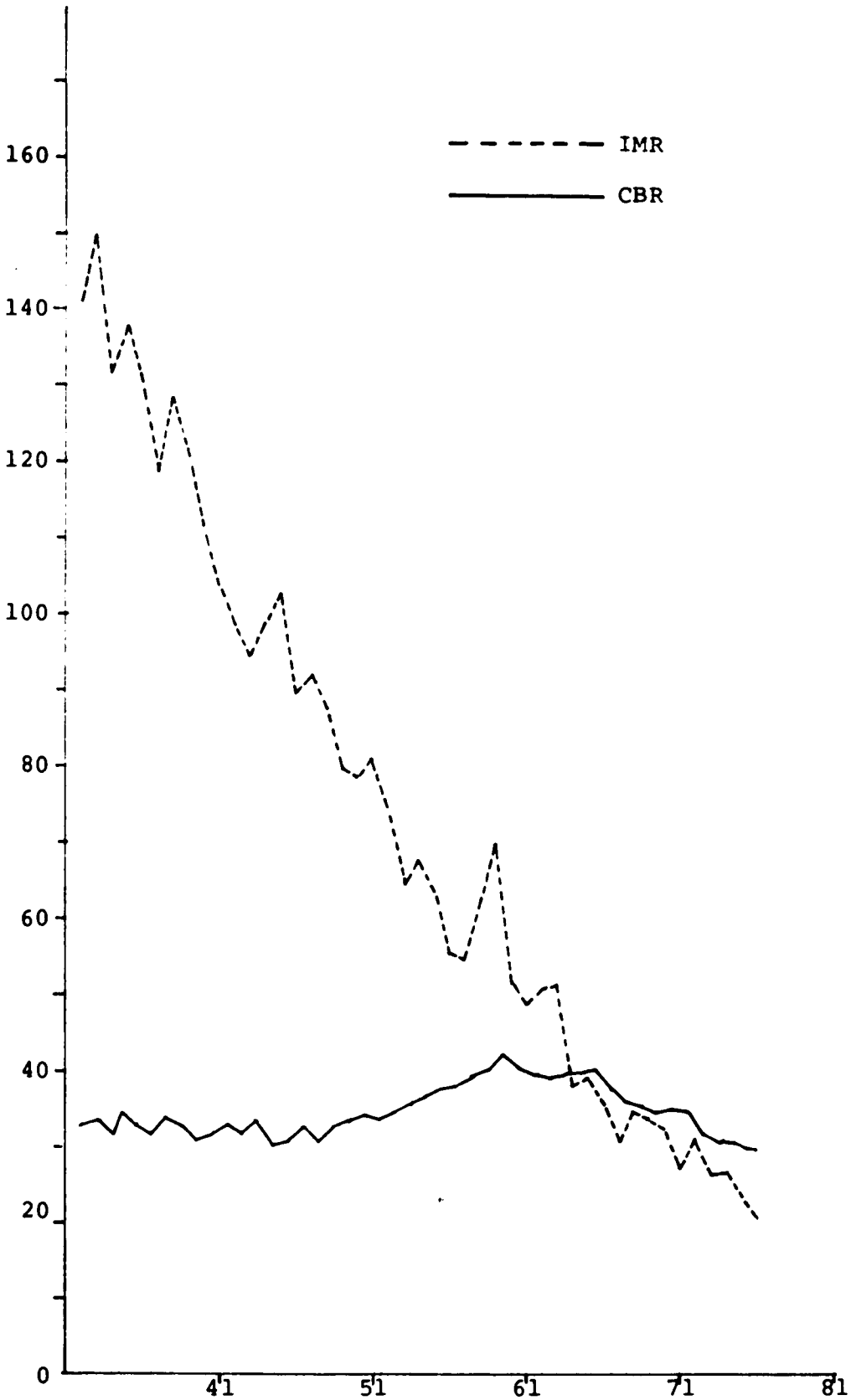

Figure B.2.7: Infant Mortality Rate and Fertility Rate in Venezuela: 1932-1976

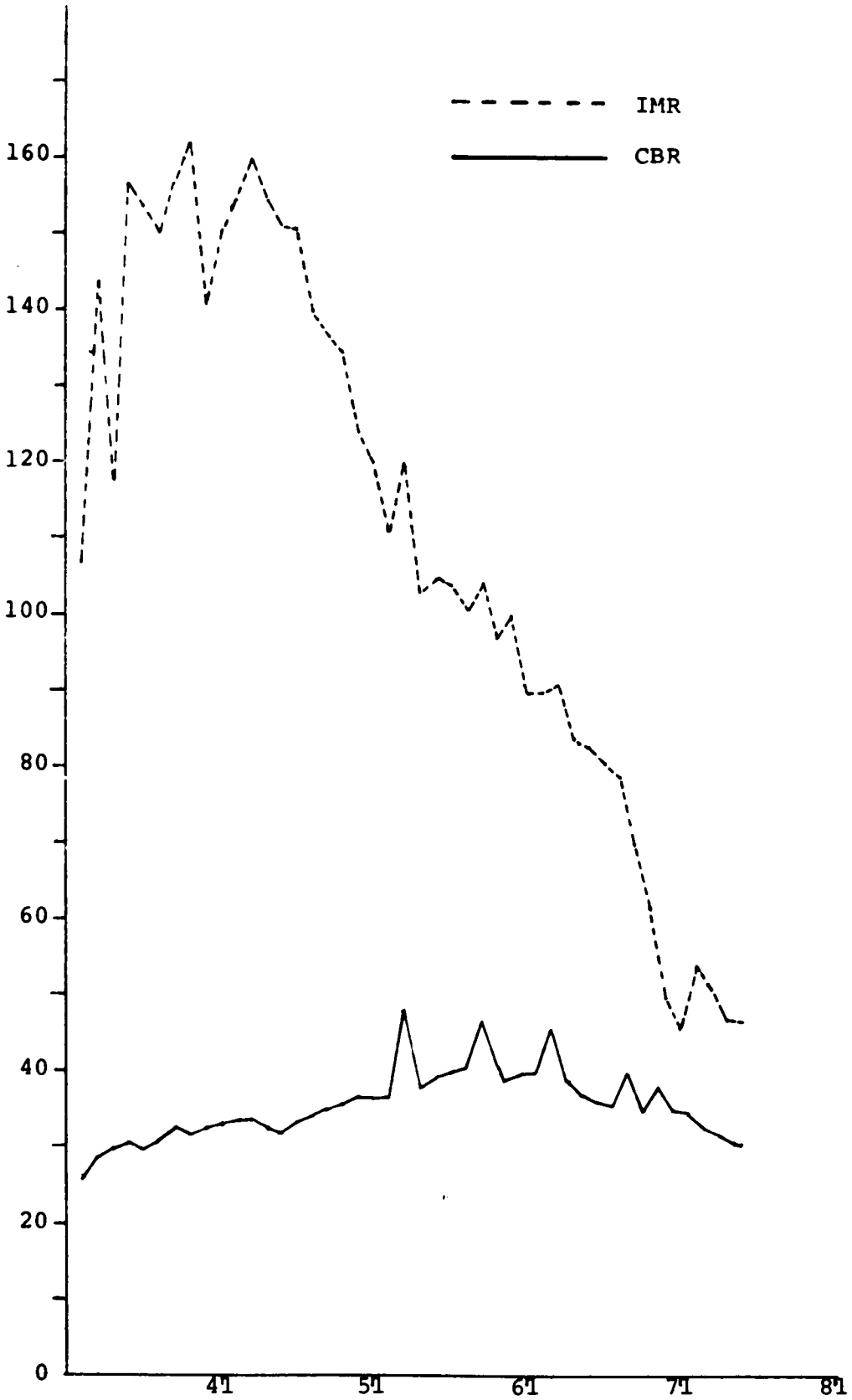

Figure B.2.8: Infant Mortality Rate and Crude Birth Rate in Columbia : 1932-1976

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