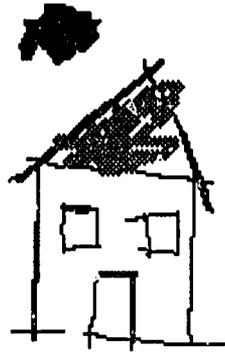


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**Seminar Report**

# Child Survival Programs: Issues for the 1990s

**The Johns Hopkins University  
Institute for International Programs**



PN-ARH-914

# Child Survival Programs: Issues for the 1990s

**REPORT OF**  
A SEMINAR HELD  
NOVEMBER 21-22, 1988 AT  
THE JOHNS HOPKINS UNIVERSITY  
SCHOOL OF HYGIENE AND PUBLIC HEALTH  
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## Contents

<b>INTRODUCTION.....</b>	<b>1</b>
<b>LEVELS, TRENDS AND PATTERNS OF CHILD MORTALITY IN THE DEVELOPING WORLD.....</b>	<b>15</b>
Kenneth Hill	
Anne Pebley	
<b>WHO USES MATERNAL AND CHILD HEALTH SERVICES? EVIDENCE FROM THE DEMOGRAPHIC AND HEALTH SURVEYS.....</b>	<b>37</b>
Shea Oscar Rutstein	
Anna Elisabeth Sommerfelt	
Juan Schoemaker	
<b>IMPACT OF THE DIRECT INTERVENTIONS.....</b>	<b>85</b>
Anne Gadamski	
Robert E. Black	
<b>INDIRECT HEALTH INTERVENTIONS WITH REFERENCE TO FAMILY PLANNING AND BREASTFEEDING.....</b>	<b>129</b>
John Hobcraft	
<b>THE INTERIM IS OVER: IMPLICATIONS OF THE CHANGING CAUSE-STRUCTURE OF MORTALITY FOR THE DESIGN OF HEALTH INTERVENTIONS.....</b>	<b>141</b>
Douglas Ewbank	
Susan Zimicki	
<b>INFANT MORTALITY RATES AND CAUSE ATTRIBUTABLE PROFILES: SOME IMPLICATIONS FOR PRIMARY HEALTH CARE DESIGN.....</b>	<b>157</b>
Norbert Hirschhorn	
Mark Grabowsky	
Robin Houston	
Robert Steinglass	
<b>DEMOGRAPHIC MODELS FOR CHILD SURVIVAL: IMPLICATIONS FOR PROGRAM STRATEGY.....</b>	<b>169</b>
Henry Mosley	
Stan Becker	
<b>A SURVEY ON SOCIOECONOMIC DEVELOPMENT: STRUCTURAL ADJUSTMENT AND CHILD HEALTH AND MORTALITY IN DEVELOPING COUNTRIES.....</b>	<b>189</b>
Jere R. Behrman	

### Workshop Agenda

### Workshop Participants

# Introduction

The goal of "health for all by the year 2000" was first enunciated at Alma Ata in 1978 (World Health Organization/UNICEF, 1978). The half way point between the establishment of the goal and the target date for its achievement was thus reached in 1988 and prompted this workshop and report. International involvement in attempts to achieve this goal have focused largely on promoting the health of infants and young children and of their mothers. This focus can be justified for a number of reasons. First, mortality rates are high in early childhood in most parts of the developing world, but are much lower for the next forty or fifty years of life. Thus children whose deaths may be averted in childhood can look forward to a lengthy period of life exposed to low risks of dying. Second, the vulnerability of children, and their inability to take responsibility for their own health, justifies society in intervening on their behalf. Third, a healthy childhood may be positively associated with greater potential productivity in adulthood, through improvements in both physical and mental status and improved receptivity to education. Large amounts of international assistance, both bilateral and multilateral, have flowed into health interventions, close to one billion dollars a year in recent years, and the majority of this aid has been directed to child survival or child health programs.

An innovative feature of these programs in the 1980s has been an emphasis on primary health care, and in particular on the widespread application of rather simple and inexpensive interventions against a small number of diseases or conditions responsible for a large proportion of child morbidity and mortality in the developing world. The most important components of these programs have been the Expanded Program of Immunization (EPI) against the common diseases of childhood and the use of oral rehydration therapy (ORT) to reduce fatalities from dehydration consequent upon watery diarrhea. Other components include nutritional interventions, including encouraging breastfeeding and growth monitoring, control of respiratory infections, and the treatment of malaria. Examples of such programs are the Centers for Disease Control's CCCD program (Combatting Communicable Childhood Diseases) and UNICEF's GOBI strategy (Growth Monitoring, Oral Rehydration, Breastfeeding and Immunizations).

These programs have undoubtedly increased the use of health interventions. The proportion of children covered by immunization has increased greatly over the last decade, as has the use of oral rehydration therapy. At the same time, mortality risks for children have declined in most developing countries. However, in some parts of the world the pace of child mortality decline is slow, and overall levels remain high. Given current rates of decline, the Alma Ata target will not be achieved.

Given this background, the Institute for International Programs of the Johns Hopkins University School of Hygiene and Public Health organized a two-day seminar to review experience gained from the implementation of child survival programs in the 1980s, and to consider the implications of this experience for programs in the 1990s. The seminar brought together experts from a range of backgrounds, from physicians to anthropologists, and with a range of perspectives on child survival interventions, from demographic evaluation to clinical implementation. The objectives of the seminar were to review the progress made towards achieving the Alma Ata target, to identify the major

obstacles to further progress, and to consider how programs could be adapted to circumvent such obstacles.

The seminar was organized into four substantive sessions, each with presented papers and discussants, and a final wrap-up session of open discussion intended to arrive at recommendations about the course of child survival programs for the 1990s.

### Session 1: The Status Quo

The first session considered the status quo of child health in developing countries. The first paper, by Hill and Pebley, examined child mortality trends in less developed countries from 1960 to the present. They conclude that overall, child mortality has declined rather fast over the period, with some slight increase in the pace of decline from the 1960s to the 1970s and early 1980s. However, the pace of decline has been uneven across world regions. Latin America and Asia have experienced very rapid declines, whereas sub-Saharan Africa and parts of south Asia have experienced much more gradual declines. The authors find little evidence that economic adjustment programs in the 1980s have had markedly adverse effects upon child mortality, at least up to the time of the most recent observations, or that child survival programs have had the effect merely of postponing deaths from one age of early childhood to another. Thus the child mortality record is encouraging, in the sense of suggesting that real gains have been made, but also prescriptive, in the sense of suggesting that a program focus on Africa and south Asia is needed.

The second paper, by Rutstein, Sommerfelt and Schoemaker, presents recent information from the Demographic and Health Surveys concerning the use of health services. Data from seven countries, two from Latin America, three from sub-Saharan Africa and two from Asia, were examined. Large variations in use were found from country to country, but these were by no means always in the directions expected on the basis of mortality differentials. Within countries, the use of health services was strongly associated with the educational level of the mother and with urban versus rural residence (both on a univariate and multivariate basis), but showed no clear, consistent association with marital status, age or parity of the mother. As the authors point out, their measures of service use reflect the combined effects of supply and demand; a low use figure could represent either no supply or no demand, whereas a high figure represents both ready availability and high demand. The survey data suggest a substantial increase in proportions immunized among the cohort aged 12 to 23 months relative to those among the cohort aged 48 to 59 months, consistent with a recent increase in the level of immunization.

In the discussion that followed, a number of questions were raised about the measures used in both papers. With regard to the Hill and Pebley paper, the discussant, Birgitta Bucht, underlined the importance of census and survey data, particularly World Fertility Survey and Demographic and Health Surveys, in improving our knowledge of child mortality in the developing world, but pointed out that there are still large gaps in our knowledge, and data sets that have to be discarded because of low quality; in general, however, the work of the United Nations Population Division has led to similar conclusions to those expressed by Hill and Pebley. Other comments included the observation that declines in child mortality would appear to be slowing, and differences

between regions would be less pronounced, if absolute declines in mortality were used in place of the relative declines adopted by Hill and Pebley; and that observations for recent periods are selected by data availability, and may not be representative of all countries. With the Rutstein et al. paper, the discussant, Carl Kendall, stressed the need for multi-instrument approaches to data collection in the area of service use, including community variables and intervention program data. Other commentators pointed out that indices of use of services based on possession of health cards are hard to interpret because of possible effects of loss of cards; that relationships between socio-economic factors and the use of services should be examined separately for preventive, curative, and birth spacing services; and that an attempt should also be made to distinguish between non-use because of lack of access and non-use despite access, though data on community characteristics may be only partial indicators of access.

In terms of implications, it was suggested that persistently high mortality in sub-Saharan Africa may result from endemic malaria and very poor nutrition, in which case intervention programs broader than the standard GOBI recipe will be required. Outliers, where mortality declines have been unusually rapid or slow, may prove a fruitful subject for investigating the importance of a variety of factors in mortality decline. Similarly, the identification of population sub-groups with low levels of service use helps to identify target groups for program interventions. Aggregate level analysis does not, however, provide a basis for drawing conclusions about the impact of investments in child survival programs, for which purposes longitudinal or case-control studies are needed.

## Session 2: Child Survival Interventions

The topic of the second session was child survival interventions. The first paper, by Gadomski and Black, reviewed performance and potential for direct interventions. For a number of interventions -- against diarrheal disease, acute respiratory infection, immunizable diseases, malaria, and malnutrition -- the paper summarizes the rationale, implementation methods, outcomes, implementation problems, and future prospects for the intervention. In general, the review is positive; intervention coverage is increasing in the developing world, and both circumstantial data at the population level and results from smaller-scale longitudinal studies are accumulating to support the view that the interventions are working to reduce child mortality. The main problems seen include the effects of lack of either socio-cultural acceptability or access on utilization, the need to maintain both enthusiasm among health workers and physical infrastructure, and questionable long-term sustainability. The exception to this optimistic review was malaria, with increasing resistance to both curative treatments and vector control threatening a major up-swing for this disease. With regard to future prospects, the paper stresses the potential for vaccine development against diseases for which there is currently no immunization widely available (diarrheas, respiratory infections, malaria) and the scope for more effective implementation of existing interventions.

The discussant, Stan Foster, described the data concerning intervention effectiveness as ranging from preliminary through intermediate to solid. Something could be learned about program strategies by looking at the characteristics of failures. A primary health care intervention process progresses from access through quality of delivery, coverage, effectiveness, reduction in morbidity and mortality from the target

disease to increased overall survival. Failures can occur at each of these stages. It is also useful to look at the characteristics of successful programs; such programs are generally decentralized, use local data for targeting, monitoring and assessing the program, and maintain an emphasis on training at all levels. These programs can be described as negotiated primary health care, with the community and government collaborating to introduce and publicize feasible, effective and affordable interventions.

The second paper by Hobcraft reviewed the potential for indirect interventions to reduce child mortality by modifying the stream of births exposed to the risk of dying. Interventions that reduce fertility can have an immediate macro-level effect on reducing absolute numbers of child deaths, and will also, at least in the short run, increase the per capita availability of resources above the level that would have been reached in the absence of a decline. Fertility change can also have important micro-level effects, through later childbearing, better child spacing, increased household resources per child, and reduced risks of maternal mortality. The evidence is that the impact of better spacing on child mortality could be substantial, with the potential to reduce the probability of dying by age 5,  $5q_0$ , by up to 30 percent in populations where a substantial proportion of birth intervals are shorter than two years and childbearing starts early. Such potential exists primarily in areas such as Latin America and the Middle East where child mortality has already declined but birth timing practices are unfavorable to child survival; in Africa and South Asia, birth intervals are already long, and the potential gains are small, though programs should emphasize the benefits of maintaining traditional spacing practices. Breastfeeding is also likely to have direct effects on child mortality through better nutrition and indirect effects through better birth spacing. Other indirect effects are also cited; maternal education is perhaps the best substantiated, though the pathways through which it operates are poorly understood; recent indications that child mortality is heavily clustered in particular households, even after other characteristics have been allowed for, suggest the existence of unobserved risk factors; a range of politico-economic factors, including political will, income distribution and mass mobilization, have also been suggested as important contributors to child mortality decline.

In his discussion of the paper, Potter accepted the existence of indirect effects on child mortality, but cautioned against drawing detailed conclusions about intervention strategies when the causal mechanisms are not understood. Though early childbearing does seem to involve higher risks for both mother and child, birth interval effects may work primarily through breastfeeding. From a policy point of view, the issues are how to promote more universal and longer breastfeeding, particularly at the same time as contraception is being stressed, how to delay early childbearing, and how to encourage pre- and post-natal care. Such behavioral practices are more volitional than, for example, immunization, and thus require different, and possibly more difficult, methods of implementation.

The general discussion picked up the themes of the limitations of interventions focusing on a very small number of diseases (such as the 'twin-engined' program supporting EPI and ORT) and of the crucial importance of community involvement and participation in intervention programs. A narrow focus prevents theoretically effective interventions from achieving comparable use effectiveness. Acute respiratory infections (ARI) provide a case in point; the reductions in ARI mortality expected from

immunization and improved nutrition have not materialized, and ARI is now regarded as a necessary focus of child survival interventions, though the nature of the intervention is problematic. Several speakers stressed the need for good data on which to base program priorities; there has been a tendency to target programs according to what experts feel to be important and right, rather than to rely on evidence of what works and what does not. The importance of household behavior, both in prevention and cure, is also now recognized, but little is known either about the mechanisms through which household behavior works or about how to change such behavior. Demand for services generated at the household and community level is clearly an important component in determining program impact on mortality, and is likely to vary with the level of community participation in program implementation as well as the perceived acceptability of particular interventions.

In summing up, Black stressed that there is some evidence that direct interventions have reduced disease-specific and overall child mortality, even though the evidence is incomplete, often requiring extrapolation from what is observed, such as a reduction in morbidity, to an impact on mortality. However, some loss of effectiveness is likely at each link in the process chain, resulting in smaller than expected mortality declines. Theoretically-effective interventions against ARI and malnutrition exist and are clearly needed, though the ability to implement such interventions effectively remains questionable. Finally, two important diseases, malaria and Acquired Immune Deficiency Syndrome (AIDS), have no clearly effective and implementable interventions, and remain areas of serious concern in the reduction of child mortality. Hobcraft concluded that four reproductive variables, increased use of family planning to reduce fertility and slow population growth, increased extent and duration of breastfeeding, delay of first births, and spacing of at least two years for subsequent births, represent important targets for child mortality reduction. However, further research is also needed into pathways (through which such factors as improved spacing, maternal education and autonomy affect child mortality), synergies (whereby specific interventions can have multiplier effects) and monitoring (how to identify high risk groups, or short run mortality changes, quickly and cheaply).

### Session 3: Factors Affecting Program Strategies

The third session was concerned with strategies for design and implementation of child survival programs, with particular emphasis on how the realities of declining mortality in the developing world should affect such strategies.

The first paper, by Ewbank and Zimicki, reviews how child survival programs must evolve in order to remain relevant as mortality declines. The programs will have to evolve beyond the narrow focus on vaccination and acute diarrhea to include ARI, malaria, growth monitoring, prenatal care and nutrition, particularly breastfeeding. The choice of expanded targets should be made on the basis of the cause-structure of deaths under five. This cause-structure varies widely from country to country, even between countries with similar overall levels of child mortality. Declining mortality from immunizable diseases such as measles and tetanus and from acute diarrhea has increased the relative prominence of other diseases such as ARI and malaria. In some countries, as many as 30 to 40 percent of deaths under five can be due to ARI, making it an essential

component of longer-haul intervention strategies. The case of malaria is similar, though intervention is problematic. A somewhat different example is AIDS; a five percent seropositive rate among mothers of childbearing age combined with a fifty percent congenital infection rate would imply a probability of dying by age five of 25 per thousand due to AIDS alone; such numbers underline the need for emphasis on sterile needles, safe blood supply and condoms for prevention. These changes in disease focus also imply changes in intervention mechanisms. Thus while EPI and ORT can be implemented by vertical campaigns and commercial outlets, many of the targets of the next round of interventions will require community services combining outreach and clinic activities. In the long run, campaigns must be replaced by routine service, and the next phase of primary health care should take this need into account by strengthening health sector infrastructure. In summary, there is a universal need for a comprehensive health system providing local services; the campaign strategy of the last decade should only be seen as a stopgap, and to increase its utility it should put greater emphasis on developing local infrastructure and training local staff.

Ron Gray in his discussion agreed that data on cause of death are needed both for choosing program strategies and for evaluation purposes. However, cause of death in childhood is a complex causal chain, and data on one single underlying cause may not be very informative. Data quality is also very poor; though verbal autopsy methods are improving, they are still weak in identifying ARI and different types of diarrhea. Since cause of death structures vary widely among populations with similar mortality levels, such levels cannot be used to infer cause structures. Data problems also affect measures of mortality in the perinatal period; misclassification of live births as still births reduces estimates of very early child mortality, a period in which there is much child mortality preventable through safe motherhood initiatives.

The second paper, presented by Robert Steinglass on behalf of Hirschhorn, Grabowsky and Houston, also took up the issue of cause of death structure and changing mortality level. Using cause of death data from historical populations, changes in the cause structure of child mortality were related to overall mortality levels. As mortality falls, cause structure changes, and program design should change from an initial emphasis on measles, tetanus, malaria and ARI, to an emphasis on diarrhea and thus ORT in combination with child feeding practices and effective immunization programs. Finally, as mortality reaches low levels, emphasis should also extend to pre-natal care and other services aimed at neo-natal mortality.

The discussant, Henry Mosley, started by saying that cross-national comparisons, and especially those also across time, can be misleading; what is needed now for program design is local-area data. The use of relationships between cause structures and overall mortality levels from historical populations in the now-developed world to arrive at conclusions about program targeting now in the developing world may also be misleading. For example, the increase in deaths to diarrhea in England and Wales in the early twentieth century as child mortality was falling may have been due to declining breastfeeding and increased use of unpasteurized milk, circumstances not generalizable to the developing world now. Further, the diversity of cause structures for given mortality levels, an example of which is the wide variation in measles mortality even in unimmunized populations, represents a major problem for designing and implementing

interventions. A further consideration is that cause of death structures are not the only factors that should be taken into account when designing interventions; for example, malaria eradication may have little effect on cause of death structure, but can be expected to have a large effect on child mortality.

The last paper in the session, by Mosley and Becker, is concerned with the implications of health dynamics for child survival program design. The process by which children die in developing countries is often a complex chain of successive infections increasing frailty and finally culminating in death. Concentrating program interventions on only a few links in the chain may not affect accumulated frailty very much, and thus may not reduce child mortality as much as clinical effectiveness measures would predict. In particular, interventions that operate through reduced case-fatality ratios rather than through reduced incidence of disease are likely to reduce child mortality by much less than expected because of increased frailty among survivors and thus increased susceptibility to other infections. The paper develops a model of child mortality incorporating specific frailty states along with incidence and case-fatality ratios; the model shows the relative efficiency of interventions that reduce incidence over those that reduce case-fatality. Heterogeneity in accumulated frailty is also likely to influence the effectiveness of interventions across socio-economic groups, the worst-off groups having the highest frailty levels and thus being least affected by curative interventions.

Antoine Augustine, the discussant, accepted the argument that the effectiveness of an intervention varies with the intervention's role in avoiding increases in frailty, but pointed out that intervention strategies were ultimately limited by feasibility. Thus an intervention that can be implemented is preferable to an intervention that cannot be implemented, even if it only achieves a portion of its theoretical effectiveness. Largely serendipitous benefits can also occur because of the ways in which the health system itself can influence health behavior at the family level. Thus an intervention such as oral rehydration therapy can have an impact beyond the reduction of mortality from dehydrating diarrhea, for example by alerting mothers to the dangers of poor hygienic practices.

The frailty model and its implications were also widely discussed. It was suggested that a more realistic model might include variations in frailty at birth, and also variations in disease incidence with accumulated frailty. The importance was underlined of a change in goals of child survival programs away from EPI as the goal to child survival as the goal. One participant suggested that a biomedical model of the Mosley-Becker type misses the point that intervention strategies have been developed on the basis of what can be done rather than on the basis of what needs to be done. A program emphasis on EPI does not mean that broader health services are not needed in developing countries or that EPI is an end in itself, but rather that EPI was a feasible starting point for intervention. The question now is how to develop programs that follow up on successful EPI, given that the easy part has been done. Choices include literacy campaigns, attempts to combat malaria, and interventions against respiratory infections, but there are no clear-cut cost or effectiveness reasons for making such choices.

#### Session 4: The Role of Socio-Economic Factors

The fourth session was concerned with the socio-economic context in which child survival programs operate, and the inter-relations between these socio-economic factors and child survival programs. The background paper for the session, prepared by Behrman, provides an encyclopedic review of economic frameworks for studying causal relations in child mortality and of evidence concerning such relations. It is impossible to summarize Behrman's paper in this review, but it is worth pulling out a few of the more important conclusions. Our knowledge of the inter-relationships between socio-economic factors and child mortality has improved over the last two decades, but there are still major gaps, largely due to the difficulty of studying processes with complex feedback mechanisms. Macro-level studies suggest a strong relation between economic indicators and child mortality, though also suggesting that child mortality reductions have out-paced economic gains; recent claims that structural adjustment policies in the developing world have had a strongly detrimental effect on child mortality appear to overstate their case. At the micro level, investigations of the determinants of a variety of outcome measures (child mortality, child morbidity, child health or health service use) have met with only modest success; though some studies have shown the importance of familial endowments in determining one or other of these outcomes, others have shown a relationship between nutritional supplements and outcomes, whereas most have shown only small effects of income. Maternal education appears as an important factor in both micro and macro level studies, though a recent study controlling for family background by studying the mortality experience of the children of mothers who were sisters found virtually no independent effect of education, suggesting that the education effect is largely a proxy for family background factors. Food consumption patterns are found to be strongly influenced by income and prices, but responses are not always favorable: factors such as taste appear to be more important than nutritional value in food purchasing decisions, so income or price effects may worsen overall nutrition. Some recent studies suggest that food or health input subsidies may redistribute income away from the poor; only those policies with positive externalities, such as measures to prevent infectious disease, or distributional benefits, such as minimum nutritional maintenance, may thus be justifiable. The paper also lists a number of research questions essential to further clarification of the issues involved.

As the first discussant, Jack Caldwell felt that the paper's emphasis on what we do not know, and on how hard it is to know, raised the question whether anything in the area of the behavioral, social and economic determinants of health was knowable according to the strict scientific standards laid out. He also questioned whether the household could be taken as the decision-making unit in health production, given doubts about how to define households and about the role of fathers in households, as well as evidence that the breakup of households has a large impact on child mortality, and indications from some studies about the role of factors exogenous to the household, such as family health workers in Sri Lanka, in mortality decline. Caldwell accepted that the effect of maternal education is indirect, pointing out that some studies suggest that the main role of maternal education on service use is through persistence: that if an initial treatment does not succeed, the educated mother will go back for alternative treatment whereas the uneducated mother will not go back at all. One implication of this finding is that it is not

the ability to read or write that produces the education effect, so literacy campaigns will be relatively ineffective substitutes for full female education. Maternal education also appears to have an effect through reducing barriers to service utilization.

The second discussant, Ralph van der Hoeven, was concerned that the narrow focus evident in the paper on production functions and thus on the individual level fails to take into account a broad range of socio-cultural factors. At the household level, bargaining among members for scarce resources has an important effect on resource allocation. Similarly in the examination of effects of stabilization and adjustment policies, he felt that Behrman's emphasis was too much on the micro level, without enough consideration of macro demand effects on employment and investment. Reductions of central government expenditure also raise important distribution issues: is the relative distribution of such expenditure maintained, or are certain social sectors such as health and education sheltered from budget cuts? Quality effects of budget cuts may not be easy to measure; for example, cutting education budgets may not affect enrollment rates, but may affect the quality of instruction received by each enrollee. Overall, price measures and individual responses have not adequately cushioned the effects of stabilization and adjustment policies, and there remains a need for social interventions.

In the discussion, doubts were raised whether adjustment programs had really had their intended effects of increasing output and employment, or whether their main effect had been to change income distribution, increasing inequality in the process. Various participants returned to the interpretation of family endowment, which could reflect an effect either wider or narrower than family context; the loss of significance of maternal education as a factor in child mortality among sisters may also indicate influences between sisters rather than family endowment factors.

#### Session 5: Implications and Recommendations

The final session was intended to integrate the presentations and discussions of the previous sessions in order to arrive at conclusions about the directions child survival programs should take during the next decade. Unfortunately, the discussion did not follow any clear sequence, tending instead to jump from topic to topic haphazardly. However, in order to make this report somewhat more comprehensible, an attempt will be made to present the discussion in a logical sequence.

The first issue is one of strategy. Assuming that the reduction of child mortality can be regarded as an end in itself, what can relatively small amounts of international assistance to developing countries do most effectively to contribute to a decline in child mortality? The choices range from highly-focussed interventions (such as EPI-ORT) with vertical implementation, through broader, perhaps community-based intervention strategies, to very broad social and infrastructural development policies. Criteria are needed to choose between these alternatives, but the criteria themselves may be open to question. For example, the cost-effectiveness of a particular intervention would seem to be an uncontroversial choice, but both political and equity issues may also be important. Participants in the workshop expressed opinions ranging from the view that a narrow EPI/ORT focus is best (on the grounds that it would have a large effect at modest cost, was feasible given current technologies, and avoided the danger of over-reaction to the whims of fashion) to the view that a broad community-based development strategy

stressing water supply and sanitation as well as mainstream health services through health centers is needed. On balance, a compromise position, maintaining a limited number of specific interventions but trying to coordinate their delivery into a strengthened health infrastructure, with flexibility to allow for local conditions and temporal changes, seemed least offensive.

Given a limited number of effective interventions as an acceptable strategy, the question arises of which specific interventions to include. Most participants supported a broadening of interventions beyond the twin engines of EPI and ORT, with strong support for interventions against acute respiratory infections and malaria. Given the convincing theoretical case in favor of prevention rather than cure, EPI, and particularly measles immunization, was accorded high priority. The apparent importance of malaria not so much as a cause of death but as a cause of frailty, particularly in sub-Saharan Africa, was used to argue for better prevention (revamping vector control measures, continuing work on vaccine development) and cure (using chloroquine therapy, or alternative treatments as necessary in chloroquine-resistant areas). One topic that was raised frequently, but never really considered in detail, was AIDS. AIDS may become a major component of child mortality, and may have indirect effects through consuming health resources that could have been put to other purposes. An effective intervention is clearly needed, but may have to depend on behavior modifications in the medium term. Many participants emphasized the need for interventions to be tailored to local conditions, using such information as may be available on cause of death structures, and to be responsive to changes as mortality declines.

Once a package of interventions has been established, the next issue is delivery. Delivery has two aspects, one accessibility, making sure that the chosen interventions are within reach of motivated families, and the other, motivation, making sure that families make use of services accessible to them. Accessibility is particularly problematic in Africa, sometimes for reasons of civil war or insurrection, about which nothing can be done, but also for reasons of very poor communications and lack of facilities. Both are serious problems, and expensive and time-consuming to fix. Participants urged ingenious and imaginative solutions, but did not come up with any concrete suggestions, beyond reducing restrictions on who can prescribe what to whom; if imaginative and effective solutions exist, they are likely to be program-specific. Effective access also requires well-trained staff, able to diagnose simple conditions and prescribe suitable treatments, and adequate supervision. Better ways of training health workers, and of monitoring and supervising their performance through health information systems, are needed.

The question of demand for services produced a wider range of suggestions. There are those who believe that demand is the most important variable in service use, and that a major pathway through which maternal education reduces child mortality is through service use. A number of participants argued strongly in favor of community involvement in intervention programs, from planning interventions through implementation to monitoring. Others felt that a focus on the community, even if desirable, was logistically impossible, and might not serve the intended purpose, of giving a sense of involvement in program implementation, since even at the community level decisions are made at the top. Vertical programs can serve as a first introduction of health activities in areas where conventional facility-based services are impracticable.

A number of other approaches to stimulating demand were also suggested. Health behavior can be influenced by pricing policies (subsidies), by education in a broad sense including Information, Education and Communication (IEC) activities, by demonstration of impact and by enforcement. Enforcement is normally impracticable, and formal education has an effect only slowly. Pricing policies in the form of cost recovery may discourage the adoption or continuation of service use, and may thus be undesirable in those parts of Africa affected by recession, but heavy subsidy is not enough by itself to stimulate adoption of health service use. The continuation of child mortality declines in countries where recession has had a major economic impact can be interpreted as an indication that child mortality declines are self-reinforcing, that once child mortality declines are evident to the population, demand for health services and health promoting behavior are greatly stimulated. Initially, however, demand can be stimulated by effective IEC campaigns linked to program activities. It was generally agreed that more consideration should be given to developing ways of modifying health-related behavior and increasing demand for services.

Once use has been established by ensuring adequate supply and stimulating necessary demand, there is a need to maximize effective use. For immunizations and some other interventions administered by health workers, efficacy is likely to be a function of infrastructure, for instance the existence and maintenance of a cold chain for heat-sensitive vaccines. However, many curative interventions are home based, and instructions for use must be given by providers, perhaps assisted, in such cases as ORT, by IEC messages. The general level of education, and the specific training of health workers, are both likely to contribute to the maximization of effective use.

Once widespread effective use of selected interventions has been achieved, the child mortality revolution should be well under way. At this stage, population sub-groups that do not use services need to be identified, and targeted programs instituted. The cause structure of child morbidity and mortality will have changed, so the structure of programs will also need to be gradually altered, increasing the range of diseases, and shifting from centrally-managed campaigns to the development of local health facilities.

### Summary and Recommendations

Child mortality has declined rapidly since the early 1960s in most of the developing world. Despite economic recession during the last decade, these declines seem to have continued at much the same pace as in the preceding decades. Declines have in general been faster in Latin America and South East Asia than in South Asia and Africa. Mortality after age one has declined faster than infant mortality, closely following patterns established by the present low mortality populations. Use of health services, as measured by percent of children immunized and percent of cases of diarrhea treated with ORS, varies widely between countries and varies within countries by education of mother and urban-rural residence. Service use appears to have been increasing quite rapidly over the last five years.

There is no conclusive evidence to link the decline in child mortality to child survival programs, though there is strong circumstantial evidence that such programs, or their main components of immunization or ORT, have contributed to the maintenance or acceleration of mortality decline in the face of economic stagnation. Potentially effective

interventions exist for almost all the major childhood diseases, the most important exception being AIDS. The interventions vary greatly, however, in how they can be implemented and how large the loss is between theoretical and use effectiveness. Nutrition programs, particularly vitamin A supplementation and encouraging breastfeeding, can reduce morbidity or mortality from most childhood diseases. Reductions in child mortality can also be expected from longer birth intervals, achieved either by longer breastfeeding or the use of contraception for birth spacing.

No clear consensus exists on how child survival programs should react to their own success. It is clear that as child mortality declines, the structure of deaths by cause will change, implying a need for changing program priorities. Where child mortality has reached quite low levels, there is clearly a need to progress from highly targeted programs to the development of broad, community-based health services. Many countries in the developing world, for example Sri Lanka and Costa Rica, have reached this point, whereas many others, including most of sub-Saharan Africa, have not. Local knowledge of disease conditions is necessary both for targeting programs and for determining when and how to make the transition to broader service provision.

Program or service efficiency is likely to be affected substantially by its effects on disease burden and acquired frailty. In general, preventive interventions that avoid increments to frailty are likely to be more effective than palliative or curative interventions that may avert death but do not avoid reductions in overall health status.

Macro level relationships between mortality and economic conditions suggest that general economic development should have a strong effect on reducing mortality. However, the relationships at the household or individual level are weak or contradictory, suggesting that the macro level relationship may be reflecting unobserved processes. The apparent resilience of child mortality declines to economic stagnation or adversity in the 1980's further undermines faith in a rapid, direct reaction of child mortality to economic measures. Education, particularly of mother, appears to play an important role in reducing child mortality in both macro and micro level studies, but recent evidence suggests that this effect may also be reflecting unobserved processes related to maternal upbringing. Remedial education activities such as literacy campaigns thus may not produce the child mortality reductions expected of improved education.

What, then, are the implications for policy? The most fundamental policy question is whether child mortality can be reduced most effectively by general economic development or whether the development process can be short-circuited to speed up child mortality reductions through specific interventions. There seems to be a clear answer here: the effects of general development are at best slow, and at worst uncertain, whereas child survival interventions can speed mortality reductions even in the face of moderately adverse economic and social conditions (severe economic or social crises, such as those in China in the late 1950s, or Uganda in the 1970s, can overwhelm any positive effects of interventions, largely through disrupting the interventions themselves). Thus child survival programs can be expected to be more effective in achieving continued mortality declines than reliance on general development.

Given that continued emphasis on child survival interventions is desirable, the next question is the form that such interventions should take. There is no single answer to this question. There are still numerous countries where child mortality is high and health

infrastructure is weak; in such countries, emphasis on EPI, which can be delivered through periodic, centrally-organized campaigns, seems the best policy. The existing retail system can also be used to support distribution of ORS and anti-malarials. As child mortality falls, demand for services is likely to increase, and emphasis can shift onto the development of a community-based health system offering a rather limited number of preventive and curative services but incorporated into a referral system. At this stage emphasis should be put on maintaining a basic immunization program, on ensuring availability of a small set of essential drugs, on developing a health information system to inform intervention design, and on devising mechanisms for identifying and then accessing under-served populations. Training and supervision are likely to be key factors in a successful transition from one phase to the next.

Research into new technologies is also required. Priority areas should clearly be prevention or cure of AIDS, a vaccine against malaria (or an acceptable vector control method), heat-stable vaccines against measles and poliomyelitis, and vaccines against ARI, especially pneumonia. From a managerial point of view, the development of health information systems that provide rather than consume information, and of methods for evaluating health needs rapidly, should also be viewed as priorities.

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# Levels, Trends and Patterns of Child Mortality in the Developing World

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## Introduction

Since World War II, there have been substantial declines in infant and child mortality rates in most developing countries, resulting from both improvements in standards of living, and national and international level public health activity. The almost universal optimism which characterized discussions in the 1950s and 1960s about prospects for bringing about a child survival revolution in the third world has been replaced in the late 1980s by worries about the complexity of achieving, and resources required to achieve, further dramatic reductions in morbidity and mortality. There are at least three major types of concerns. First, the widespread implementation of economic stabilization programs in the developing world in the 1980s has led to understandable concerns about the effects of the ensuing economic recessions in slowing the pace of further child mortality reductions. Second, because of the practical (and often political) difficulties in organizing comprehensive community-based primary health care programs (Mosley, 1985), much of the public health effort of national governments and international agencies (for example the EPI and related CCCD programs<sup>3</sup>) has focussed on immunizable diseases and on malaria vector control and treatment activities. This focus on narrow interventions as opposed to broadly-based community health development has led to concerns about possible substitution effects in morbidity and mortality, e.g., that many children who no longer die of measles because they are immunized will simply die of other diseases, like diarrhea. Third, there are several relatively recent developments in international disease patterns, which are likely to have a major impact on trends in infant and child mortality rates in the next several years. The two most important are the AIDS epidemic and the spread of chloroquine-resistant malaria, both of which are likely to have a particularly devastating effect on infants and children in sub-Saharan Africa.

In the first part of this paper we review recent levels, trends and patterns of child mortality in the developing world. In the second part, evidence from this review and from other sources is used to examine the first two issues described above, i.e., evidence about the consequences of economic stabilization programs for infant and child mortality, and about whether programs aimed at particular diseases merely change the cause pattern of deaths. We have not attempted to address the third issue, because of the difficulty of

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<sup>3</sup> These programs are supplemented with ORT programs in most countries.

assessing the future effect of AIDS and the potential resurgence of malaria on infant and child mortality.

### Data Sources

Any study of child mortality in the developing world is bedeviled by shortcomings in the data of availability, accuracy and timeliness. The situation has been improving over the last two decades, however. Vital registration is gradually becoming more complete, particularly in the Americas and East Asia. Indirect estimation of child mortality levels and trends from information on proportions of children surviving by age of mother has proved surprisingly satisfactory and the necessary information has been widely collected over the last two decades. National surveys such as those coordinated by the World Fertility Survey and more recently the Demographic and Health Surveys that have included complete birth histories have contributed further to our knowledge of levels, patterns and differentials of child mortality. Despite these improvements, there are still large gaps, both of countries for which there are no reliable data and of countries with insufficient data from which to draw inferences about trends. It can always be argued that these gaps are a fatal flaw in any interpretation of the data available, on the grounds that the countries with data are systematically different from those without in respect to key organizational or developmental characteristics. We prefer to argue, however, that countries with no data are not evidence of any sort, and to focus our attention on countries with reasonably reliable estimates.

The task of examining levels and trends of child mortality for the developing world has been made easier by two recent publications, one a review of mortality under the age of five for the whole world, both estimates and projections covering the period from 1950 to 2025 (United Nations, 1988), and the other a review of all the child mortality data available for sub-Saharan Africa (Hill, 1987). The figures included in the United Nations publication represent the results of a major review of evidence regarding child mortality for all countries of the world with populations of 300,000 or more. The problem with using the U.N. numbers is that each country has to have a number for each quinquennium from 1950-55 to 2020-25. Clearly, for 1985 onwards, the numbers given are extrapolations from past trends. However, even up to 1980-85, many of the numbers are based on interpolation, extrapolation or what might be called ecological extrapolation (from neighboring countries) and there is no way of determining from the publication which numbers have an empirical basis and which do not. Hill's numbers are also the product of careful evaluation of the underlying data, but since data for sub-Saharan Africa are scarce these estimates are of very uneven quality, particularly for the more distant past, and are often hard to interpret.

In what follows, estimates from the U.N. report have been used for North Africa, Asia and the Americas, but an attempt has been made to exclude all those "estimates" not based on data. It is hoped that the numbers left are soundly based and relatively reliable. The selection has been done largely by the authors, with help from the author of the UN report, and may be somewhat subjective; it is entirely our responsibility. Numbers for the most recent past available from very recent surveys, such as the Demographic and Health Surveys, have also been incorporated where possible and where consistent with earlier

Table 1 : Countries Classified by Probability of Dying by Age 5 and Quinquennium, 1960-65 to 1980-85

Part A : Latin America

Range of Probability of Dying by Age 5, 5q0	Quinquennium				
	1960-65	1965-70	1970-75	1975-80	1980-85
350-399					
300-349					
250-299					
200-249	Peru		Bolivia Haiti	Haiti	
150-199	Ecuador Brazil	Honduras Peru Guatemala Ecuador	Honduras Guatemala Peru		Haiti
100-149	Colombia Chile Mexico Costa Rica	Brazil Colombia Mexico Chile	Ecuador Domin. Rep. Brazil Mexico	Peru Ecuador Brazil	Peru
50- 99	Panama Cuba Jamaica Argentina Puerto Rico Trinidad Uruguay	Costa Rica Panama Argentina Jamaica Cuba Uruguay Trinidad	Colombia Paraguay Chile Panama Costa Rica Argentina Uruguay	Domin. Rep. Mexico Colombia Venezuela Chile	Ecuador Domin. Rep. Brazil Mexico
25- 49		Puerto Rico	Jamaica Cuba Trinidad Puerto Rico	Uruguay Argentina Panama Costa Rica Jamaica Trinidad Cuba	Argentina Colombia Panama Uruguay Chile Trinidad
<25				Puerto Rico	Costa Rica Cuba Puerto Rico

Source: Largely based on U.N. (1988), excluding countries and periods with no empirical support, and updating estimates for Brazil, Dominican Republic, Ecuador, Peru, and Colombia on basis of DHS results, and Haiti on basis of EMMUS results.

17

Part B : Asia

Range of Probability of Dying by Age 5, 5q0	Quinquennium 1960-65	1965-70	1970-75	1975-80	1980-85
350-399					
300-349					
250-299	Nepal	Nepal	Yemen		
200-249	Turkey Papua-NG Indonesia	Pakistan Bangladesh Turkey Indonesia	Nepal Bangladesh Pakistan India	Yemen Bangladesh Pakistan	Bangladesh
150-199	Jordan China	Papua-NG Syria Jordan	Turkey Indonesia	India Turkey Indonesia	India
100-149	Thailand Philippines Kuwait Sri Lanka	Thailand Philippines China	Syria Jordan Philippines		
50- 99	Rep. Korea Malaysia	Sri Lanka Rep. Korea Kuwait Malaysia	Thailand China Sri Lanka Malaysia Rep. Korea Kuwait	Syria Philippines Jordan Thailand Sri Lanka China	Philippines Thailand
25- 49	Hong Kong Singapore	Hong Kong Singapore		Rep. Korea Malaysia Kuwait	Malaysia Sri Lanka Kuwait
<25			Singapore Hong Kong	Hong Kong Singapore	Hong Kong Singapore

Source: Largely based on U.N. (1988), excluding countries and periods with no empirical support, and updating estimates for Sri Lanka and Thailand on basis of preliminary DHS results and for Bangladesh on basis of preliminary DMTS results.

Part C : Africa

Range of Probability of Dying by Age 5, 5q0	Quinquennium				
	1960-65	1965-70	1970-75	1975-80	1980-85
350-399	Sierra Leone Malawi	Sierra Leone			
300-349	Gambia	Malawi Gambia	Malawi Mali	Mali	
250-299	Liberia Cent Af Rep Burkina Senegal Togo	Mozambique Senegal Egypt Liberia Burkina Cent Af Rep Benin Ivory Coast	Mozambique Liberia Senegal Burkina		Mali
200-249	Cameroon Tunisia Burundi Tanzania Rwanda Ghana Uganda Zambia Sudan	Somalia Tanzania Togo Burundi Cameroon Rwanda Tunisia Sudan	Egypt Benin Rwanda Burundi Cent Af Rep Somalia Tanzania Ivory Coast Cameroon	Liberia Senegal Rwanda Benin	Liberia Senegal
150-199	Kenya Lesotho Nigeria Congo Zimbabwe	Ghana Lesotho Zambia Nigeria Kenya Congo Zimbabwe	Lesotho Ghana Nigeria Kenya	Egypt Nigeria Ghana	Ghana
100-149			Zimbabwe	Zimbabwe	Egypt
50- 99					
25- 49					
<25					

Source: Largely based on Hill (1987), excluding countries and periods with weak empirical support, and updating estimates for Ghana, Liberia, Mali and Senegal on basis of preliminary or final DHS results.

estimates. Hill's results, again with additions from more recent surveys, have been used for sub-Saharan Africa. We have focussed exclusively on the period from 1960 to 1985, and have excluded all countries with an estimated population in 1980 of less than one million.

### Child Mortality Levels in Developing Countries

Table 1 shows countries of the developing world by region (Africa, the Americas, and Asia), quinquennium (from 1960-65 to 1980-85) and estimated probability of dying between birth and age 5 ( ${}_5q_0$ ). This indicator has been chosen in preference to other measures of child mortality because it represents cumulative mortality throughout early childhood to an age at which mortality rates are relatively low, and because it is generally well-estimated by indirect estimation techniques using proportions dead among children ever born. Countries are grouped according to ranges of values of  ${}_5q_0$ , for instance 150 to 199 per thousand live births, rather than by point estimates (the point estimates used are given in Appendix 1). Order within groups does, however, represent approximate ranking by  ${}_5q_0$ .

Three things stand out from Table 1: first, that child mortality is, and has been, higher in Africa than in the other two regions; second, that even within regions and time periods there is a tremendous range of child mortality conditions; and third, that in all three regions, child mortality has generally been falling over the last two and a half decades. For the quinquennium 1960-65, the median country by probability of dying by age five fell in the 50-99 range in the Americas, in the 100-149 range in Asia, and in 200-249 range in Africa. By the quinquennium 1980-85, the median Americas country and the median Asian country were both in the range 25-49, whereas the median African country was still in the 200-249 group. The general downward shift in child mortality over the period can be seen from the broad northwest-southeast pattern shown in all three parts of Table 1, though most clearly for the Americas. For Asia, there appear to be two distinct patterns, one of rapid decline along a northwest-southeast axis, and one of much slower decline along an almost horizontal axis. For Africa, patterns are harder to see because of the small number of observations for the two most recent quinquennia.

### Recent Trends in Child Mortality

In order to look in more detail at recent trends in child mortality, the percentage changes in  ${}_5q_0$  from one quinquennium to the next have been calculated for all countries having estimates deemed to be reliable for at least three consecutive periods. Percentage declines (a negative sign indicates an increase) are shown in Table 2.

Across all countries, the median decline is 11 percent for the period 60-65 to 65-70, 13.5 percent for the period 65-70 to 70-75, 16 percent for the period 70-75 to 75-80, and 17 percent for the most recent period 75-80 to 80-85. Thus in terms of percentage declines, there has been a slight acceleration in the pace of child mortality decline from the 1960s to the 1970s, with the higher rates of decline apparently maintained in the early 1980s. There are clear differences in the pace of decline by region. For the Americas and Asia, the percent declines tend to be from 10 to 30 percent per quinquennium,

Table 2 : Percent Decline in Probability of Dying by Age 5, 5q0,  
from One Quinquennium to the Next by Country, 1960-1985

Country	Percent Decline in 5q0 from first quinquennium to second			
	60-65 to 65-70	65-70 to 70-75	70-75 to 75-80	75-80 to 80-85
Cuba	21	26	38	29
Dominican Rep	*	*	18	10
Haiti	*	*	11	9
Jamaica	19	23	33	*
Puerto Rico	28	26	24	14
Trinidad	6	26	14	13
Costa Rica	21	27	45	31
Mexico	11	12	13	11
Panama	15	17	31	21
Argentina	6	15	17	13
Chile	18	29	34	46
Uruguay	-2	4	6	31
Brazil	9	10	14	10
Colombia	12	25	28	34
Ecuador	11	13	15	22
Peru	12	14	12	24
China	30	27	30	*
Hong Kong	32	34	24	25
Republic of Korea	23	20	21	*
Indonesia	11	14	10	*
Malaysia	21	14	26	11
Philippines	11	11	12	7
Singapore	26	26	30	25
Thailand	13	23	23	21
Bangladesh	*	0	3	3
India	*	*	9	16
Nepal	10	8	*	*
Pakistan	*	5	12	*
Sri Lanka	14	9	16	39
Jordan	24	23	24	*
Kuwait	32	25	24	36
Syria	*	22	22	*
Turkey	14	11	14	*

Table 2 : Continued

Country	Percent Decline in 5q0 from first quinquennium to second			
	60-65 to 65-70	65-70 to 70-75	70-75 to 75-80	75-80 to 80-85
Malawi	3	3	*	*
Central Af. Rep.	12	14	*	*
Burkina	4	1	*	*
Senegal	0	2	12	17
Liberia	5	1	12	9
Cameroon	10	11	*	*
Burundi	7	1	*	*
Rwanda	3	-5	2	*
Tanzania	3	4	*	*
Ghana	10	11	14	-7
Nigeria	7	12	0	*
Kenya	11	11	*	*
Lesotho	5	3	*	*
Zimbabwe	1	5	6	*
Mali	*	*	7	10
Egypt	*	14	23	20
Benin	*	22	22	*

Notes: Percent decline from period i to period i+1 calculated as  
 $100[1 - 5q_0(i+1)/5q_0(i)]$   
 Only countries with at least three consecutive estimates of 5q0 deemed  
 reliable are included  
 \* indicates not available

FIGURE 1: PERCENT DECLINES IN 590 BETWEEN QUINQUENNIA BY TIME PERIOD AND REGION; 25TH, 50TH AND 75TH PERCENTILES OF COUNTRIES

PERCENT DECLINE  
BETWEEN QUINQUENNIA

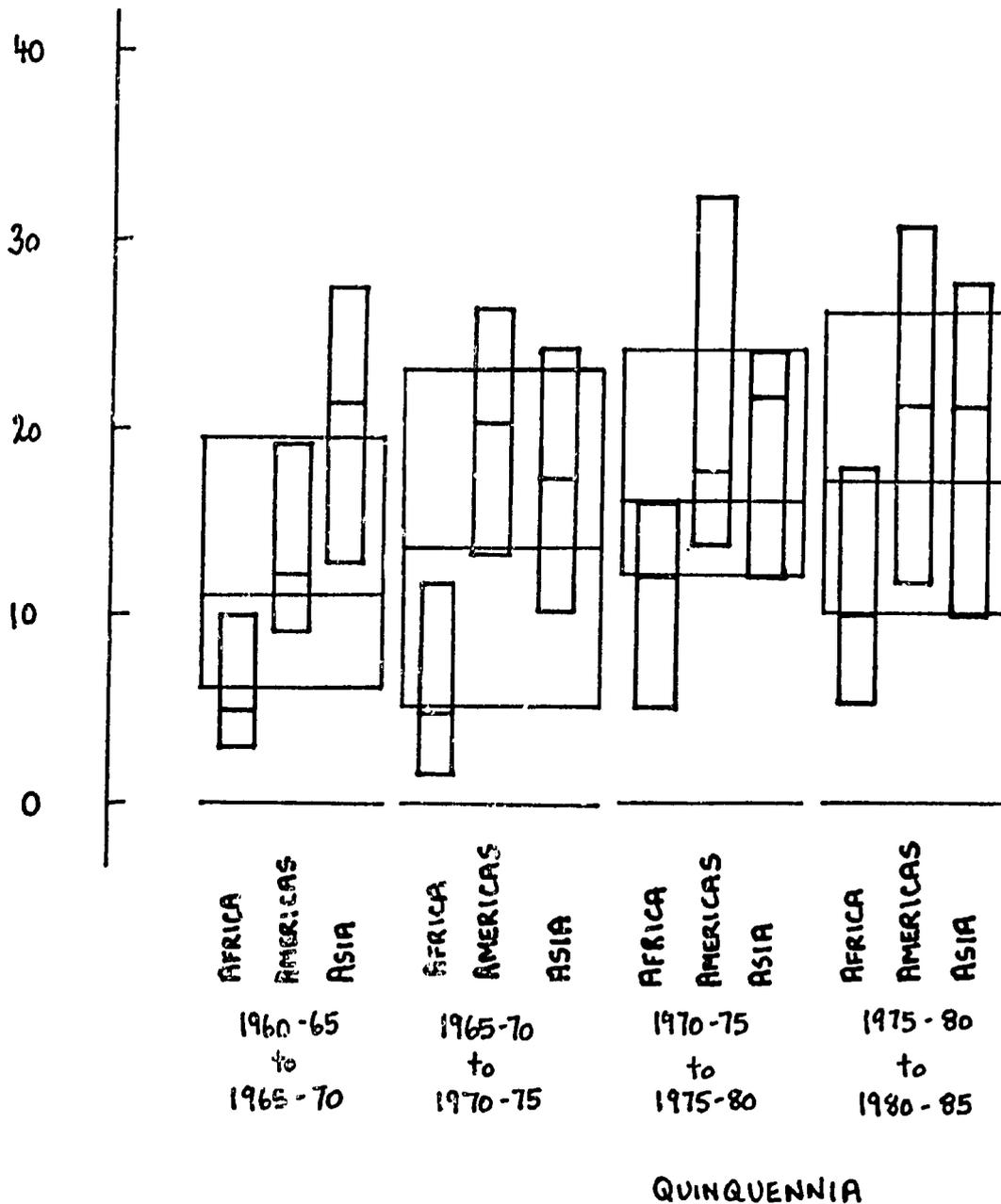
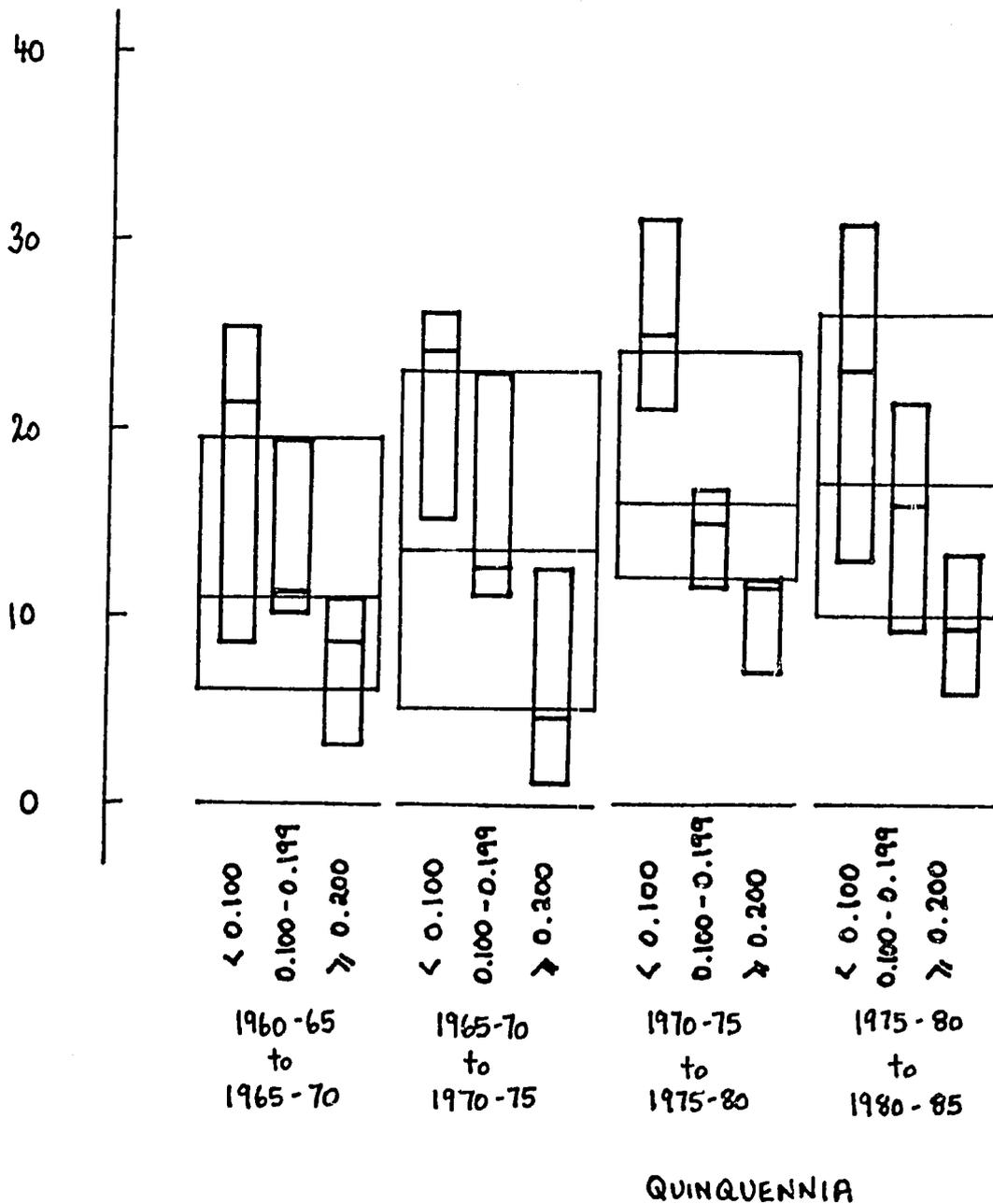


FIGURE 2: PERCENT DECLINES IN  $\dot{Y}$  BETWEEN QUINQUENNIA BY TIME PERIOD AND INITIAL  $\dot{Y}$  LEVEL; 25TH, 50TH and 75TH PERCENTILES OF COUNTRIES

PERCENT DECLINE  
BETWEEN QUINQUENNIA



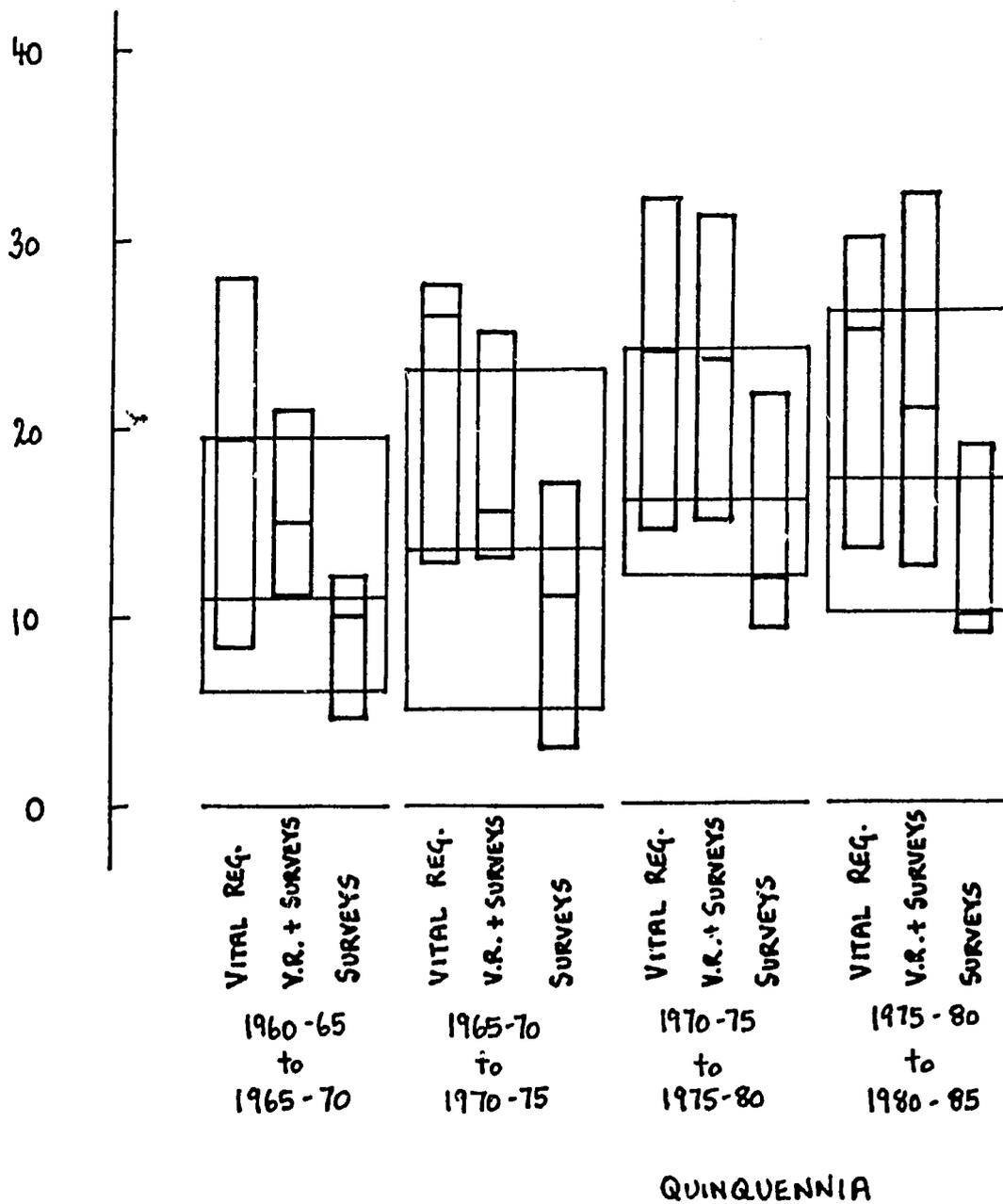
whereas for Africa they range from about zero to 15 percent. The median declines are, for the Americas, 12, 20, 17.5 and 21 percent, for Asia, 21, 17, 21.5 and 21 percent, and for Africa, five, 4.5, 12 and 10 percent, for the quinquennia 1960-65 to 1965-70, 1965-70 to 1970-75, 1970-75 to 1975-80, and 1975-80 to 1980-85 respectively. Child mortality appears to have been declining more slowly in Africa than elsewhere in the developing world throughout the period studied. This pattern is shown more clearly in Figure 1, where percent declines between quinquennia are plotted in the form of percentile boxes by region (each box represents the 25th, 50th and 75 percentiles of the country percent declines; the broad box for any time period includes all the observations, the slim boxes represent the three regions separately; the lower horizontal line of each box represents the percent decline below which 25 percent of countries fall, the upper horizontal line the percent decline above which 25 percent of countries fall, and the central horizontal line represents the median decline). The declines for Africa are clearly lower than the declines for the Americas or Asia.

The presentation in Table 2 and Figure 1 may be challenged on two grounds. The first is that percentage decline, which gives the same score to a decline of five points from a starting point of 50 as to a decline of 20 points from a starting point of 200, is not a suitable index of change. Since Africa started at a higher level, on average, its percent declines may tend to be smaller than for those regions which started at a lower level. On the other hand, percent decline has an intuitive appeal over absolute decline, since it seems it should be programmatically easier and less expensive to move  $s_q_0$  from 220 to 200 than from 30 to 10. There is no right answer, and no perfect yardstick, but to see whether starting level has a major influence, Figure 2 plots percent change between quinquennia as percentile boxes by range of  $s_q_0$  at the start of the period. There does seem to be a clear starting point effect, with slower declines in those countries with an initial level of 200 or more, an intermediate pace of decline for those countries with an initial level between 100 and 199, and faster declines for those countries with an initial level below 100. The pace of decline within these initial level groups appears to have remained roughly constant across time periods, however.

The second ground for reservations about the apparently accelerating pace of child mortality decline into the early 1980's is the selection for countries with very recent data. These countries tend to be those with reliable vital registration data or those with a recent demographic survey. It could be argued that the conditions that govern data availability also predispose towards more rapid mortality change. There is no completely satisfactory answer to such an argument, but as a partial answer the percentage declines by quinquennia are shown in Figure 3 in percentile boxes arranged by the type of data on which they are based. The three categories of data type used are first, vital registration, second, vital registration supplemented by censuses or surveys, and third, censuses or surveys only. The pattern that emerges is somewhat less clear than that seen in Figure 2, by initial level of child mortality, but seems to show little difference between the vital registration and supplemented vital registration groups, but slower declines throughout for the survey only group. What is important, however, is that there is no indication of any change over time in the pace of decline within groups. Thus the apparent acceleration of the pace of percent decline in the late 1970s and early 1980s may be the result of selection effects, but such effects are unlikely to be masking any real deceleration in the

FIGURE 3: PERCENT DECLINES IN  $590$  BETWEEN QUINQUENNIA BY TIME PERIOD AND TYPE OF DATA SOURCE; 25TH, 50TH AND 75TH PERCENTILES OF COUNTRIES

PERCENT DECLINE  
BETWEEN QUINQUENNIA



pace of decline.

### The Pace of Mortality Decline

Several observers have argued that the pace of mortality decline slowed in developing countries in the period from the mid-1960 through the mid-1970s, compared with the two decades following World War II (Preston, 1985; Gwatkin, 1980; Palloni, 1985). Gwatkin (1980) believed that the slow down was an important indication of the limits of exogenously-developed, vertical, high tech programs to reduce mortality in the absence of major changes in the standard of living. Others argue that the reason was partly that commitments of international assistance for health purposes, particularly malaria control, lagged during this period (Preston, 1985). While generalizations have been made to the entire third world from examination of changes in estimates of the expectation of life over this period, the slow down was probably principally a Latin American phenomenon. Reliable mortality data for sub-Saharan Africa are not available for the 1950s (or even the early 1960s, for most countries), so that it is not possible to draw conclusions about the pace of mortality change during this period for this region. While the pace of mortality decline may have slowed during this period in some parts of non-industrialized Asia, it is clear that the mortality decline in China accelerated, rather than decelerated, in the late 1960s and the early 1970s. The same is probably true also of India (Bhat et al, 1984).

In contrast to other areas in the developing world, reasonably reliable data for the period from the 1950s through the 1970s are available for a number of Latin American countries. Careful examination of these data does suggest a slow down in the pace of mortality decline in the 1960s and early 1970s relative to the post-war period (see Palloni, 1985). The principal reason is that the infant and child mortality associated with malaria had been brought under control in most countries relatively rapidly in the post-war period, bringing about a substantial mortality decline, and these declines were hard to match in later years. Nonetheless, as the figures in Table 2 show, in the period subsequent to 1960, many Latin American countries have experienced impressive declines in infant and child mortality. Several countries now have levels of infant and child mortality approaching those of industrialized countries.

A more recent concern has been about the effect of economic stabilization policies, undertaken by a number of countries in the early 1980s, on the pace of infant and child mortality decline. In general, the data presented above indicated that there is no clear evidence that the overall pace of child mortality decline has changed greatly between the 1960s and the 1980s. Child mortality reductions in the 1970s appear to have been slightly faster than in the 1960s, but on the evidence now available, declines in the early 1980s appear to have maintained the pace of declines in the 1970s. Although the number of observations for the most recent period is small, the structural adjustment policies of the early 1980s do not seem to have slowed mortality declines substantially, if at all, at least not yet. There are individual exceptions, however. Child mortality does appear to have increased somewhat in Ghana from the late 1970s, at a time of severe economic recession. On the other hand, child mortality in Jamaica and Chile, both countries with negative growth of GNP per capita from 1965 to 1986 (World Bank 1988) has declined

rapidly, and in Peru, another country with unfavorable recent economic conditions, the declines in child mortality appear to have accelerated into the early 1980's. Thus whatever the pros and cons of structural adjustment programs from other points of view, they should not be criticized on the grounds that they raise, or slow declines in, child mortality.

### Postponing Rather than Averting Child Deaths?

In general, an intervention targeted at one particular disease can have one of four effects (assuming it is at least minimally effective): it can (i) reduce mortality from the disease, with synergistic benefits for other diseases, thus reducing cause-specific mortality from the disease and reducing overall mortality by rather more; (ii) reduce mortality from the disease only, reducing cause-specific mortality and overall mortality by a corresponding amount; (iii) postpone deaths from earlier to later childhood, either from the same or from different causes; and (iv) merely shift deaths from the target cause to different causes at the same ages of childhood. A broad spectrum intervention would not have effect four, and would not be expected to have effect three, but would be expected to reduce mortality rates over a range of targeted causes.

The issue of synergism or reverse synergism, that is, effect (i) or either effect (iii) or effect (iv), is primarily associated with two major diseases--measles and malaria--and the issues in each case are somewhat different. In the case of measles mortality, the issues are summarized by Foster (1984, p. 128):

From the perspective of child survival, health planners are challenged by three conflicting hypotheses: 1. Measles immunization will increase child survival in proportion to measles deaths prevented. 2. Measles immunization will, because of its synergistic impact with other major causes of under-5 mortality, increase survival in excess of measles deaths prevented. 3. Measles immunization will not increase child survival because deaths prevented through immunization will be replaced by deaths from other causes.

Evidence on the effects of reducing measles incidence is limited and comes primarily from studies in sub-Saharan Africa where severe measles is currently a more important cause of death among children than in other areas of the third world. One of the studies (Stephens, 1984) strongly suggest that reduction of measles mortality has a larger effect on overall levels of under 5 mortality than would be expected from the number of measles deaths alone. In other words, this study indicates that reducing the incidence of measles not only reduces deaths from measles but also reduces deaths from other causes as well, most probably because children who survive a severe case of measles are at greater risk of dying from other causes than children who never had measles<sup>4</sup>.

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<sup>4</sup> A recent study by Aaby et al. (1988) indicates that reduction in the incidence of measles as a consequence of immunization programs also results in less severe cases of measles for unvaccinated children who do contract measles, since they are exposed to a lower

Some of the issues in the case of malaria are the same: will reduction in the incidence of malaria reduce child deaths from other diseases as well, or will many children whose deaths are prevented by a malaria control program go on to die in childhood of other diseases? Evidence from the mortality declines in Sri Lanka and Guyana as a result of malaria eradication programs suggests that the control of malaria substantially reduces child deaths from both malaria and from other causes as well (Molineaux, 1985). However, there is also evidence from a study in Garki, Nigeria that control of *P.falciparum* malaria prevented fewer deaths than was expected by looking at malaria-specific mortality rates, because the "very removal of malaria allowed more deaths from other causes." (Molineaux, 1985,p.35)<sup>5</sup>

An additional concern in the case of malaria is that reduction in the frequency of inoculation (being bitten by mosquitos carrying parasites) as a consequence of a malaria control program may change the age-pattern of infection. Populations exposed regularly to malaria develop a temporary form of immunity to malaria infection which is continually reinforced by new exposure to the parasite. In a population living in a malarious area infants are exposed to malaria at an early age and, if they develop an infection and survive, they begin to build up immunity. Therefore, malaria mortality is concentrated at fairly young ages, although not in the first months of life when infants appear to be protected by residual maternal immunity (Molineaux, 1985). However, if malaria control is reasonably effective, children are more likely to receive their initial inoculation at a later age. Therefore, the effect of a malaria control program may be to change the age distributions of malaria deaths from the first years of life to later in childhood. Evidence on this issue is scarce and contradictory. However, since it is clear that effective malaria control programs can bring about major reductions in malaria-related mortality, it is likely that the overall mortality rates from malaria would be substantially reduced at all ages throughout childhood, even if a shift in the distribution of ages at death from malaria occurred.

Returning to the general effects of a targeted intervention, effect (iv) above will have no effect on either levels or age patterns of childhood mortality, though cause-specific rates will change. Thus if aggregate child mortality is declining, pure substitution of causes is not occurring; substitution is an important consideration when evaluating individual interventions, but is not important in examining aggregate changes.

The third effect, postponement, has to be given some upper age limit since all deaths are postponed rather than averted without such a limit. The effect of postponement will be to leave unchanged the probability of dying by this limit age, but to reduce the probability of dying in the first part of the age interval, and increase it in the second part of the interval. A reasonable upper age limit to use may be age five, since the probability of dying between age five and age 15 is, under normal circumstances, only a small fraction of the probability of dying before age five (typically around one fifth), suggesting much lower frailty above age five and thus little chance of postponement to

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dose of measles virus.

<sup>5</sup>However, it is not clear whether death rates from other causes increased or whether only the number of deaths from other causes increased.

such ages. Use of an index of child mortality such as the infant mortality rate, with a young age cut-off, might indicate a substantial decline in child mortality under conditions of postponement where no such decline in the probability of dying by age five had occurred.

WFS and DHS maternity history data allow us to examine age patterns of mortality under age five as overall childhood mortality has declined. If child survival programs were having substantial postponement effects, we would expect to see changes in the relationship between mortality under the age of two and mortality between the ages of two and five. Unfortunately, there are only three countries with both WFS and DHS surveys and tabulations from the DHS of probabilities of dying by both age two and age five. However, for those three countries (Peru, Republica Dominicana and Ecuador) the relationship between the probability of dying by age two,  ${}_2q_0$ , and the probability of dying between age two and age five,  ${}_3q_2$ , has conformed very closely to model patterns as the overall level of mortality before age five has declined. Figure 4 shows these relationships for a number of five year periods, in combination with comparable relationships from the four families of Coale-Demeny (1982) model life tables. Peru and Ecuador follow the model relationships for the 'South' or 'West' families extremely closely over almost two decades and a halving of mortality by age five. The Republica Dominicana data are less consistent between the WFS and DHS, but the relationships still fall within the bounds of model expectations. Thus the available data do not indicate any postponement of child mortality under the age of five.

## Conclusions

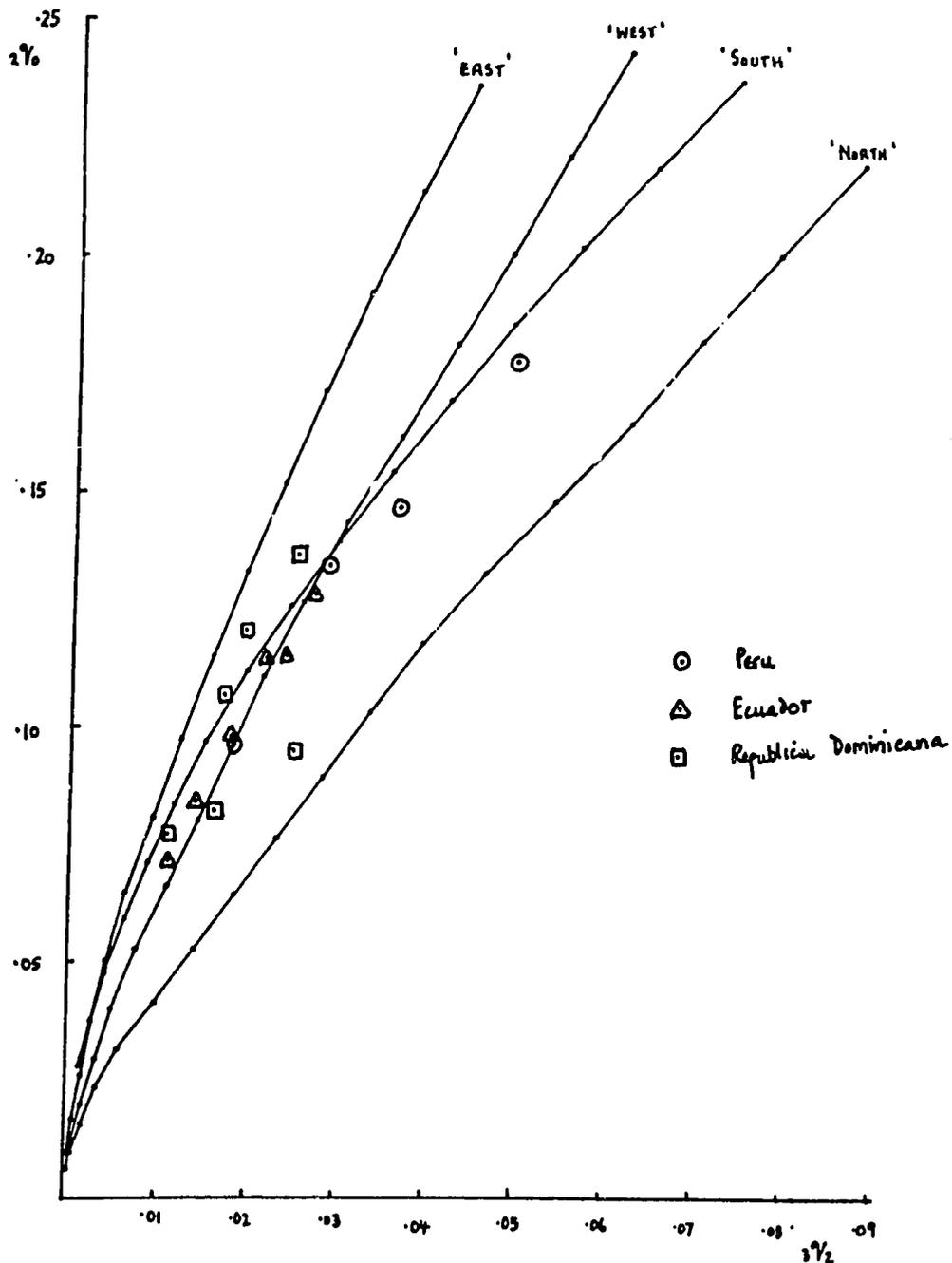
This review of levels and trends of child mortality in the developing world from the early 1960s to the present gives rise to several important conclusions.

First, the disparities in child mortality between countries and regions of the developing world are enormous and appear to have widened over the period. The difference between best and worst in the early 1960s seems to have been a factor of about seven, and by the early 1980's this factor had increased to about 15, even if two countries with mortality levels of developed countries, Hong Kong and Singapore, are excluded.

Second, child mortality declines in sub-Saharan Africa seem to be slower than those in the other two regions. This result may be associated more with the initial high mortality level in Africa than with the region itself, however. Other high mortality populations, such as those of the Indian subcontinent (excluding Sri Lanka) and Haiti, have also experienced below average declines. However, even the success stories of sub-Saharan Africa, such as Kenya and Zimbabwe, have not experienced rates of decline similar to those of countries of other regions with similar child mortality levels in the early 1960s.

Third, though related to the second, there does seem to be a general pattern whereby percentage declines in child mortality for countries with high initial child mortality are lower than those for countries with moderate or low initial child mortality. The dividing line seems to be a level of  ${}_3q_0$  of around 150 per thousand. Once this level is breached, declines appear to be rapid and self-sustaining, whereas above this level, declines appear slower and more vulnerable to other factors. This third conclusion is

FIGURE 4: RELATIONSHIPS BETWEEN THE PROBABILITY OF DYING BY AGE 2 AND THE PROBABILITY OF DYING BETWEEN AGE 2 AND AGE 5: DOMINICAN REPUBLIC, ECUADOR AND PERU



rather speculative, however, and as with all threshold theories requires further explanation of the mechanisms involved.

Fourth, from the available evidence, we find little basis to conclude that there has been a general slowdown in the pace of mortality decline in the early-1980s as a consequence of economic conditions, or other circumstances. Furthermore, while there does appear to have been some slowing of the pace of decline in Latin America in the 1960s, we conclude that this slowdown may well have been limited to Latin America, and did not necessarily signal that the future potential of public health programs to reduce mortality in all areas of the third world had been partly exhausted, as some observers suggested.

Finally, we conclude that there is little evidence to support the notion that public health interventions merely change the causes or delay the occurrence of child deaths rather than prevent them. This is an area where considerably more research is needed. However, the evidence that exists suggests that the interaction of diseases is very important, and that eliminating or reducing the incidence of one disease is likely to reduce mortality from several causes.

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Appendix 1

Estimates of 5q0 by Country and Period

	60-65	65-70	70-75	75-80	80-85
<u>Latin America</u>					
Cuba	0.077	0.061	0.045	0.028	*
Dominican Rep	*	*	0.132	0.098	0.088
Haiti	*	*	0.232	0.207	0.189
Jamaica	0.077	0.062	0.048	0.032	*
Puerto Rico	0.054	0.039	0.029	0.022	0.019
Trinidad	0.053	0.050	0.037	0.032	0.028
Costa Rica	0.112	0.088	0.064	0.035	0.024
Guatemala	*	0.193	0.162	*	*
Honduras	*	0.195	0.171	*	*
Mexico	0.127	0.113	0.100	0.087	0.077
Panama	0.097	0.082	0.068	0.047	0.037
Argentina	0.072	0.068	0.058	0.048	0.042
Chile	0.136	0.112	0.079	0.052	0.028
Uruguay	0.053	0.054	0.052	0.049	0.034
Bolivia	*	*	0.244	*	*
Brazil	0.152	0.139	0.125	0.107	0.086
Colombia	0.135	0.119	0.089	0.064	0.042
Ecuador	0.175	0.156	0.136	0.116	0.090
Peru	0.218	0.194	0.159	0.147	0.112
<u>Asia</u>					
China	0.162	0.113	0.083	0.058	*
Hong Kong	0.047	0.032	0.021	0.016	0.012
Republic of Korea	0.099	0.076	0.061	0.048	*
Indonesia	0.225	0.201	0.173	0.155	*
Malaysia	0.091	0.072	0.062	0.046	0.041
Philippines	0.128	0.114	0.101	0.089	0.083
Singapore	0.042	0.031	0.023	0.016	0.012
Thailand	0.136	0.118	0.091	0.070	0.055
Bangladesh	*	0.228	0.228	0.221	0.215
India	*	*	0.218	0.199	*
Nepal	0.290	0.260	0.240	*	*
Pakistan	*	0.239	0.226	0.200	*
Sri Lanka	0.101	0.087	0.079	0.066	0.040
Jordan	0.197	0.150	0.116	0.088	*
Kuwait	0.107	0.073	0.055	0.042	0.027
Syria	*	0.160	0.125	0.097	*
Turkey	0.239	0.206	0.184	0.159	*
Yemen	*	*	0.290	0.249	*
Papua-New Guinea	0.232	0.193	*	*	*

<u>Africa</u>	60-65	65-70	70-75	75-80	80-85
Burundi	0.243	0.226	0.223	*	*
Kenya	0.199	0.177	0.158	*	*
Malawi	0.355	0.344	0.333	*	*
Mozambique	*	0.282	0.282	*	*
Rwanda	0.229	0.222	0.234	0.230	*
Uganda	0.212	*	*	*	*
Tanzania	0.237	0.229	0.219	*	*
Zambia	0.207	0.187	*	*	*
Zimbabwe	0.155	0.153	0.145	0.137	*
Cameroon	0.249	0.225	0.200	*	*
Cent.African Rep.	0.293	0.257	0.220	*	*
Congo	0.180	0.161	*	*	*
Egypt	*	0.280	0.240	0.186	0.148
Sudan	0.200	0.200	*	*	*
Tunisia	0.245	0.210	*	*	*
Lesotho	0.197	0.188	0.183	*	*
Benin	*	0.255	0.237	0.215	*
Burkina Faso	0.283	0.273	0.269	*	*
Cote d'Ivoire	*	0.255	0.217	*	*
Gambia	0.347	0.343	*	*	*
Ghana	0.218	0.196	0.174	0.150	0.160
Liberia	0.294	0.279	0.275	0.243	0.220
Mali	*	*	0.325	0.302	0.272
Nigeria	0.196	0.182	0.161	0.161	*
Senegal	0.282	0.282	0.275	0.242	0.200
Sierra Leone	0.392	0.381	*	*	*
Togo	0.267	0.227	*	*	*

# Who Uses Maternal and Child Health Services? Evidence from the Demographic and Health Surveys

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## The Demographic and Health Surveys

The Demographic and Health Survey (DHS) program, funded by U.S.A.I.D., is a follow-on activity to the World Fertility and Contraceptive Prevalence Surveys. The purpose of the DHS program is to assist less developed countries in conducting nationally representative population and health sample surveys to provide information for policy and program decision-making and for scientific research. In its first phase during 1984-89, the DHS will have assisted 30 countries to conduct 35 surveys. During its second phase, 1988-93, the DHS will assist 25 countries to conduct 30 more surveys. The data collected by the DHS surveys cover information on fertility and infant and child mortality levels and trends, use of family planning, attitudes towards fertility and family planning, marital status, breastfeeding, various maternal and child health indicators, anthropometry and socioeconomic characteristics. The respondents to these surveys are women 15 to 49 years of age<sup>2</sup>. In some countries of Asia and the Near East only ever-married women were interviewed; in the rest never married women were also interviewed. The sample sizes of the surveys range between 3,000 and 10,000 respondents.

## The Health Content of the DHS

The health content of the DHS focuses on maternal and child health and provides information for many indicators used to evaluate major child survival programs. The following information was collected for each child born in the last five years: Whether the mother received prenatal care and who provided the care, whether the mother received at least one injection to protect the baby against neonatal tetanus, and who assisted the delivery; Whether the child breastfed, age at stopping and reasons for stopping; For living children: Vaccinations from health card records against tuberculosis (BCG), diphtheria, pertussis and tetanus (DPT), poliomyelitis and measles; Use of oral rehydration therapy--ORT (home solutions and prepared packets--ORS) for the treatment of diarrhea; whether medical treatment was received for diarrhea; And in Africa whether medical treatment was received for cough and fever. In certain countries, additional questions were asked about the preparation of ORS and whether solid foods and liquids were given as usual during diarrhea. For the last child, information is obtained on the frequency of breastfeeding, whether bottle-fed and whether received food supplementation. See appendix 1 for the health section of the "B" core questionnaire.

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<sup>2</sup> In a couple of countries the upper age limit of respondents was forty-four years.

## **The Use of Maternal and Child Health Services: Individual Indicators**

Several questions in the health section of the DHS questionnaire indicate use of the maternal and child health services of a country. These services include those available in both the public and private sectors of the economy, and in this paper we do not distinguish between them. Indeed, for only a few countries is information available from the DHS for some of the indicators as to where the services were obtained. The frequency of contact with health services will depend on both the availability of services (supply) and the demand for and the need to use the services.

For the illustrative purposes of this paper, we have selected to use the survey data of seven countries from three continents: Brazil and Peru from Latin America, Burundi, Liberia and Senegal from Africa, and Sri Lanka and Thailand from Asia. The choice of these countries was dictated in part by the availability of and permission to use data at the time of writing.

### **Basic Health Indicators**

Table 1 presents a number of basic health indicators that give some overview of the extent to which the mothers in reproductive ages have used services related to their own health as well as that of their children. Three basic demographic indicators (infant mortality rate, child mortality rate and total fertility rate), and three socioeconomic indicators (percentage of women with primary education, percentage of women with no education, and percentage of women residing in urban areas) are presented in this table as well. The purpose of this table is to provide a general overview of the characteristics of the countries that were included in this study.

If infant and child mortality are considered indicators of the situation of children in the countries, then it is evident that the African children are in the most precarious conditions, whereas the two Asian countries show the lowest levels of mortality. The African countries show the smallest proportions of contraceptive use and the lowest educational levels. In Burundi, for instance, 80 percent of the women have no education, and only one percent of the currently married were using modern contraceptives at the time of the survey.

Two of the selected indicators are the proportion of women who have received prenatal care from a medically trained person (physician, nurse, trained midwife) and the proportion of deliveries performed by a medically trained person. Since the definition of a "medically trained person" may vary from country to country, comparison among countries should be done with caution.

### **Health Indicators by Education and Residence**

In the sections that follow these health indicators are examined in their association with the educational level and the type of area of residence (rural vs. urban) of the mothers. Education indicates the amount of information to which the women have been exposed. Presumably the higher the educational level the broader their horizons and the more they will be willing to resort to efficient health services. On the other hand, education is also an indicator of social class or socioeconomic status. Women with little

or no education very likely belong to the more under-privileged social groups, and because of that their access to health services may be much more limited. It is for this reason that regardless of the country and the social group under study, education proves to be closely associated with demographic and health-related behavior.

Residence in an urban area indicates an easier access to better services, and it is usually associated with better education and access to more information.

### Infant and Child Mortality

Figure 1 shows the infant mortality rates ( ${}_1q_0$ ) according to the mother's education. This figure reveals the inverse correlation between the educational level of the mothers and child mortality. In the case of Peru, for instance, the mortality rates of the infants whose mothers have had no education are almost five times higher than the rates of children whose mothers have higher education (124 against 22). This tendency is evident in all these countries, with the only exception being Liberia. However, in this country the number of women with primary or higher education is very low, so these rates are not reliable. The same pattern is observed for child mortality, with the one exception again being Liberia (Figure 2).

The infant and child mortality rates as shown in Figures 3 and 4 for type of area of residence follow the expected pattern, the mortality rates being consistently lower in urban areas. One exception is found in Sri Lanka, where the infant mortality rates in rural areas are slightly lower than in urban areas. Child mortality, however, is substantially higher in rural areas also in Sri Lanka.

The gap between urban and rural areas varies greatly from country to country, probably reflecting variation in the geographical concentration of health services and other differences. In the case of Peru, the infant mortality rates in rural areas are almost twice as high as those in urban areas (54 against 101), whereas in Sri Lanka, as we have seen, there is hardly any difference.

### Prenatal Care and Deliveries

The differentials in mortality levels are, at least partially, due to differences in use of health services. As indicators of use we have observed the percentage of women that had pre-natal care and deliveries attended by medically trained personnel. Figures 5 to 8 show these percentages according to education and area of residence of the mother. The more educated the mother, the more likely it is that she will have had prenatal care and deliveries performed by a trained person, as can be seen in Figures 5 and 7.

Use of these services varies substantially from country to country. Again, Peru seems to be the country where the social gaps are greatest. Among the women with no education, 20 percent had prenatal care and only 12 percent were delivered by a medically trained person, while among the women with higher education these proportions are 98 and 97 percent, respectively. In Sri Lanka, on the other hand, the disparity between the uneducated and the more educated is more modest.

Area of residence is also a very important factor. As expected, women in urban areas tend to obtain prenatal care and be assisted at delivery by medically trained

personnel. In this instance also, Sri Lanka proves to be an exception since urban-rural differences are either trivial or very small (see Figures 6 and 8).

### Health Cards

The proportion of children with health cards is an indicator of the coverage of the immunization programs in the various countries. It can also be considered as an indicator of the extent to which women have access to health services directly affecting their children.

The bars in Figures 9 and 10 show the expected pattern. The percentage of children with cards is larger among children whose mothers are more educated and who live in urban areas. The gap between the uneducated and the more educated, and between urban and rural areas is not as wide as for the other indicators we have examined. These smaller gaps probably reflect the result of vaccination campaigns, which by their nature tend to reach a larger population and a wider range of groups than other health services.

### Immunization

The information about a child's immunization status was obtained from records kept by the mother, e.g. from immunization or "Road-to-Health" cards. The interviewers copied the dates from the card onto the questionnaire making it possible to calculate the age at immunization. If the mother did not have an immunization record, she was asked if the child had ever received any immunizations. The immunization coverage rates shown in Figures 11 to 13 consider a child to be vaccinated only if the information was obtained from a written record, and only if the child had received the vaccination during the first year of life. The denominator is all children in the age group 12 through 23 months.

Three vaccines, BCG, the third dose of polio, and measles, were chosen to illustrate the differences in coverage rates between countries, and the similarities in the patterns of coverage among subgroups in each country. The pattern for the third dose of DPT was almost identical to that shown for the third dose of polio vaccine.

### Area of Residence

All three immunizing agents showed better coverage in urban than in rural areas, however only minimal differences were observed for Sri Lanka. Over 80 percent of children 12-23 months of age in Sri Lanka had received BCG, and almost 70 percent were given three doses of polio vaccine, while under 40 percent had received measles vaccine by 12 months of age. A large differential was found for Senegal, where 10 percent of children in rural areas and almost 50 percent of children in urban areas had received BCG, less than one percent had been given three doses of polio vaccine in rural areas, compared to 17 percent in urban areas<sup>3</sup>. Consistently higher coverage rates in urban compared to rural areas were also seen in Brazil, Peru, Burundi, Liberia and Thailand.

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<sup>3</sup> Soon after the completion of the DHS Survey in Senegal, an accelerated program to boost immunization coverage rates was carried out. Cluster sample surveys carried out afterward showed much higher immunization coverage rates than those shown here.

## Education

In all countries, children of mothers with secondary education had the highest immunization coverage rates, while the lowest rates were seen in children of mothers who had no education. The smallest difference between the education groups was seen for Burundi. In Brazil 29 percent of children whose mothers had no education, compared to 73 percent of children whose mothers had secondary education had been given BCG. Peru, Liberia, Senegal, Sri Lanka and Thailand all showed a similar pattern.

## Secular Trends

Figures 14 and 15 show the cumulated percent of children vaccinated against poliomyelitis and measles for two age cohorts, children 12 through 23 months old, and children 48 through 59 months old. In these figures the percentages are calculated based only on the children who had a health card.

Although many children are not yet reached by immunization services, all the countries included here demonstrated improvement in the vaccination coverage rates over the five years covered by the survey. For example, in Peru only 17 percent of children with an immunization card who were 4 years old at the time of the survey had received the third dose of polio vaccine before they had reached 12 months of age. In comparison over 33 percent of children with an immunization card in the youngest age cohort (12 through 23 months of age) had been given three doses of polio vaccine during the first year of life. In Sri Lanka, measles vaccination given during the first year of life increased from two percent of the oldest age cohort, to almost 50 percent of the youngest cohort.

## Treatment of Diarrhea

Figure 16 shows the percent of children treated with oral rehydration solution (ORS) made from packets among children who had experienced diarrhea at some time during the 2 weeks prior to the survey. Only a small proportion of children with diarrhea received oral rehydration fluid made from packets in Brazil (9%), Peru (4%), Liberia (7%), and Senegal (2%). In Burundi (30%), Sri Lanka (29%), and Thailand (37%) a larger proportion of the mothers had used ORS packets.

In all countries, ORS packets were used more frequently in urban than in rural areas; the differences were especially large in Burundi and Sri Lanka.

The level of the mother's education did not affect the use of ORS packets as consistently as occurred with immunization coverage rates.

## **The Use of Maternal and Child Health Services: Index of Use of Maternal and Child Health Services**

In order to summarize the use of maternal and child health services, an index was developed by combining the indicators available from the standard recode files of the DHS surveys. This index should be taken only as a rough guide to contacts with the health services. The index has four components: care during pregnancy and childbirth, vaccination, treatment of children's diarrhea and use of family planning. Due to limitations in the availability of this information, the index is restricted to mothers of living children

between ages 12 and 59 months at the time of interview. Mothers of younger children were excluded because their children may not have lived long enough to have been vaccinated. The specific indicators that form the index and the point values that each contributes are shown in Table 2.

A few words about the construction of the index: The purpose of the index is to measure mothers' contacts with health services, but many of the specific indicators in the DHS pertain only to living children under five years of age. For these indicators, if a woman has more than one child between ages 12 and 59 months, a subtotal is made for each child, and then the maximum value for any child formed part of the mother's index. The treatment of diarrhea is based only on children who were reported as having had the disease in the last two weeks. Due to this restriction, children who had the disease but were not treated by a medically-trained person or received ORT had one-half point deducted from their partial index score, and treated children had one point added to their score. The negative points were assigned so that an approximate average of zero would result for all children reported to have had the disease, equivalent to the zero points for children who were reported not to have had the disease. In Liberia, the questions on prenatal care and delivery were only asked for the last child, but we think that this had little effect on the index value.

### Analysis of the Index

The mean values of the health services usage index (HSUI) for each of the seven countries are shown in Figure 17. Sri Lanka, Thailand and Brazil show the highest values and Burundi and Senegal the lowest. The value for Liberia is higher than expected, perhaps due to the relatively high number of deliveries that were reported to have been performed by a trained person in this latter country (see Table 1).

In order to ascertain who scores low and high on the HSUI, a multiple classification analysis (a form of dummy variable regression) was performed with the index value as the dependent variable. Predictors (independent variables) chosen for this analysis were type of area of residence, level of education, and mother's age, parity, and marital status, all at the time of the interview.<sup>4</sup> A second analysis was performed with literacy substituted for level of education (see Table 3).

Given that the analysis was on the level of the individual mother, the predictive value of the variables used (as shown by the R-squares) is surprisingly high in Peru (48 percent of the variance in the index explained) and Senegal (34%). Even though the equation was statistically significant, the chosen predictors did not do too well in Brazil and Burundi, where only 8 and 9 percent of the variance was explained, respectively.

Substituting literacy for level of education did not alter the amount of

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<sup>4</sup> The categories for the predictors were: residence (urban, rural); level of education (none, primary, secondary, higher--no distinction by completeness); literacy (cannot read, reads with difficulty, reads easily); mother's age (15-19, 20-29, 30-39, 40-49); parity (1 to 4 children, 5 or more); marital status (never married, married or in a consensual union, formerly in a union).

variance explained except in Thailand, where it rose by three percentage points to 18 percent.

Table 4 summarizes the results of the regression analyses. In all the countries education, literacy and type of area of residence were significant at the one percent level. Age of mother was not significant in Peru and Senegal but was significant at the one percent level in the remaining countries. Marital status was not significant in the African countries, and parity was not significant in Peru, Burundi, Senegal and Sri Lanka.

In Figures 18 to 23, the adjusted mean values of the index are shown for the seven countries according to the categories of the predictor, one predictor per figure while holding constant the values of the other predictors used. The differentials shown between categories are those that remain after controlling for the predictors not shown in the figure.

### Education and Literacy

Even after removing the effects of residence, age, parity and marital status, the use of health services varies markedly according to education in Peru, the African countries and Thailand but only to a lesser extent in Brazil and Sri Lanka (Figure 18). In these latter two countries only mothers with no education use the health services substantially less as evidenced by the index.<sup>5</sup> When literacy in three categories is used in the regression instead of level of education, about the same level of explanatory power is achieved (Figure 19).

### Type of Area of Residence

Figure 20 shows the effect that the type of area of residence has upon the use of health services. In all countries rural mothers are at a disadvantage in comparison with urban mothers. In Brazil and Sri Lanka the differences between rural and urban areas are small but are still statistically significant. The differences in use are particularly large for Peru, Burundi and Senegal.

### Current Marital Status

Marital status may affect access to health services for several reasons: a mother with a husband or a partner may be in a better economic position, she may be more likely to have someone to take care of the children (either the husband or in-laws) while she is away from home using the health service, and in some cases she may be entitled to use a subsidized health service (social security for example) because of her husband's job.

Figure 21 reveals that women in a marital union in the Latin American and Asian countries have indeed made more use of the health services than other women (note that never married women were not included in the surveys in Sri Lanka and Thailand).

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<sup>5</sup> In Figure 18 the values for higher education are not shown for Burundi and Senegal because of the small number of cases (less than 25).

In the African countries, the results are not the same. Only small and statistically insignificant differences exist for Burundi and Liberia, and in Senegal never married mothers have made more use of the health services.<sup>6</sup> Perhaps the lower cultural importance of marriage in these African countries conveys less of an advantage to married mothers.

### Age and Parity

The relationships between mother's age and use of maternal and child health services are shown in Figure 22. We did not expect strong relationships for either this variable or the next, parity, shown in Figure 23. However, in four of the seven countries, mothers who were under 20 did show lower use than mothers 20-29. In Brazil, Burundi, Sri Lanka, and Thailand the over-forty mothers use the services less than mothers 20 to 39. The differences are not large in any of the countries except Burundi, where the trend with age is consistent and significant. In Burundi the mean value of the index is half a point higher for mothers 20-29 than mothers 40-49.

We chose to divide women into two parity categories, one to four children and five or more, because the unadjusted means by number of children ever born (not shown) appeared to differ most at this point. In only three of the countries, Brazil, Liberia and Thailand, is use significant by parity, and only in Thailand is there really much of a difference. In Thailand, mothers with fewer than five children have a score for health service use more than three-quarters of a point higher than mothers with five or more children. The difference for Thailand may be related to use of family planning, which forms part of the use index.

### Use by Socioeconomic Groups

Although the regressions discussed above show the effect of individual characteristics on the use of maternal and child health services, efforts to improve use generally target population groups which combine several characteristics. We have selected three sets of characteristics as examples of target groups for efforts to improve use of maternal and child health services. Our two groups are Low Rural (thought to be typical of the majority of persons in rural areas), Low Urban (disadvantaged adolescent mothers), and the third, High Urban (younger advantaged mothers) is shown for contrast. The specific characteristics of each group are shown in Table 5. By using the results of the regressions we can predict the values of the index of health service use for each of these groups.

Figure 24 gives the results of the calculations for the groups in each country. In all countries except Sri Lanka, there are quite large differences in use between the groups. The differences are particularly large between all three groups in Peru. In the African countries and Thailand, the main differences occur between the rural and the urban groups. In Brazil, the rural and urban low groups show almost no difference.

Comparing the same groups among countries, Figure 24 shows that the group with

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<sup>6</sup> This last result is not quite significant at the five percent level in the regression with education but is significant at that level in the regression with literacy.

the most variation is the Low Rural, which has a mean score of only 0.86 in Peru but the corresponding group in Sri Lanka has a score more than three points higher (4.21). Indeed this latter group has a higher mean score than the High Urban of Brazil, Peru, Liberia, and Senegal.

### Use of Health Services and Mortality

The index of health service use is very highly associated with the level of mortality of children less than five years of age. The mean values of the index for the seven countries are plotted against the level of under-five mortality in Figure 25. Only Liberia appears to have a somewhat too high level of health service use given its high level of mortality. We suspect this is the result of an overstatement in the Liberian DHS of the amount of prenatal care received. For the other six countries the correlation between the index and mortality is surprisingly high. Indeed, when a quadratic regression was applied to the data of the six countries excluding Liberia, the variation in the index accounts for more than 99 percent of the variance in mortality. We have not been able to detect a methodological problem that could result in this high level of association, but the result needs further testing.

### **Summary and Conclusions**

The Demographic and Health Surveys do shed some light on the use of maternal and child health services, although the subject has still not been fully illuminated. Basic health indicators for the seven countries examined here show that there is a great diversity among countries. Infant mortality rates vary from 25 to 144 deaths per thousand births. For child mortality rates (ages one to four years) the divergence is even greater, from 10 to 114. There are similar large disparities among the countries for prenatal and delivery care, immunization, use of ORT for diarrhea and use of family planning.

Within each country, there are large disparities among population groups. When individual indicators of use of health services are examined by education and for rural/urban residence, it is the uneducated and the rural who have used maternal and child health services the least but the differences among groups also varies markedly according to country. Sri Lanka appears to have the least disparities in use of prenatal and delivery care.

Immunization coverage also shows that differences in Sri Lanka are minimal but in the other countries coverage varies substantially by education and residence. However, analysis of coverage of immunization according to birth cohort of children shows that recent changes in immunization programs have improved coverage rapidly but that even the youngest cohorts are still far from adequately protected.

Oral rehydration still has not achieved adequate levels of coverage in the countries shown here. In no country does the use of ORS packets exceed 40 percent of the children with recent episodes of diarrhea.

In order to summarize the information on use of maternal and child health services, an index was built using the individual indicators and a multivariable analysis was performed. Again, the index reveals that there are large disparities among countries in their use of health services. Some of this disparity can, however, be attributed to differing

levels of education and residence, the two most important variables even after age, parity and marital status are held constant. These latter variables do not substantially affect use of health services. Brazil and Sri Lanka show only small differences by either education or residence indicating that health services are much more accessible in these countries than in the others, especially in Sri Lanka.

Combining the characteristics into illustrative target socioeconomic groups, large differences in use of health services appear between the disadvantaged groups and the privileged group except in Sri Lanka, where even low status rural mothers have relatively high use of health services. The differences are especially large among all the groups in Peru. Similar analyses should be performed to indicate the status of other potential target groups.

We feel that the DHS provides some valuable information for decision-makers and researchers on the use of maternal and child health services. We have only shown results for seven countries here and will extend this analysis to the rest of the DHS surveys that contain health information as the data become available. Large improvements in knowledge and decision-making would also result from following up with subsequent nationally representative monitoring. The DHS health component could also be strengthened with more detailed questions on source of service used and treatment of diseases other than diarrhea in all countries in future DHS surveys.

# Table 1.

SELECTED HEALTH, DEMOGRAPHIC AND SOCIO-ECONOMIC INDICATORS BY COUNTRY.

INDICATORS	COUNTRY						
	Brazil	Peru	Burundi	Liberia	Senegal	Sri Lanka	Thailand
<b>HEALTH INDICATORS</b>							
Percentage of Women who had Prenatal Care by Trained Persons	73	55	79	83	?	95	77
Percentage of Women who had Tetanus Toxoide Prior to Last Birth	--	--	59	71	31	66	65
Percentage of Deliveries Performed by Trained Persons	81	49	19	58	?	88	66
Percentage of Children Under 5 Years of Age Who:							
Have Health Cards	66	39	47	34	24	77	29
Immunized According to Their Mother's Report	--	57	26	32	39	--	54
Have Received all Immunizations, According to Health Card	--	--	46	16	23	47	35
Percentage of Children with Diarrhea in Last 2 Weeks who:							
Were Given ORS	9	9	30	7	2	29	37
Were Given Home Solution	2	46	8	3	5	10	6
Percentage of Women in Reproductive Ages and Currently Married who:							
Have Ever Used a Modern Method 1/ of Contraception	82	65	2	16	6	--	82
Are Currently Using a Modern Method of Contraception	57	23	1	6	2	41	64
Percentage of Children Less than 2 SD Below the Mean of Height for Age	--	--	22	--	23	28	22
<b>DEMOGRAPHIC INDICATORS 2/</b>							
Infant Mortality Rates (100) x 1,000	76	76	75	144	86	25	35
Child Mortality Rates (401) x 1,000	11	--	83	89	114	10	10
Total Fertility Rates	3.7	4.1	6.8	6.3	6.6	4.8	2.4
<b>SOCIO-ECONOMIC INDICATORS</b>							
Percentage of Women in Reproductive Ages who:							
Have had Primary Education	41	38	18	18	14	30	79
Reside in Urban Areas	76	68	4	43	41	16	18
Have No Education	7	11	80	63	77	11	10

-- Data not published or not available.

1/ Modern methods of contraceptions are: sterilization, pills, condoms, injections and vaginal methods.

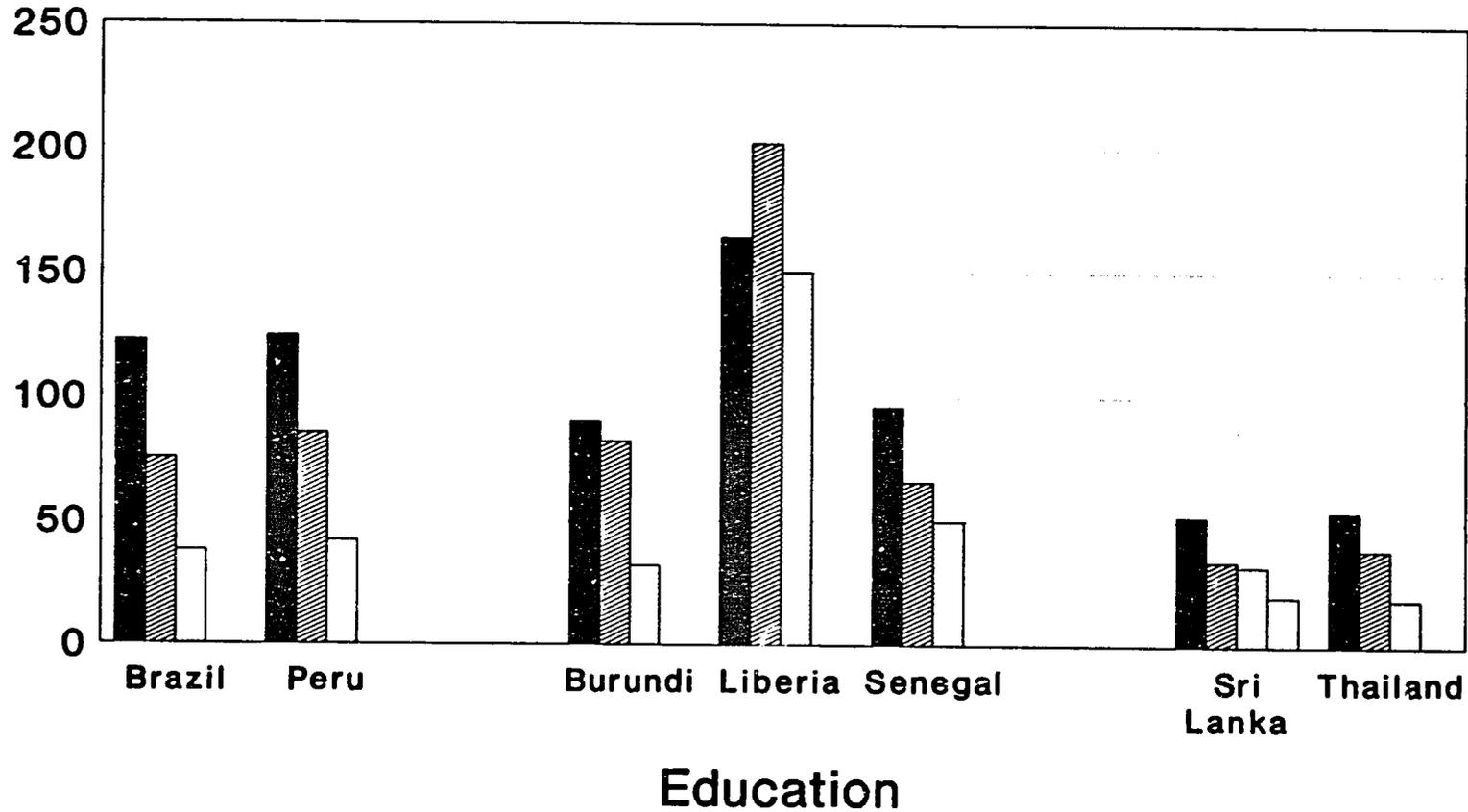
2/ These rates are estimated for the five-year period prior to the survey.

Source: Demographic and Health Surveys

47

Figure 1.

# INFANT MORTALITY BY EDUCATION



**Legend:**

- No Education** (Solid black)
- Primary** (Diagonal lines)
- Secondary** (White with black outline)
- Higher** (White with black outline)

**Brazil:**

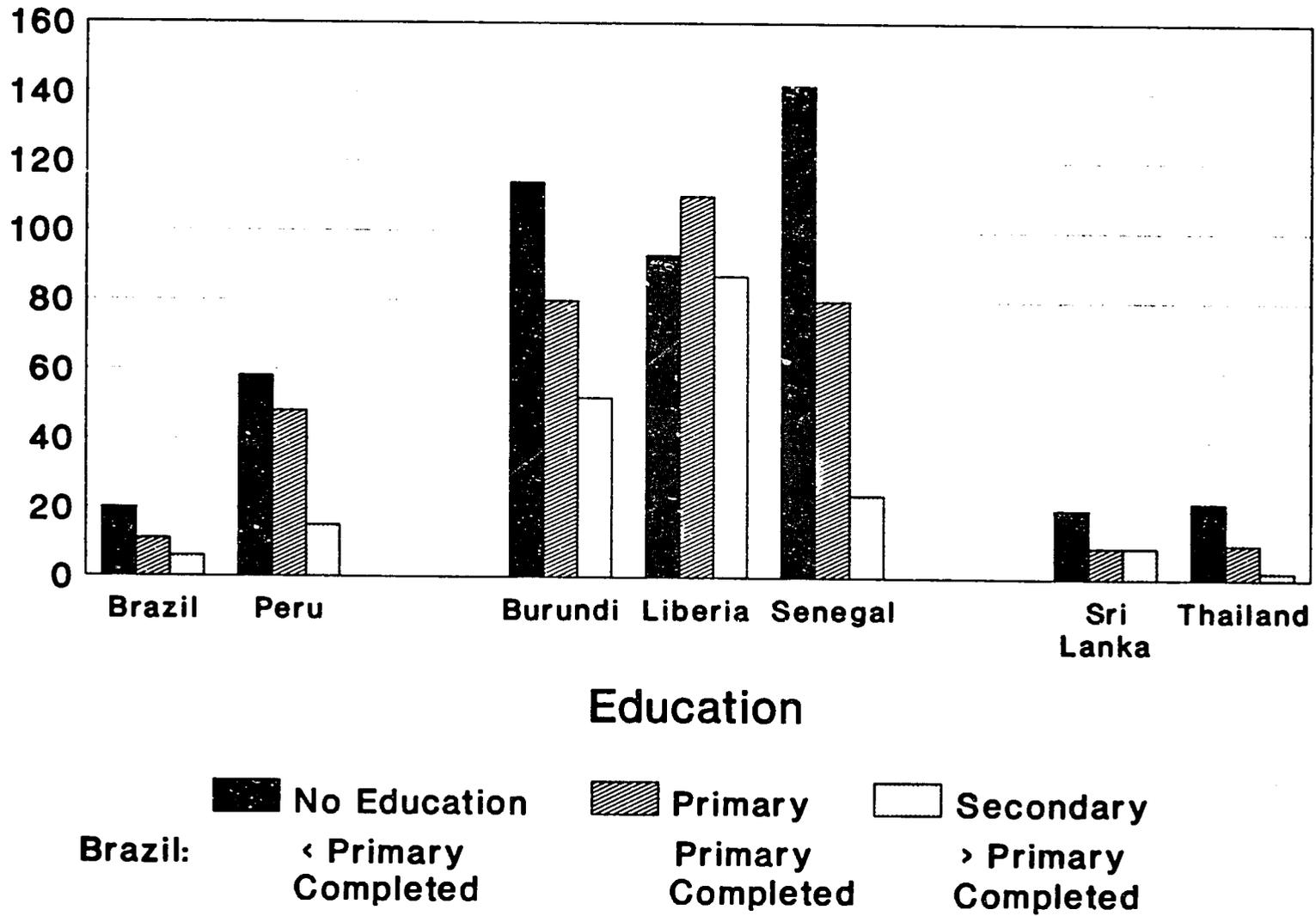
- ◀ Primary Completed
- Primary Completed
- ▶ Primary Completed

Demographic and Health Surveys

84

Figure 2.

## CHILD MORTALITY BY EDUCATION

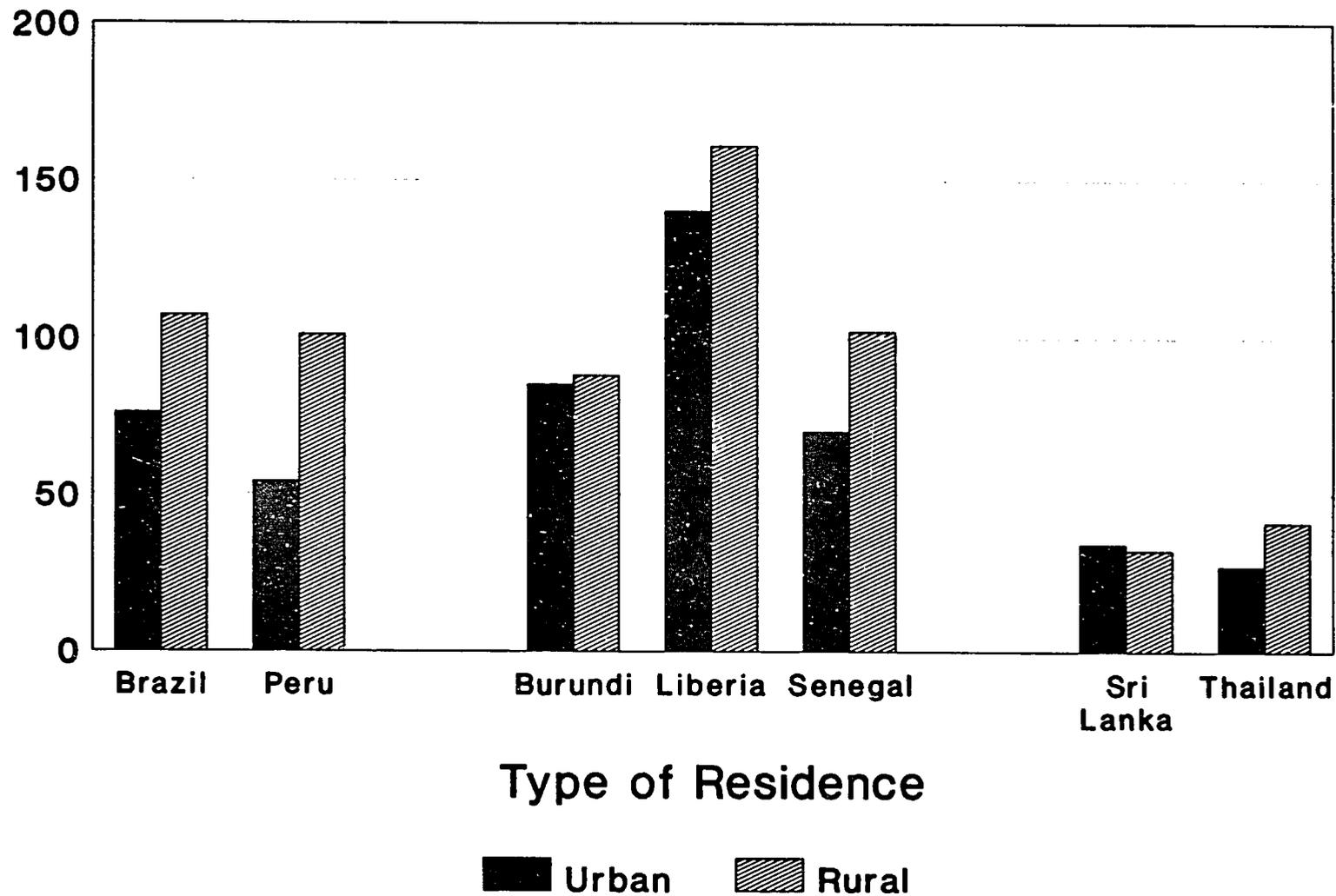


Demographic and Health Surveys

29

Figure 3.

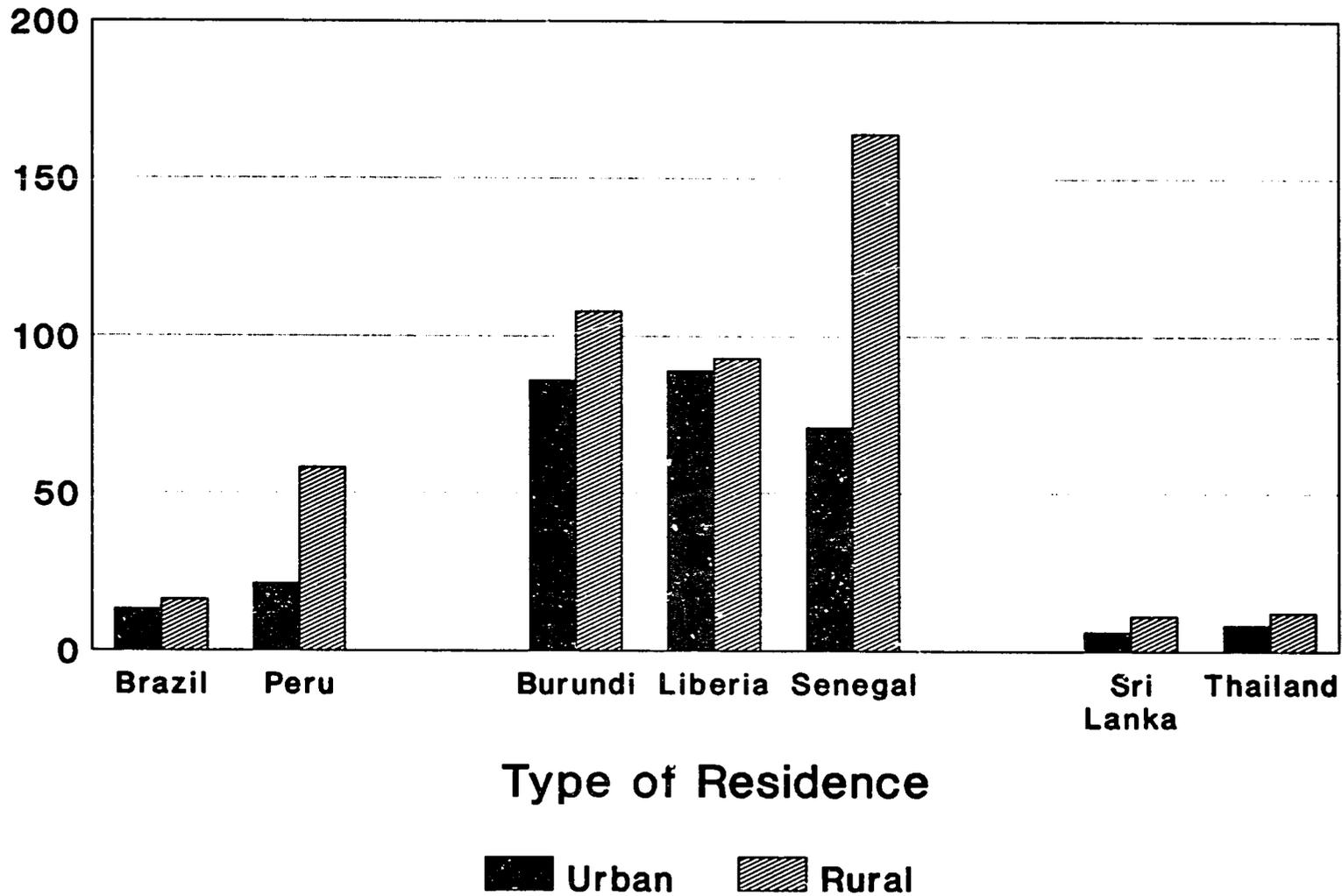
## INFANT MORTALITY BY RESIDENCE



5

Figure 4.

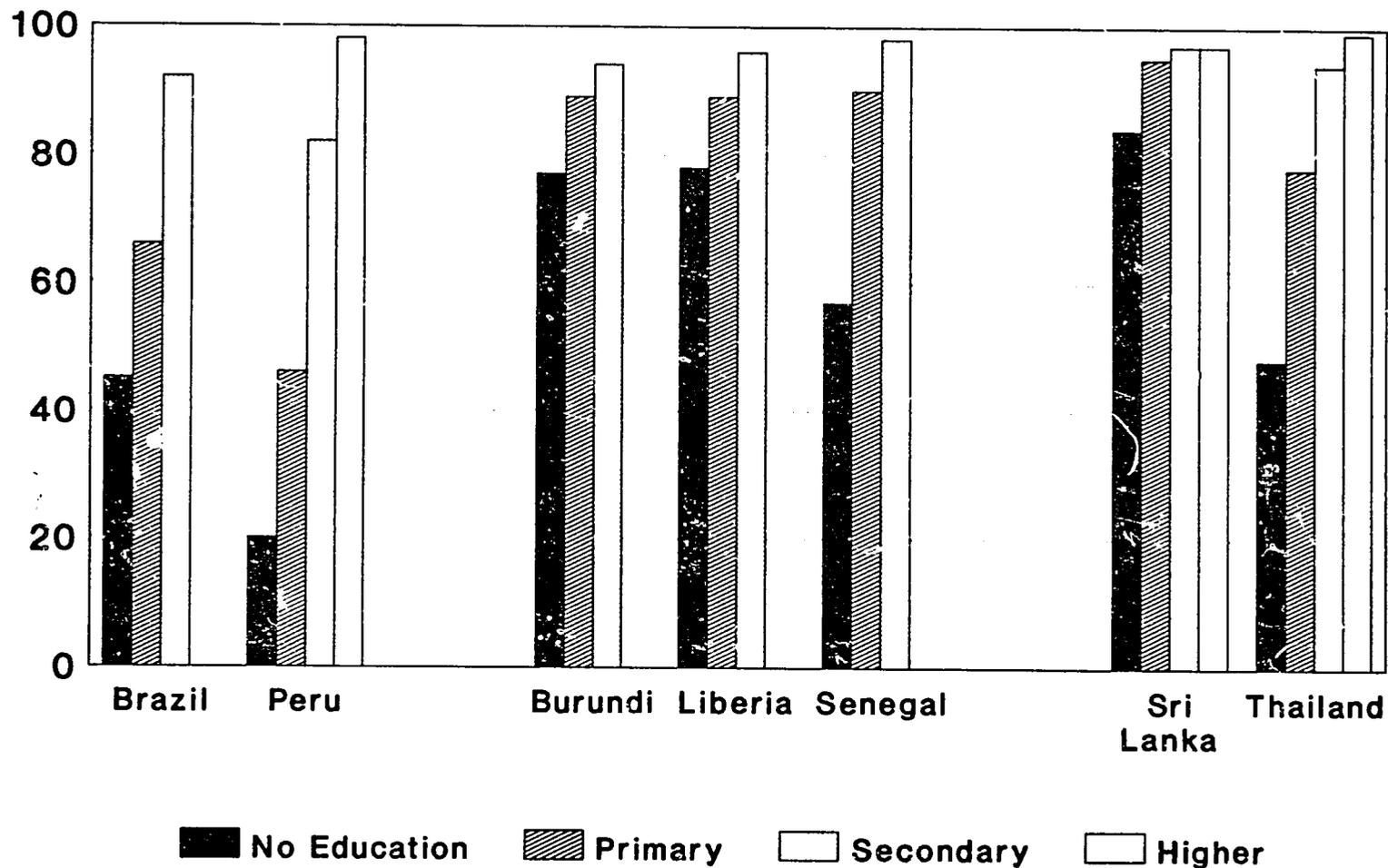
### CHILD MORTALITY BY RESIDENCE



Demographic and Health Surveys

Figure 5.

# PERCENTAGE THAT RECEIVED PRENATAL CARE BY TRAINED PERSON, BY EDUCATION

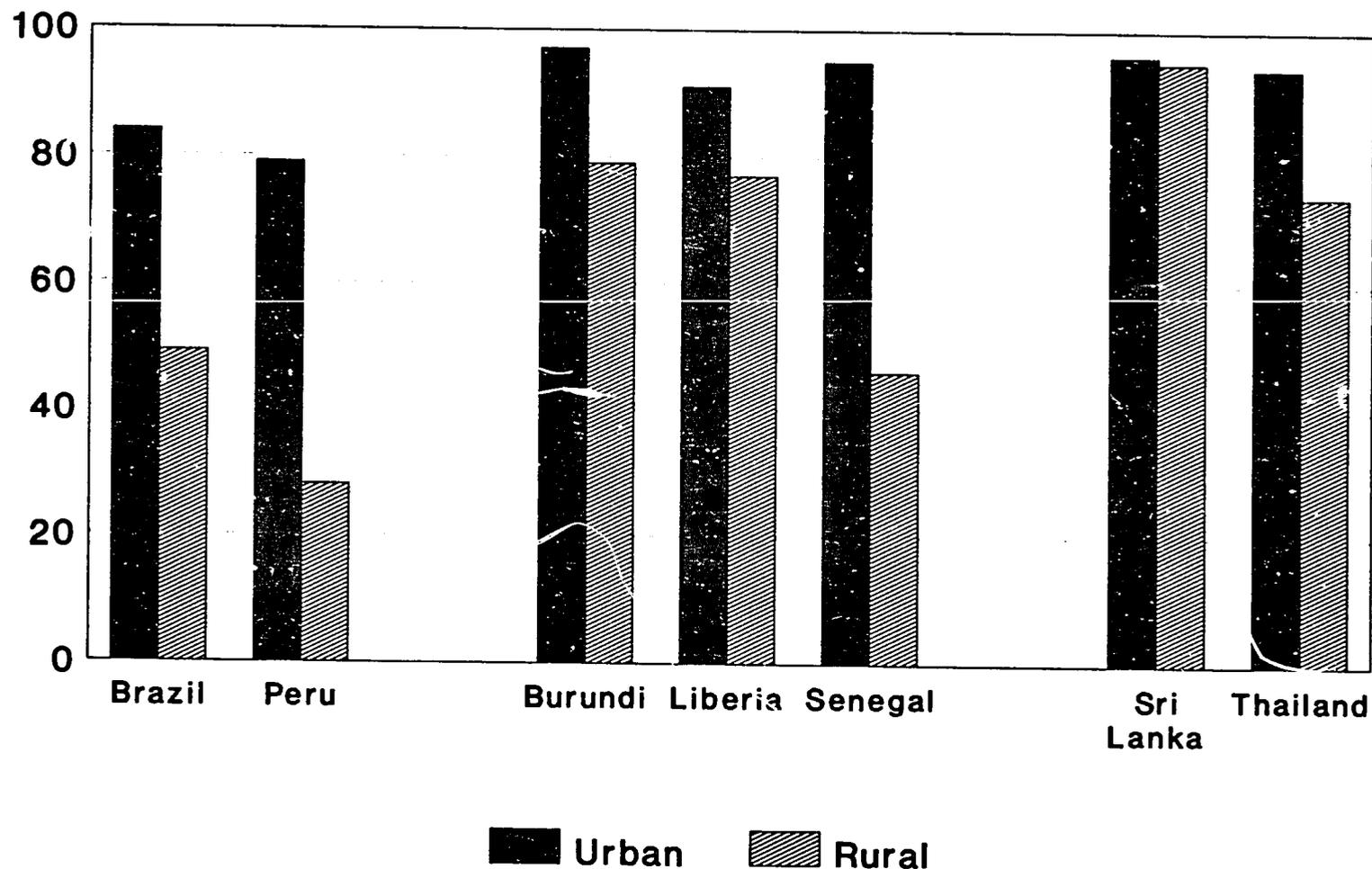


Demographic and Health Surveys

8

Figure 6.

# PERCENTAGE THAT RECEIVED PRENATAL CARE BY TRAINED PERSON, BY RESIDENCE

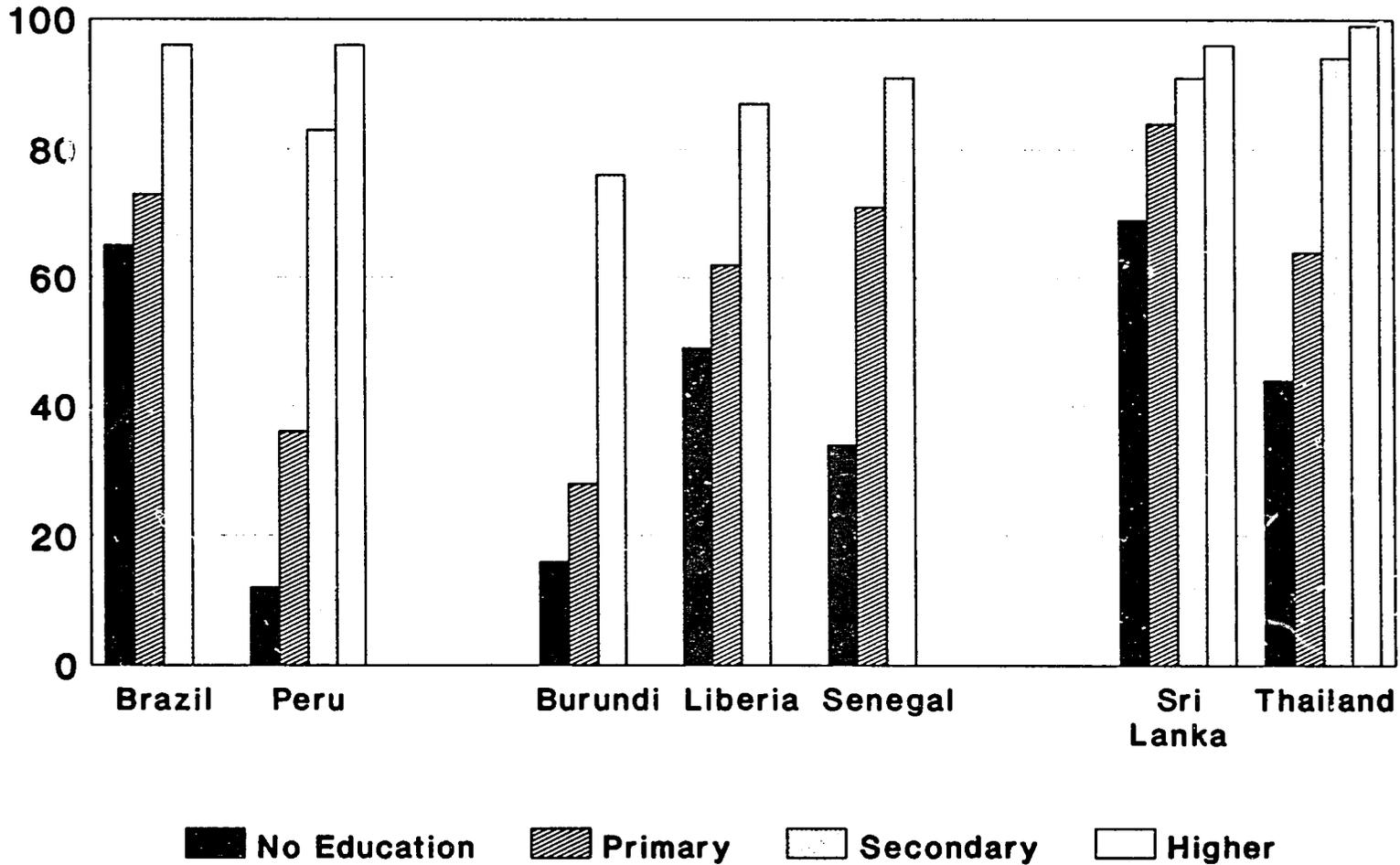


Demographic and Health Surveys

53

Figure 7.

# PERCENTAGE OF BIRTHS DELIVERED BY A TRAINED PERSON, BY EDUCATION

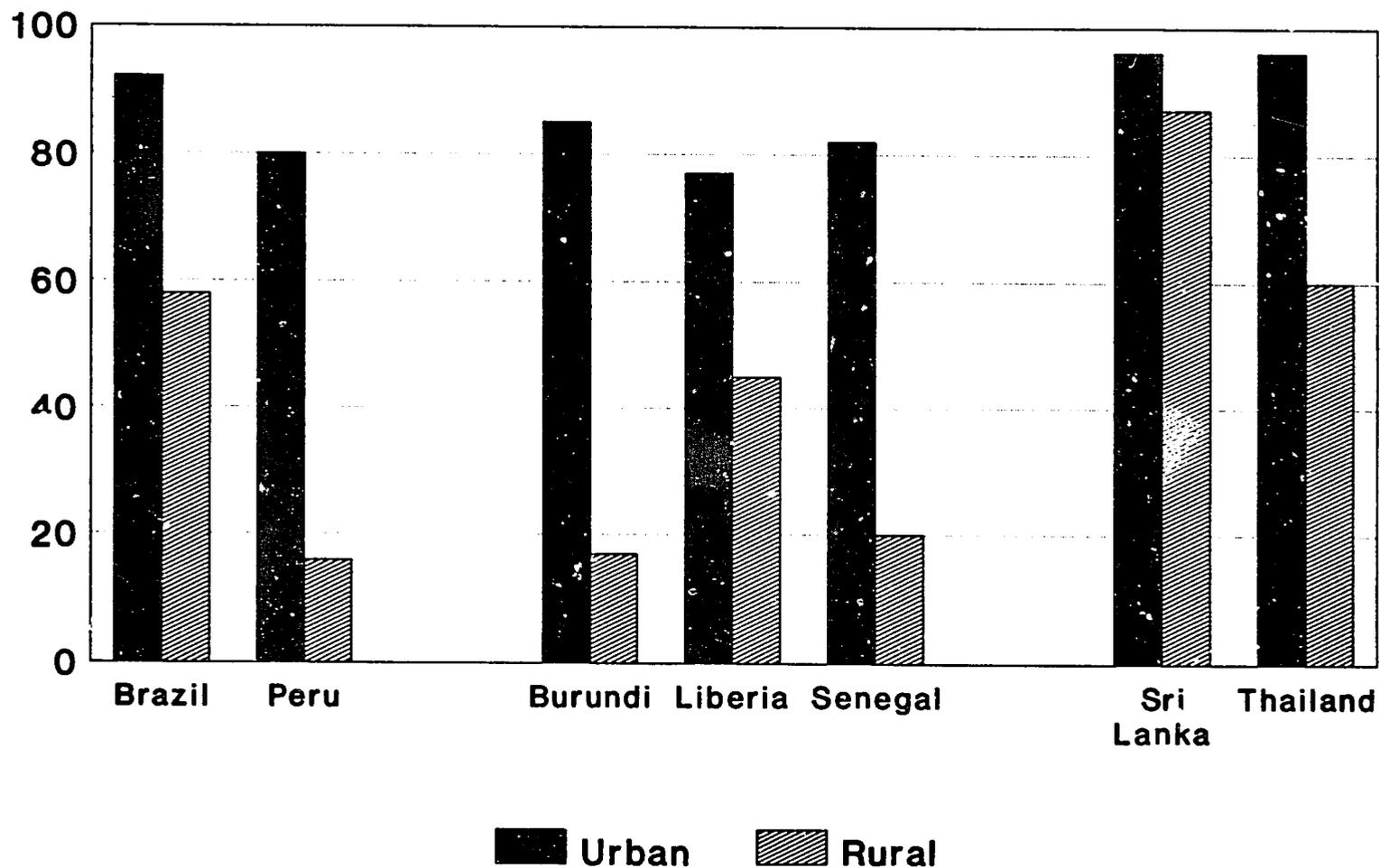


Demographic and Health Surveys

94

Figure 8.

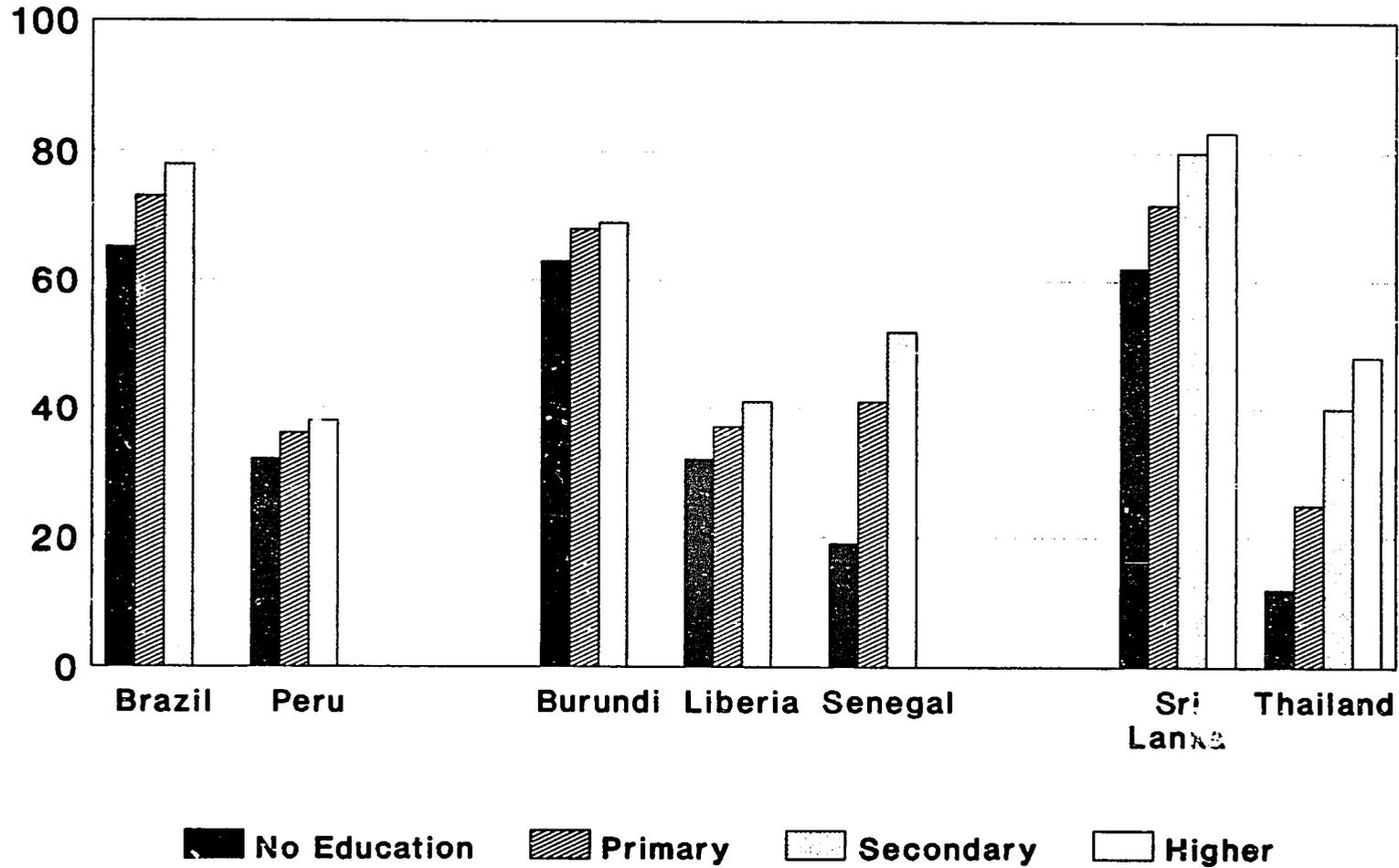
# PERCENTAGE OF BIRTHS DELIVERED BY A TRAINED PERSON, BY RESIDENCE



53.

Figure 9.

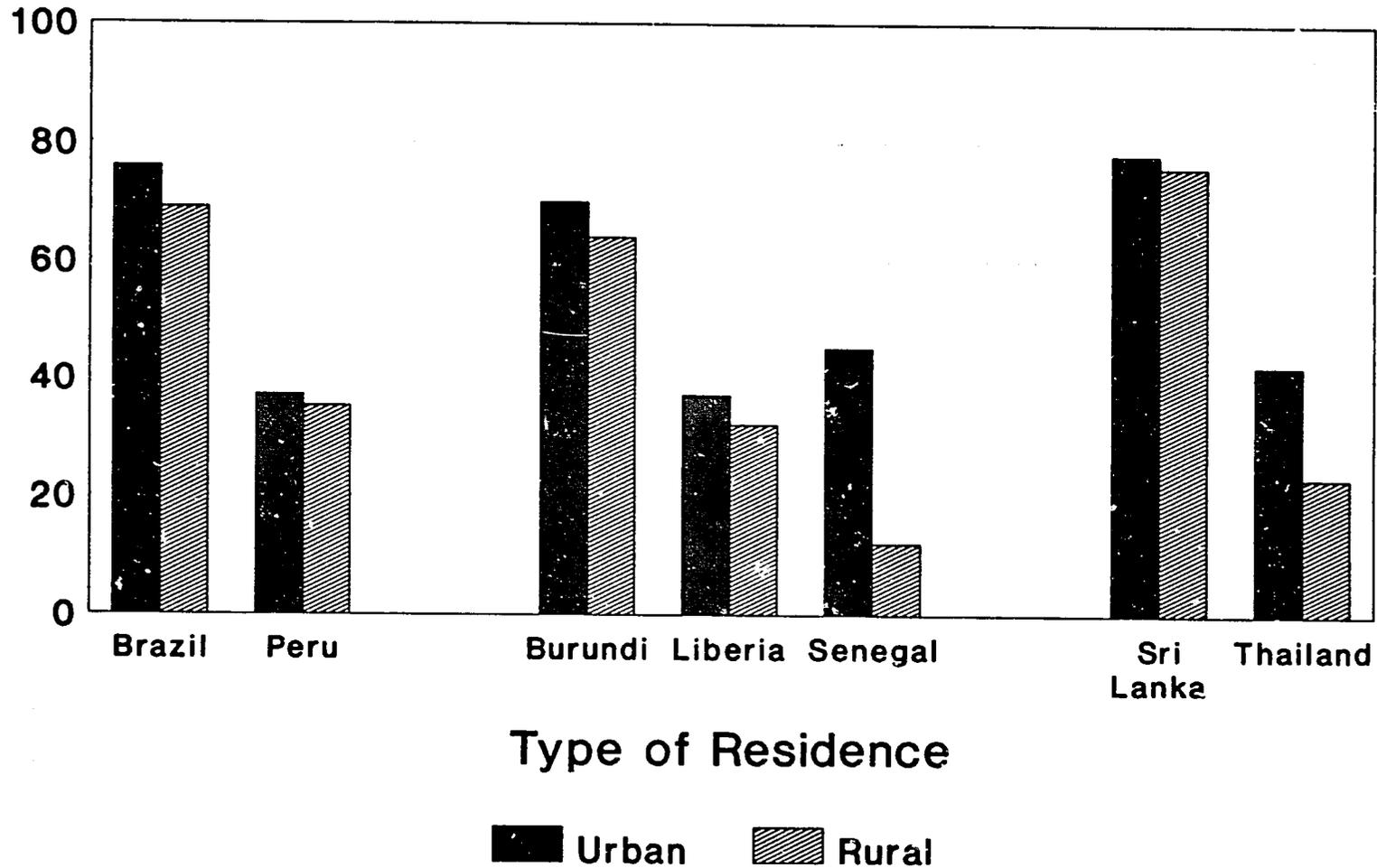
## PERCENTAGE OF CHILDREN WITH HEALTH CARDS, BY EDUCATION



Demographic and Health Surveys

Figure 10.

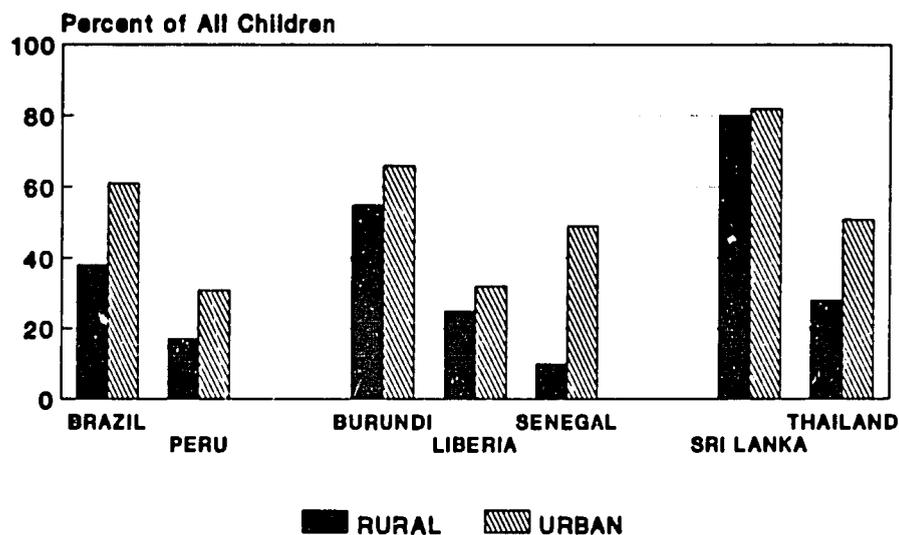
## PERCENTAGE OF CHILDREN WITH HEALTH CARDS, BY RESIDENCE



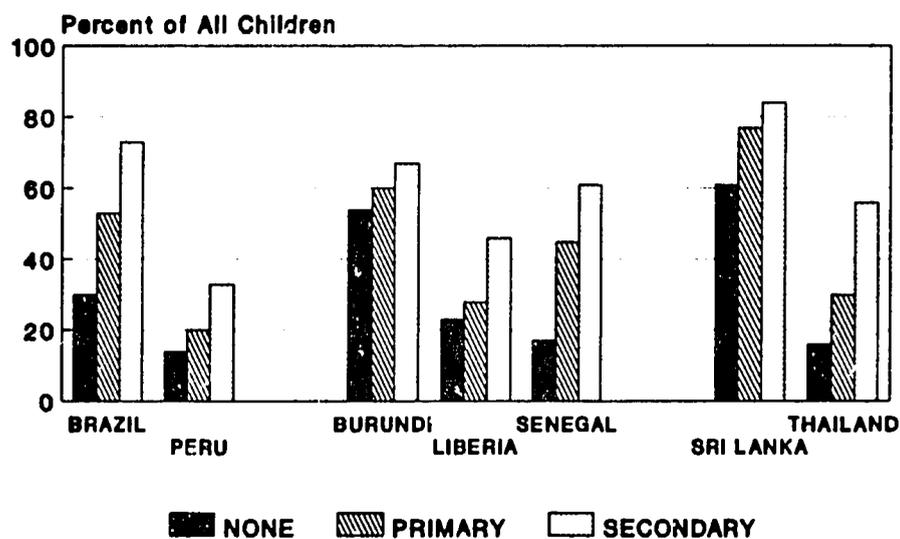
**Figure 11.**

**CHILDREN (12-23 MONTHS OLD) GIVEN BCG  
IN THE FIRST YEAR OF LIFE ACCORDING TO  
A HEALTH CARD**

**BY RESIDENCE**



**BY MOTHER'S EDUCATION**

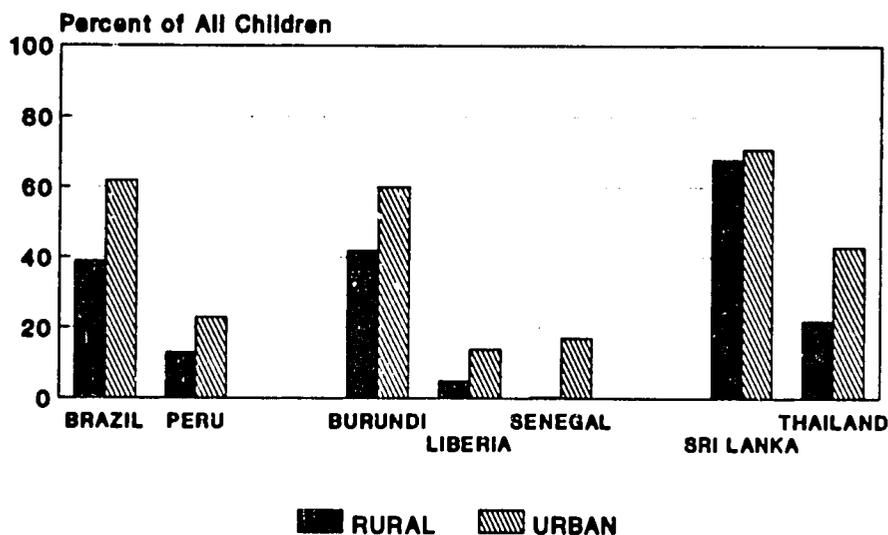


**Demographic and Health Surveys 1986-87**

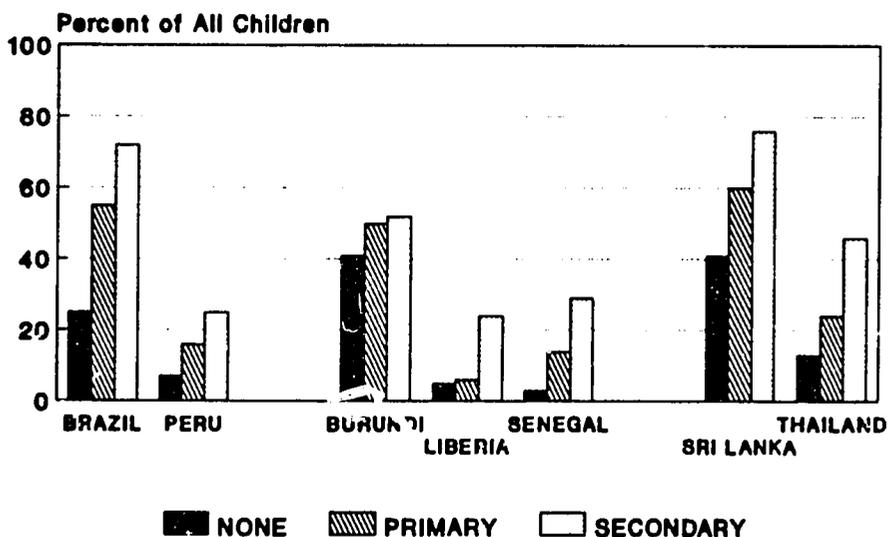
Figure 12.

CHILDREN (12-23 MONTHS OLD) GIVEN POLIO 3  
IN THE FIRST YEAR OF LIFE ACCORDING TO  
A HEALTH CARD

BY RESIDENCE



BY MOTHER'S EDUCATION



Demographic and Health Surveys 1986-87

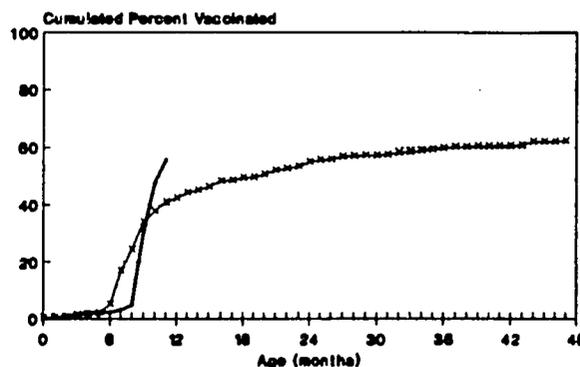
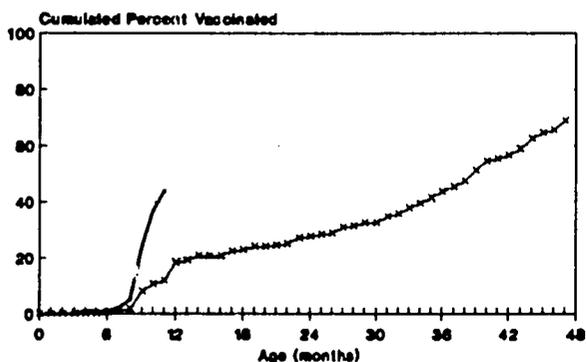
59

Figure 13.

CHILDREN WITH A HEALTH CARD GIVEN  
MEASLES VACCINE BY AGE COHORT

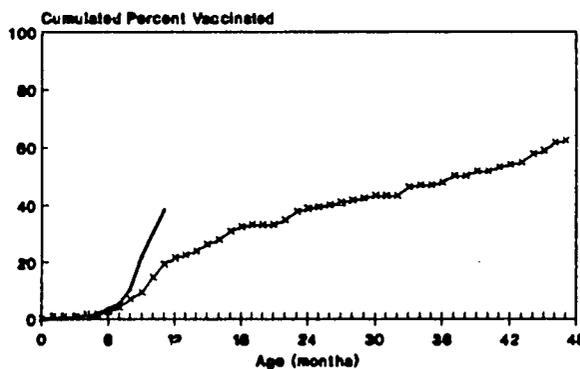
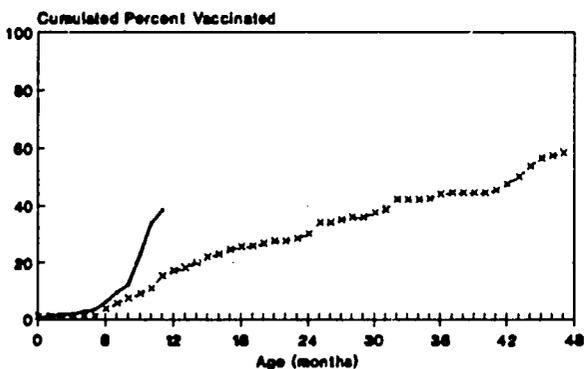
PERU 1986

BRAZIL 1986



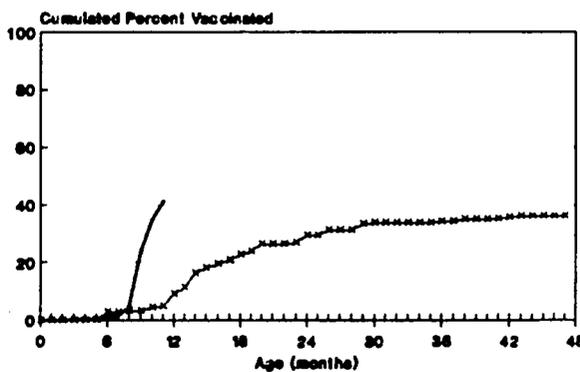
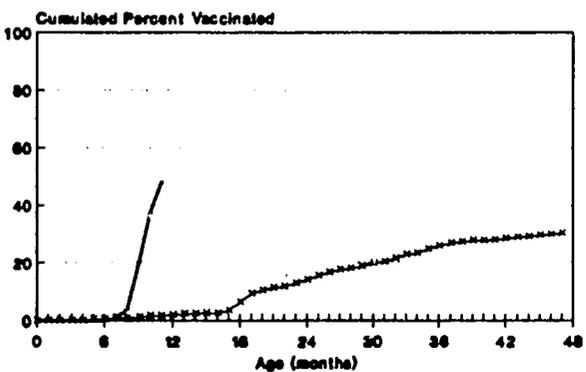
LIBERIA 1986

SENEGAL 1986

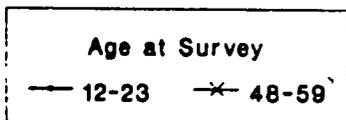


SRI LANKA 1987

THAILAND 1987



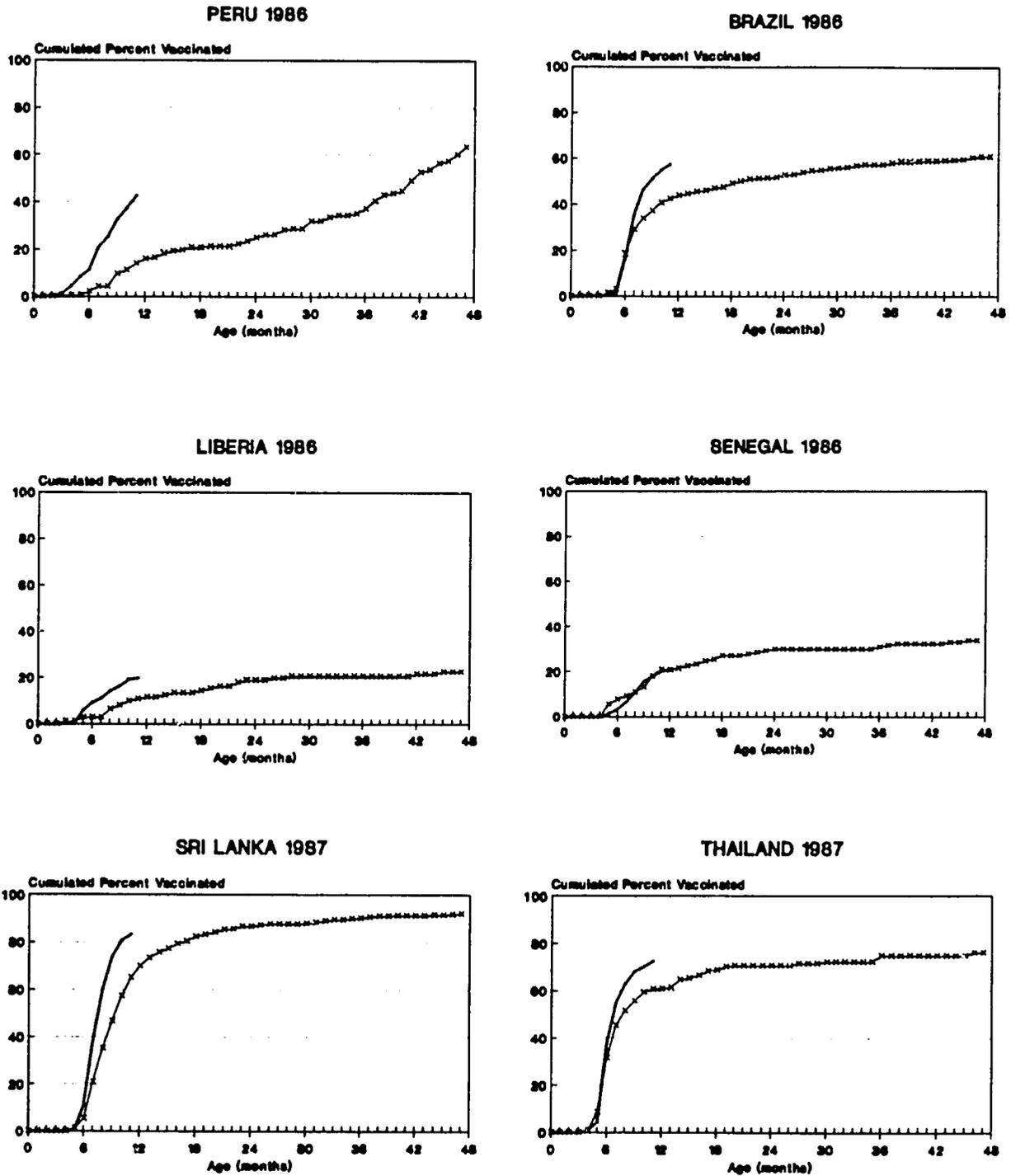
Demographic and Health Surveys



69

Figure 14.

CHILDREN WITH A HEALTH CARD  
GIVEN POLIO 3 BY AGE COHORT



Demographic and Health Surveys

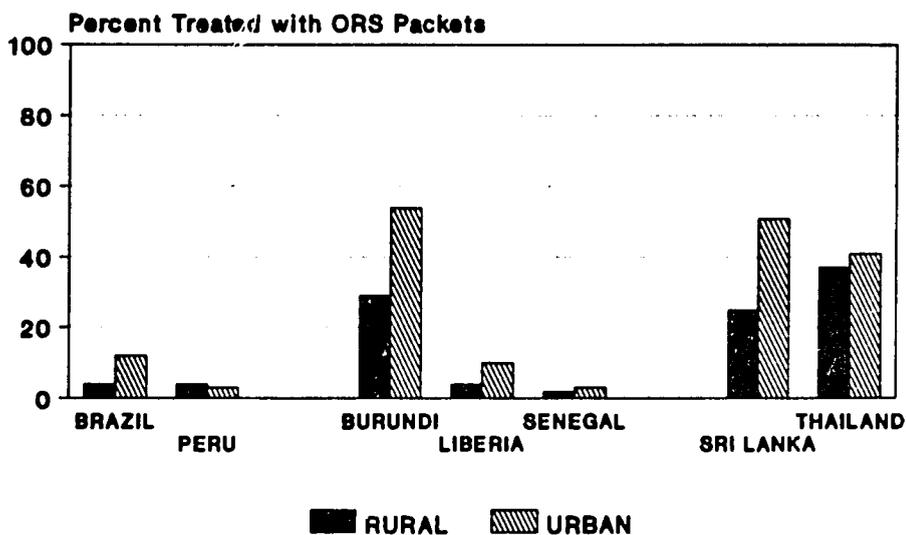
Age at Survey  
— 12-23    × 48-59

61

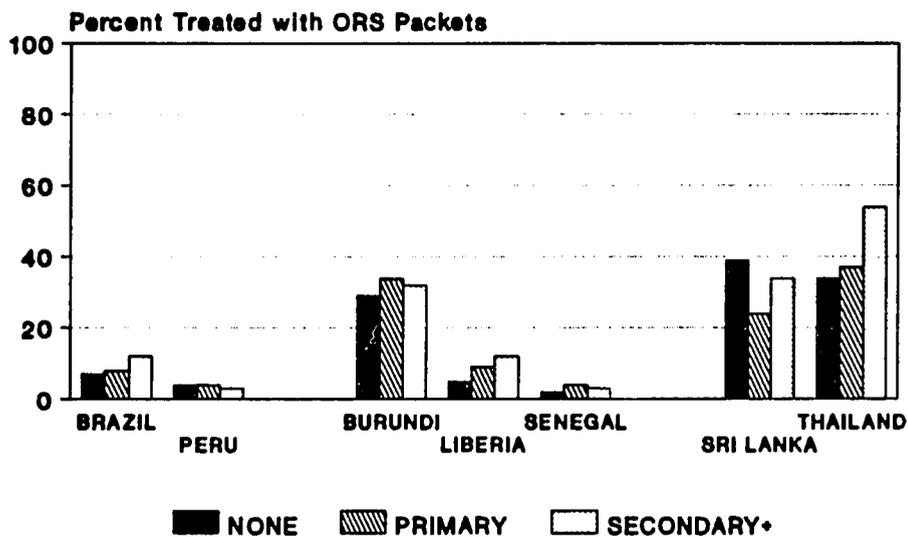
Figure 15.

CHILDREN (0-59 MONTHS OLD)  
WITH DIARRHEA IN THE LAST 2 WEEKS  
TREATED WITH ORS PACKETS

BY RESIDENCE



BY MOTHER'S EDUCATION

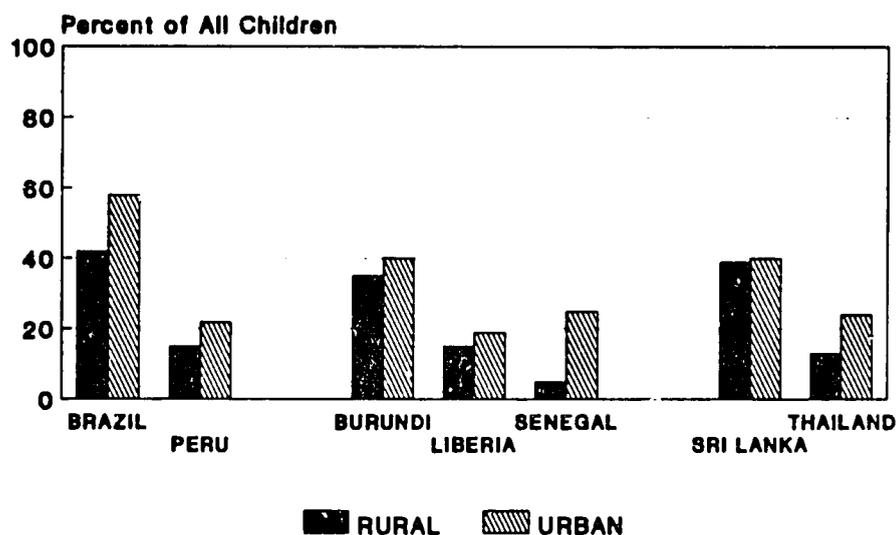


625

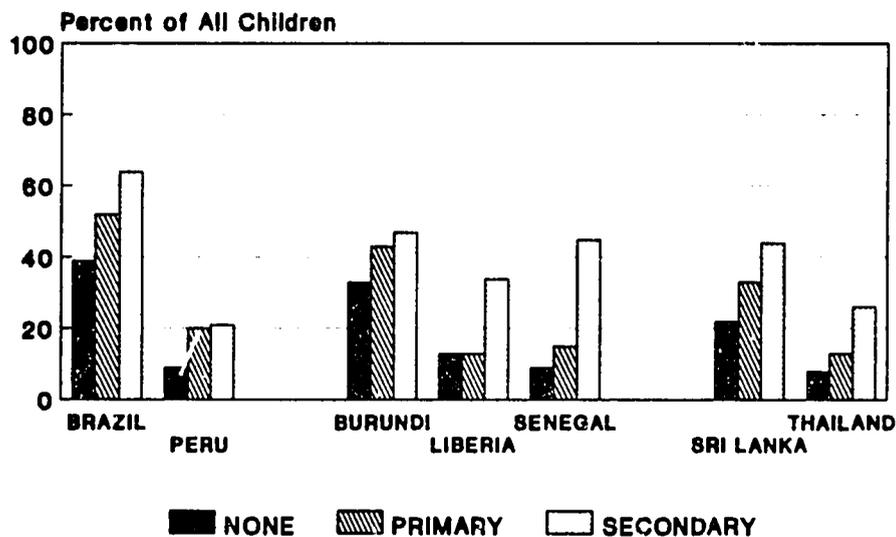
Figure 16.

CHILDREN (12-23 MO. OLD) GIVEN MEASLES VACCINE  
IN THE FIRST YEAR OF LIFE ACCORDING TO  
A HEALTH CARD

BY RESIDENCE



BY MOTHER'S EDUCATION



Demographic and Health Surveys 1986-87

Table 2.

MATERNAL AND CHILD HEALTH SERVICE USE INDEX

For Mothers of Living Children 12 to 59 months old

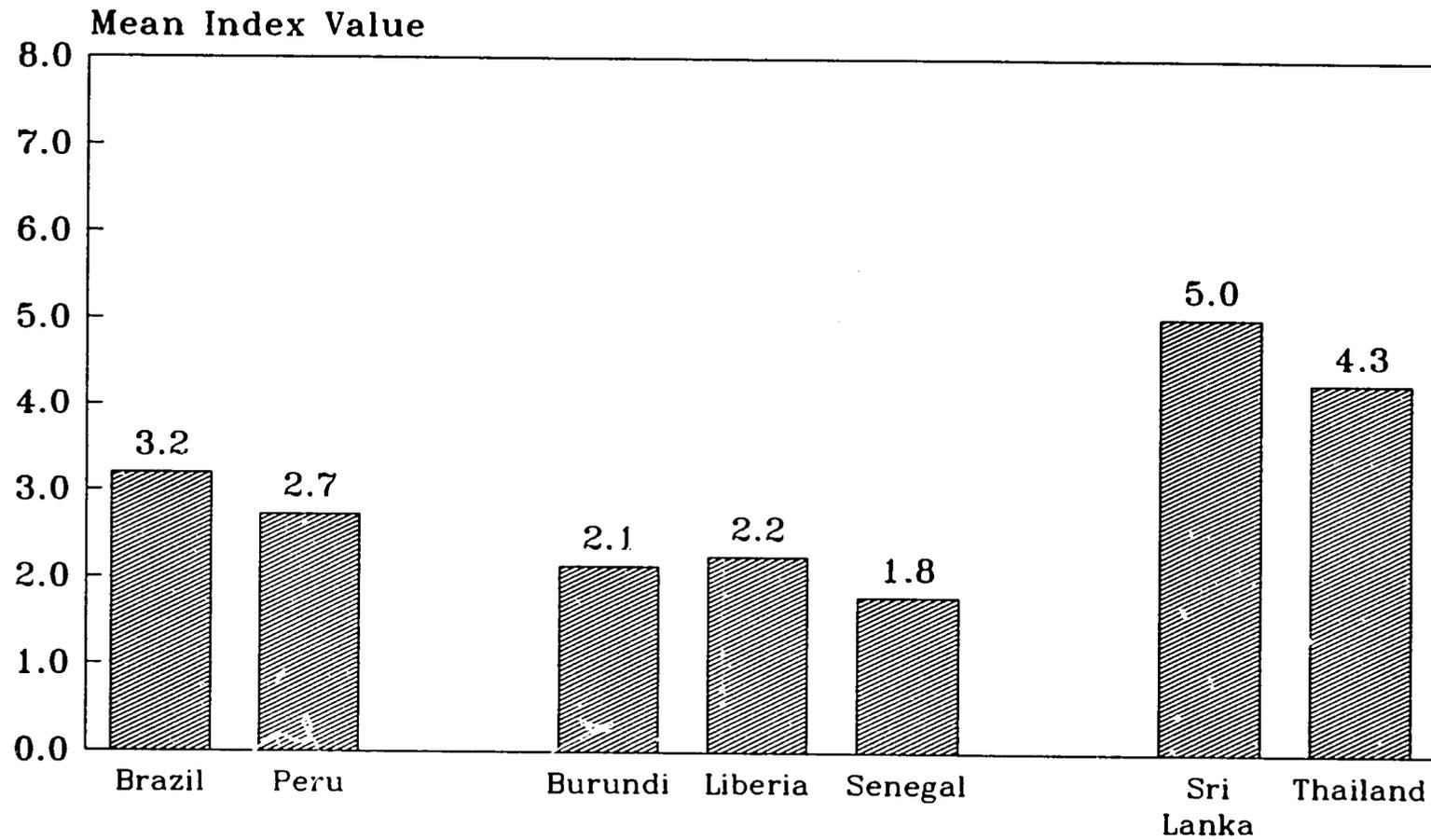
<u>Indicator of Use</u> <u>For Each Child:</u>	<u>Point Value in Index</u> <u>Maximum for any child 12 to 59:</u>
Prenatal Care	1 point if by trained person
Delivery Care	1 point if by trained person
Prenatal Tetanus Toxoid	1 point if received
Health Card	1 point if seen by interviewer
Mother's report of vaccination	1 point if reported any
	<u>For each child with disease:</u>
Diarrhea treatment and Oral Rehydration Therapy	1 point if seen by trained per. 1 point if used in last episode (-1/2 point if did neither)
	<u>Plus point value for mother:</u>
Ever Use of ORT	1 point if ever used
Ever Use of Family Planning	1 point for Pill, IUD, Norplant Injection or Sterilization 1/2 point for condom, vaginals

Index Range: -0.5 to 8.0

194

Figure 17.

VALUE OF HEALTH SERVICES USAGE INDEX  
BY COUNTRY



6

**Table 3.**

**REGRESSION ANALYSIS OF HEALTH SERVICE USE**  
(Multiple Classification Analysis)

Dependent Variable: Index of Health Service Usage

Independent Variables: Type of Area of Residence  
Age of Mother at Interview  
Parity at Interview  
Marital Status at Interview

MCA No. 1: Level of Education

MCA No. 2: Literacy

<u>General Results</u>	<u>Cases</u>	<u>R-squared</u>	
		(1)	(2)
Brazil	2003	.077	.080
Peru	1721	.477	.483
Burundi	2044	.094	.100
Liberia	2427	.154	.156
Senegal	2218	.336	.335
Sri Lanka	2450	.111	.103
Thailand	2404	.155	.184

# Table 4.

Multiple Classification Analysis of Health Service Usage  
According to Socioeconomic and Demographic Characteristics  
Based on an Index of Health Service Usage

From Seven Demographic and Health Surveys

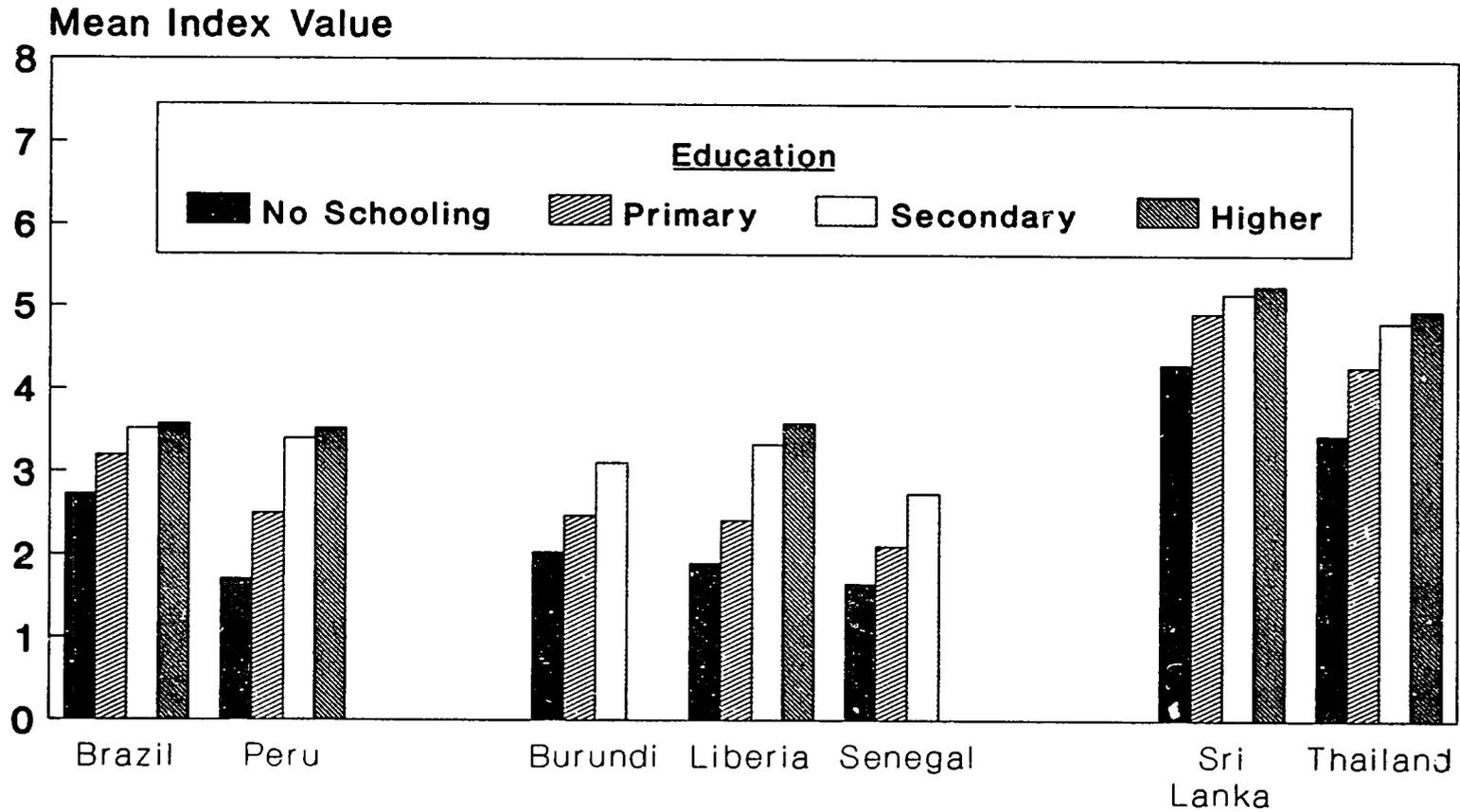
	Brazil	Peru	Burundi	Liberia	Senegal	Sri Lanka	Thailand
Total (Grand Mean)	3.20	2.72	2.13	2.24	1.78	5.03	4.28
Weighted No. of Cases	2003	1721	2043	2427	2218	2445	2401
Adjusted Mean Values for Characteristic							
Residence	**	**	**	**	**	**	**
Urban	3.33	3.42	3.35	2.44	2.72	5.25	4.68
Rural	2.89	1.84	2.09	2.10	1.28	4.99	4.20
Education	**	**	**	**	**	**	**
None	2.73	1.71	2.03	1.90	1.66	4.30	3.45
Primary	3.20	2.50	2.47	2.42	2.11	4.92	4.28
Secondary	3.52	3.40	3.11	3.33	2.74	5.16	4.82
Higher	3.57	3.52	--	3.59	--	5.26	4.96
Age of Mother	**	ns	**	**	ns	**	**
15-19	3.11	2.69	--	2.12	1.69	4.73	3.75
20-29	3.32	2.72	2.29	2.10	1.75	5.05	4.31
30-39	3.11	2.71	2.04	2.50	1.83	5.07	4.32
40-49	2.81	2.75	1.81	2.36	1.80	4.75	4.15
Marital Status	**	**	ns	ns	ns	**	**
Never Married	2.41	2.12	2.11	2.19	2.02	--	--
In Union	3.23	2.77	2.14	2.24	1.78	5.05	4.30
Formerly in union	3.14	2.34	1.97	2.28	1.55	4.51	3.80
Parity	*	ns	ns	**	ns	ns	**
< 5	3.15	2.73	2.11	2.36	1.78	5.05	4.37
5 +	3.34	2.70	2.16	2.04	1.78	4.97	3.56
R-squared	0.077	0.477	0.094	0.154	0.336	0.111	0.155
MCA with Literacy instead of Education							
Literacy	**	**	**	**	**	**	**
Reads Easily	3.36	3.21	2.62	3.31	2.57	5.16	4.61
Reads Poorly	3.10	2.25	2.32	2.48	2.04	4.82	4.12
Cannot Read	2.75	1.74	1.98	1.90	1.66	4.47	3.41
R-squared	0.080	0.483	0.100	0.156	0.335	0.103	0.184

\*\* Significant at the 1% level  
\* Significant at the 5% level  
ns Not significant at the 5% level  
-- Not applicable or Less than 25 in base

67

Figure 18.

# USE OF HEALTH SERVICES BY EDUCATION (Regression Estimates)



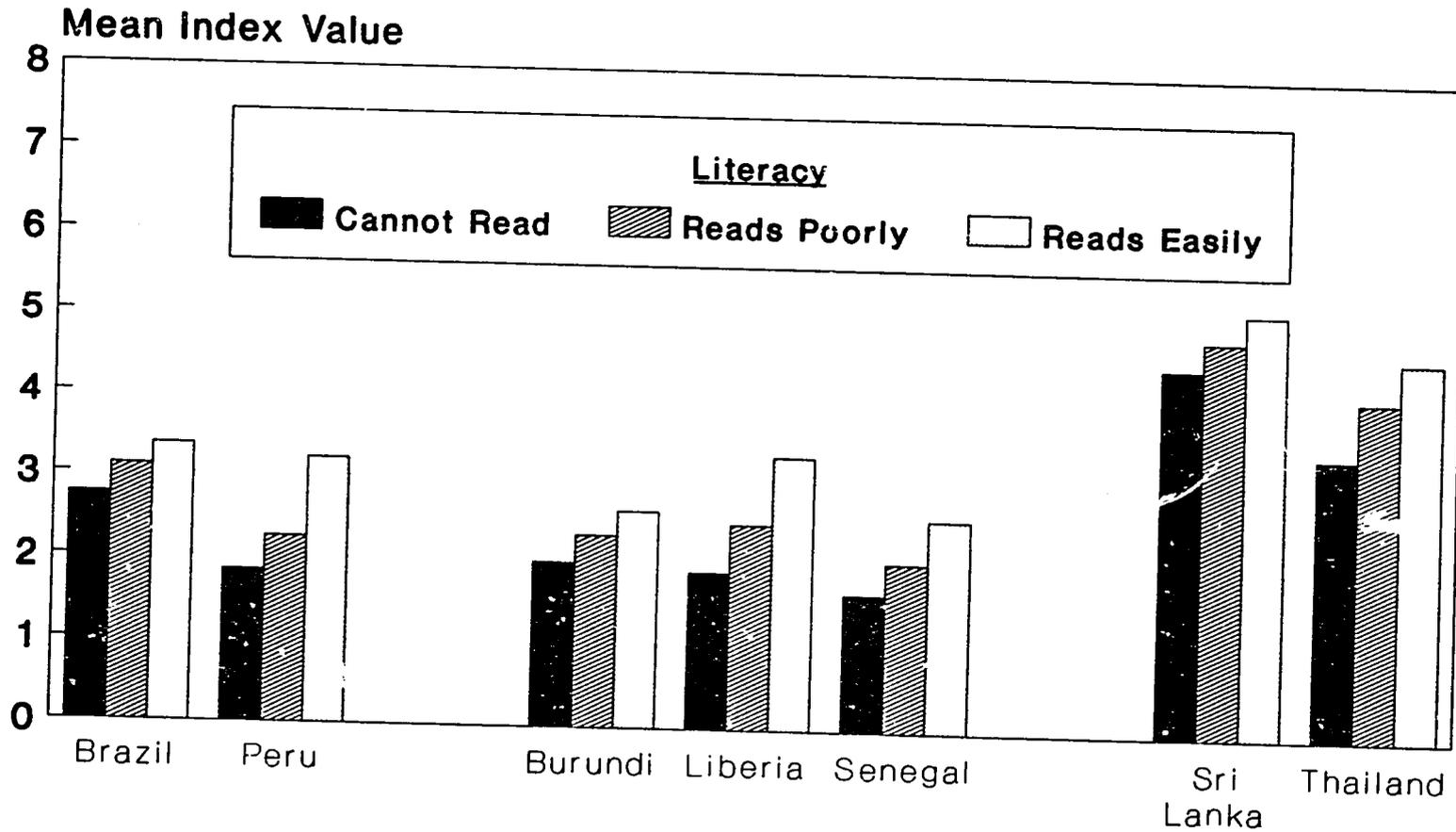
Demographic and Health Surveys

9.

Figure 19.

# USE OF HEALTH SERVICES BY LITERACY

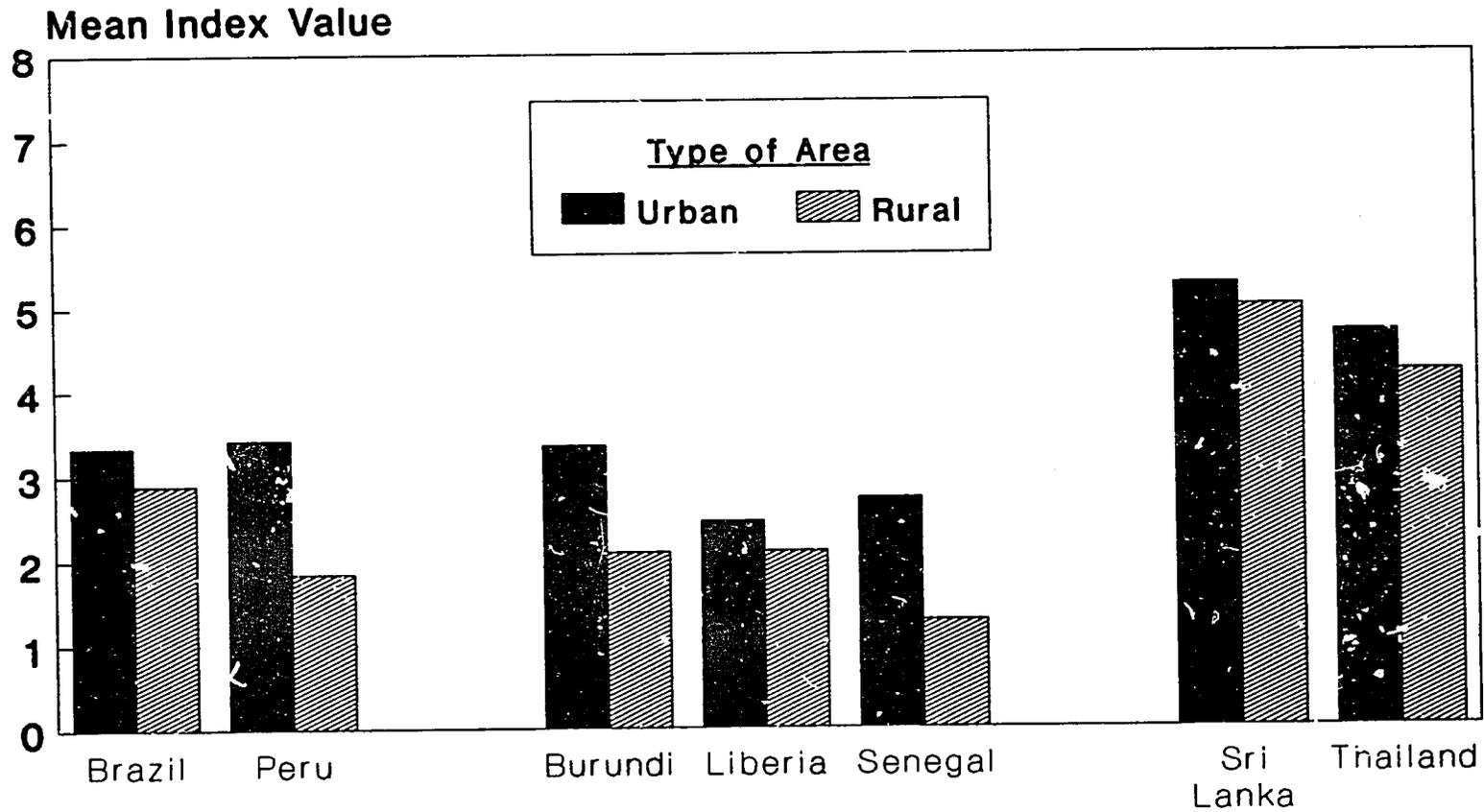
(Regression Estimates)



Demographic and Health Surveys

Figure 20.

# USE OF HEALTH SERVICES BY TYPE OF AREA OF RESIDENCE (Regression Estimates)



Demographic and Health Surveys

70

Figure 21.

# USE OF HEALTH SERVICES BY CURRENT MARITAL STATUS (Regression Estimates)

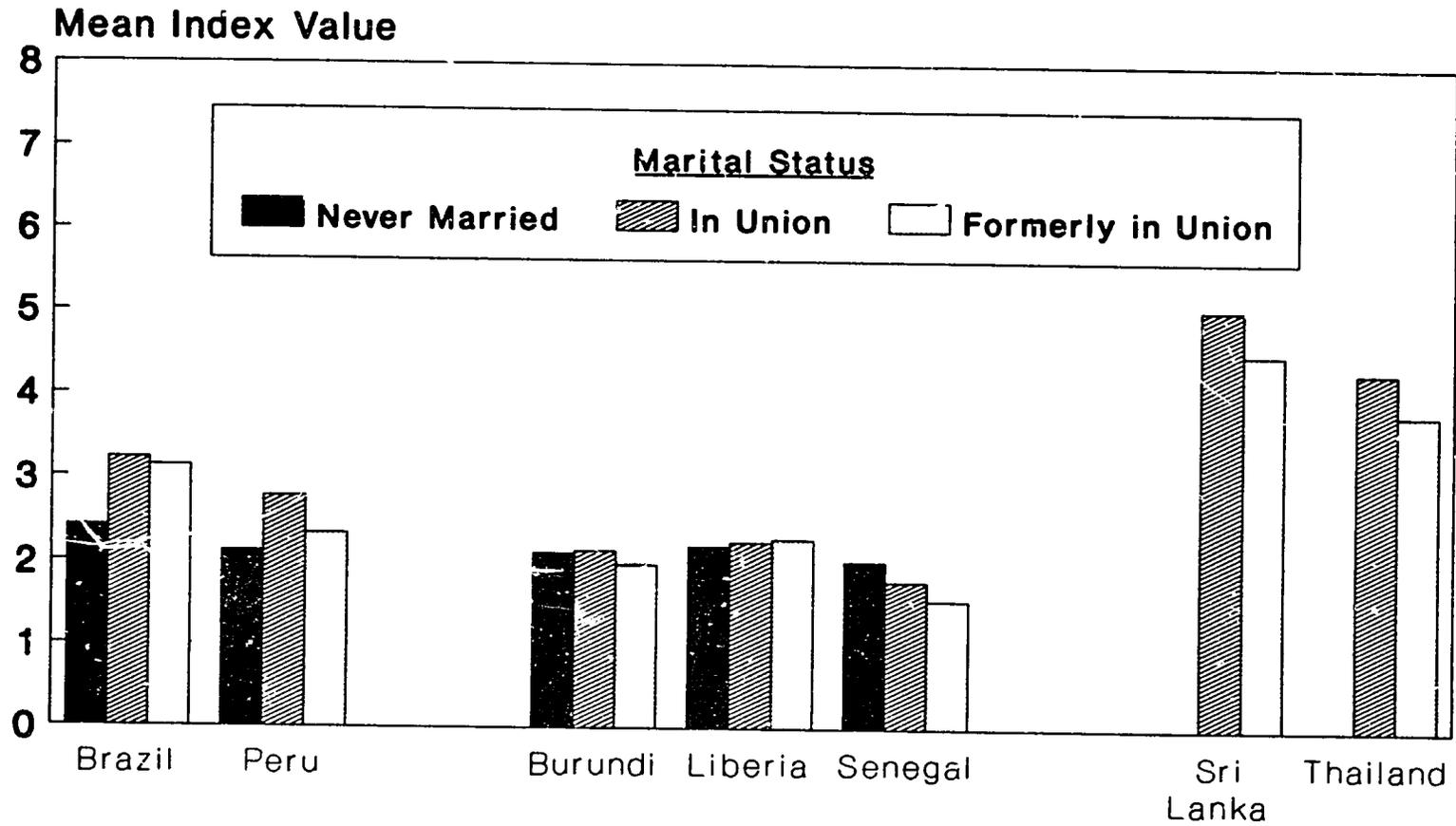
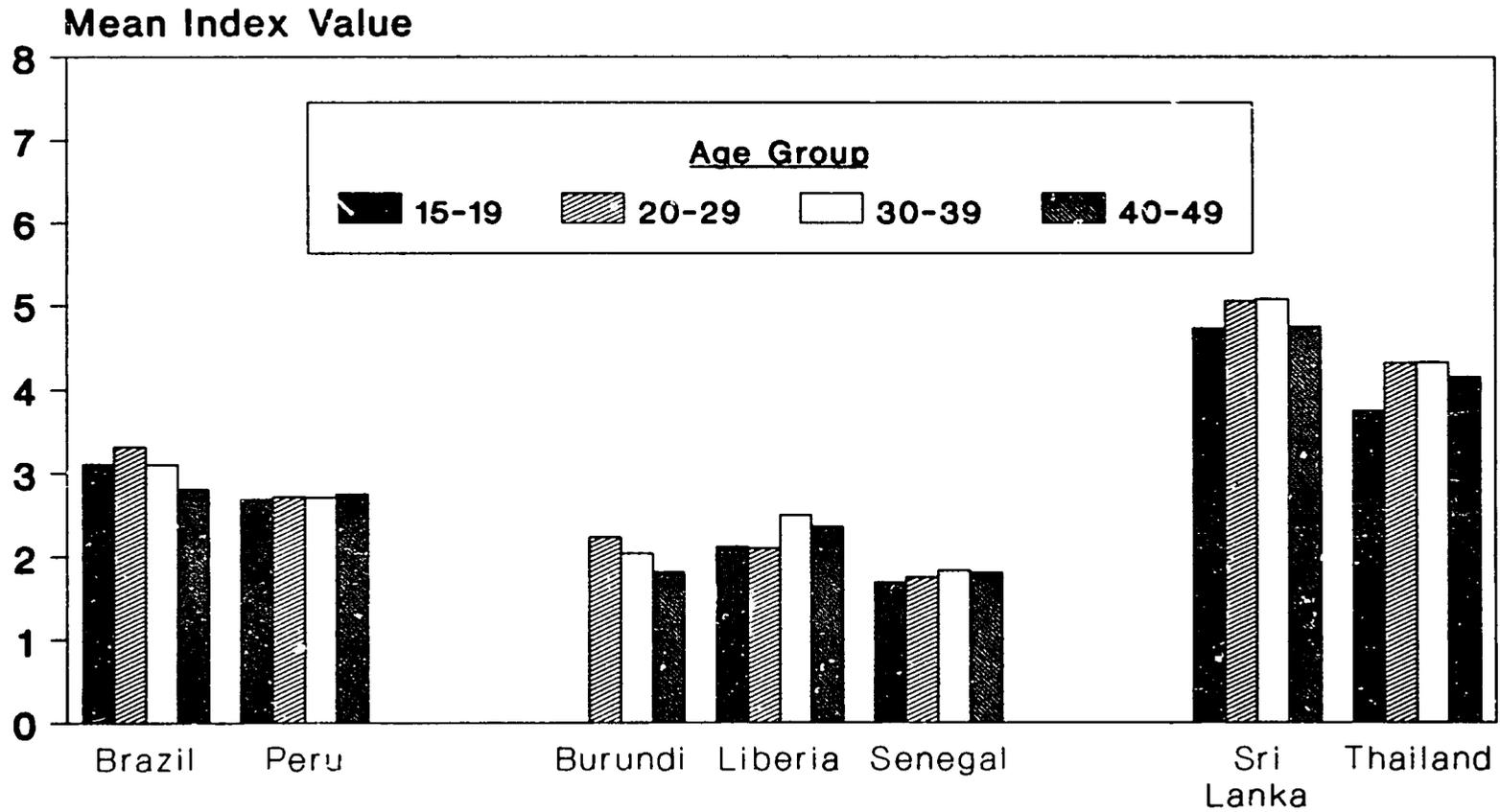


Figure 22.

# USE OF HEALTH SERVICES BY MOTHER'S AGE (Regression Estimates)

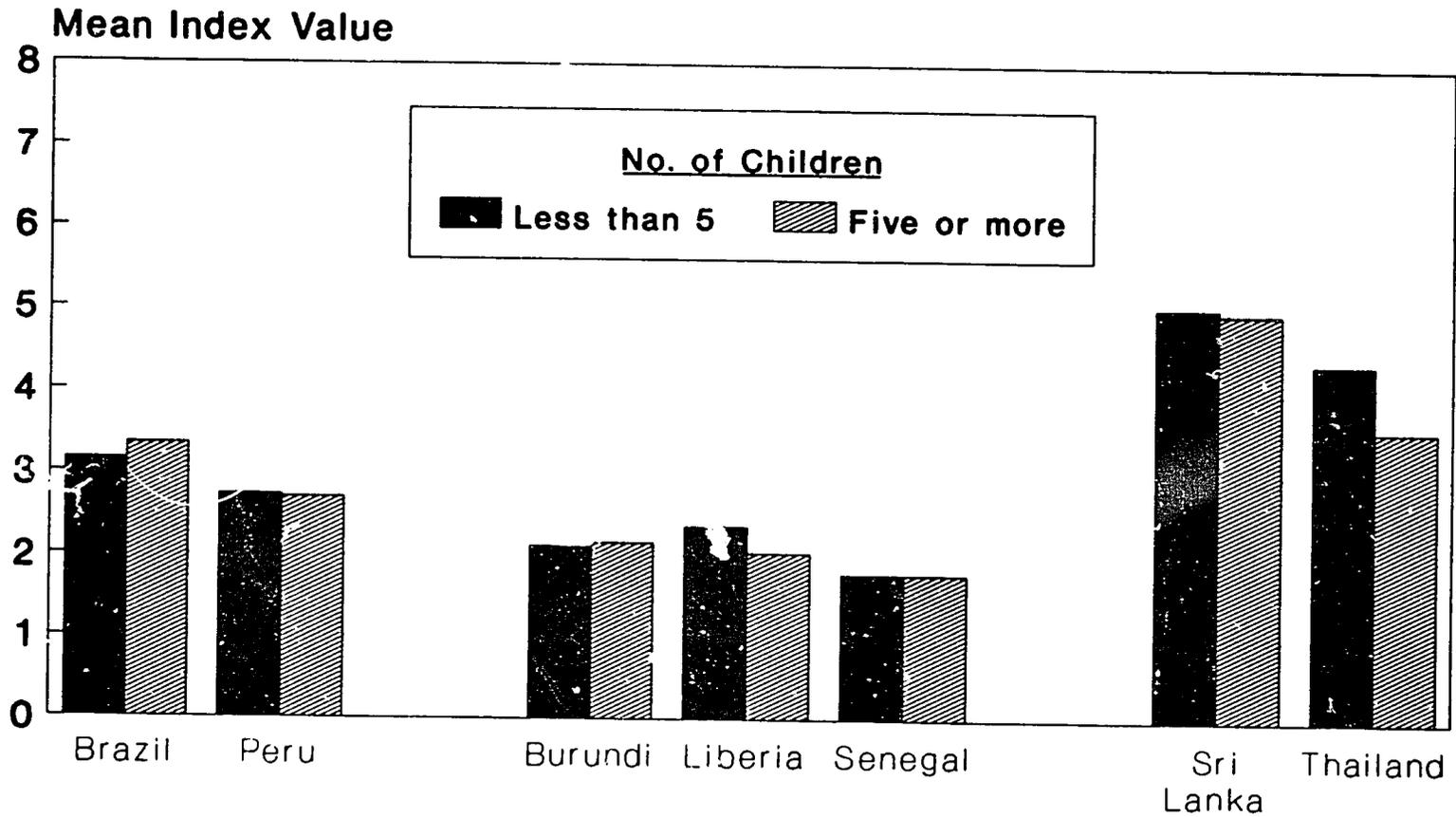


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72

Figure 23.

# USE OF HEALTH SERVICES BY PARITY (Regression Estimates)



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23

**Table 5.**  
**Socioeconomic Groups**

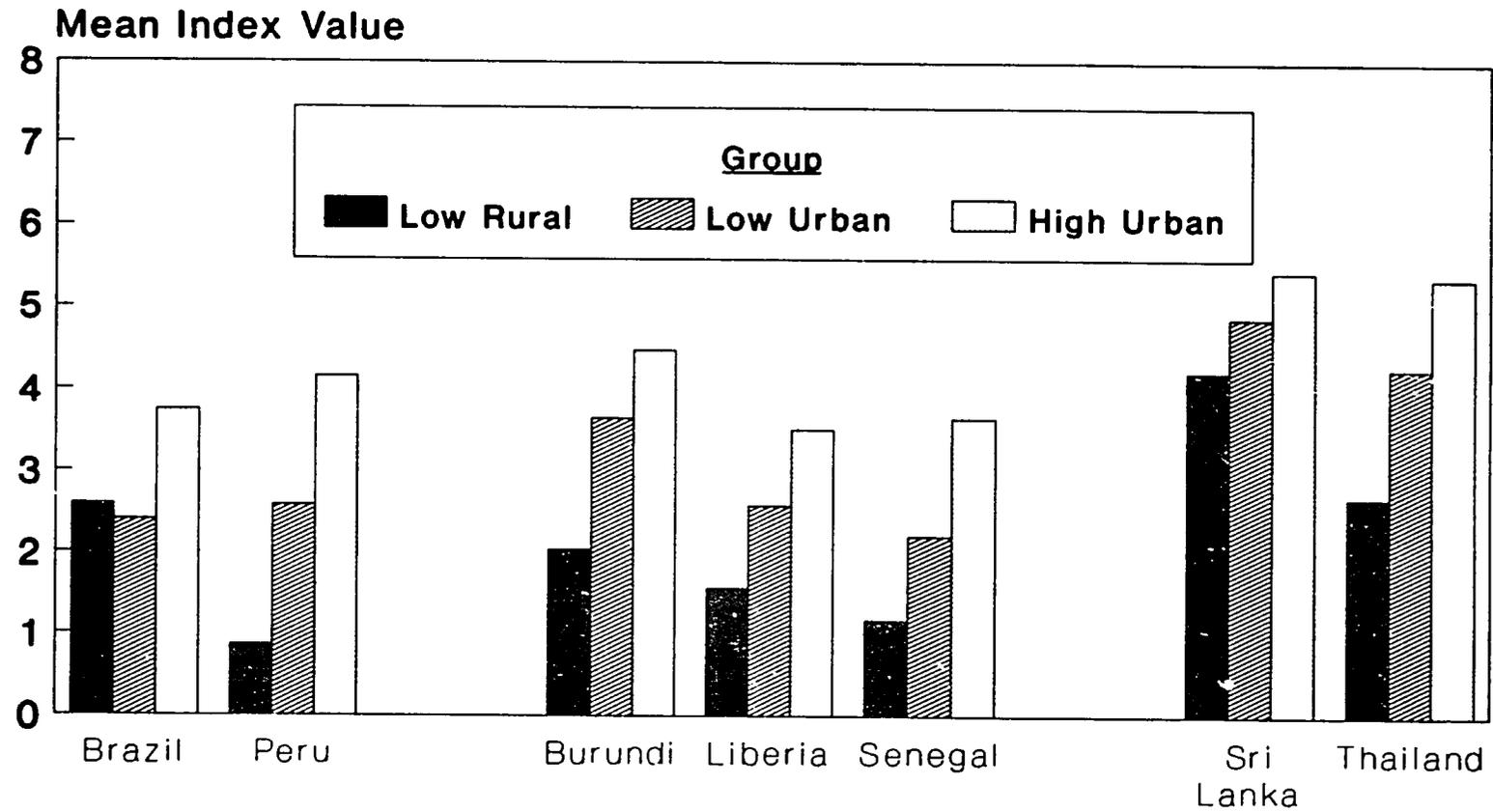
<u>Low Rural</u>	<u>Low Urban</u>	<u>High Urban</u>
Rural Residence	Urban Residence	Urban Residence
No Education	Primary Education	Secondary Education
All Ages	Under Age 20	Aged 20 to 29
In Union	Never Married*	In Union
Five or more children	Less than 5 Children	Less than 5 children

\* In Union for Sri Lanka and Thailand

74.

Figure 24.

# USE OF HEALTH SERVICES BY SOCIOECONOMIC GROUP (Regression Estimates)

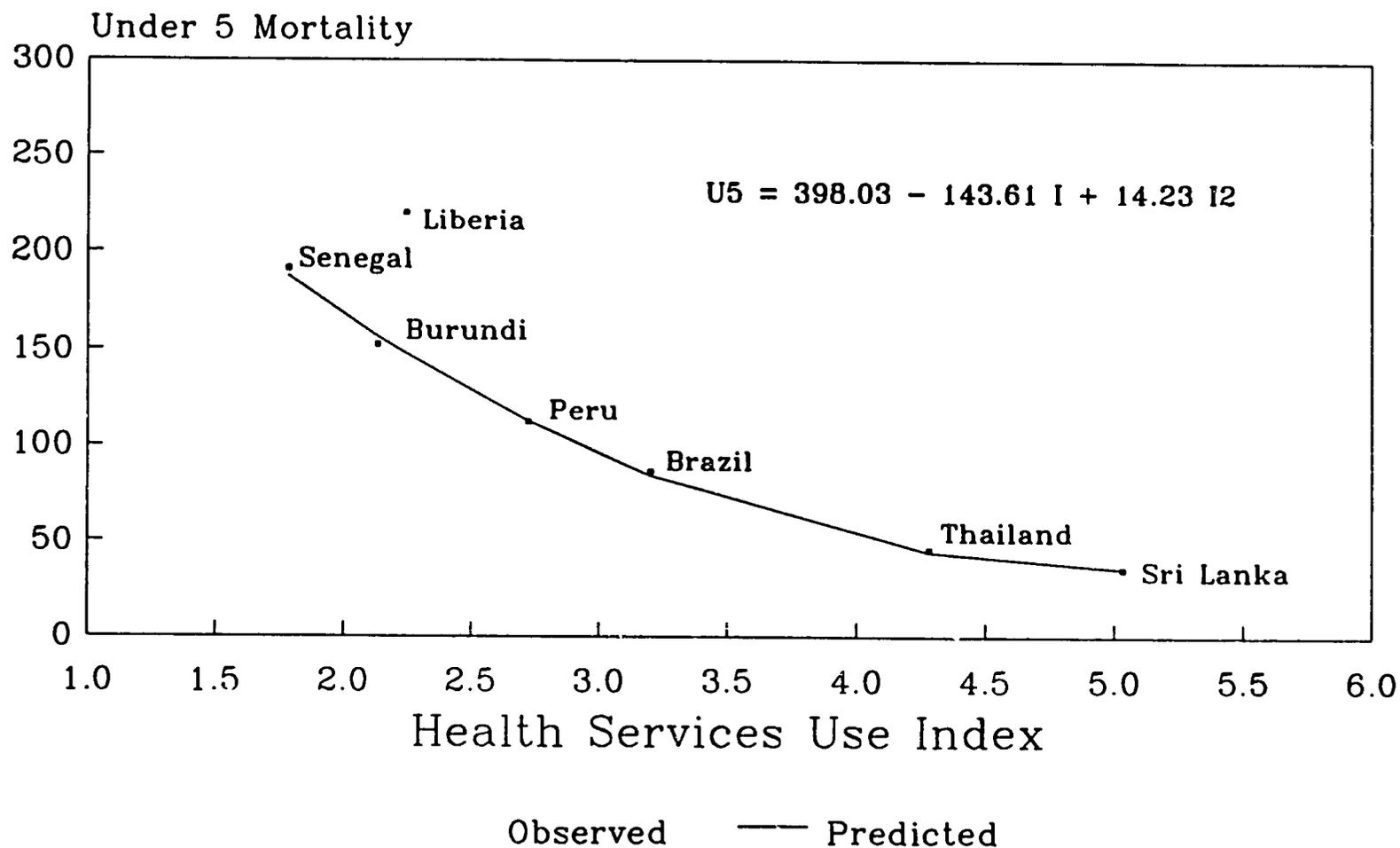


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45

Figure 25.

# Level of Under Five Mortality According to Index



76.

# APPENDIX 1

## SECTION 4. HEALTH AND BREASTFEEDING

401 CHECK 214:  
 ONE OR MORE LIVE BIRTHS SINCE JAN. 1982\*  NO LIVE BIRTHS SINCE JAN. 1982\*  (SKIP TO 428)

402 ENTER THE NAME, LINE NUMBER, AND SURVIVAL STATUS OF EACH BIRTH SINCE JAN. 1982\* IN THE TABLE. BEGIN WITH THE LAST BIRTH. ASK THE QUESTIONS ABOUT ALL OF THESE BIRTHS.

LINE NUMBER FROM Q. 212	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	LAST BIRTH NAME	NEXT-TO-LAST BIRTH NAME	SECOND-FROM-LAST NAME	THIRD-FROM-LAST NAME
	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>			
403 When you were pregnant with (NAME) were you given any injection to prevent the baby from getting tetanus, that is, convulsions after birth?	YES.....1 NO.....2 DK.....8	YES.....1 NO.....2 DK.....8	YES.....1 NO.....2 DK.....8	YES.....1 NO.....2 DK.....8
404 When you were pregnant with (NAME), did you see anyone for a check on this pregnancy? IF YES: Whom did you see? PROBE FOR THE TYPE OF PERSON AND RECORD THE MOST QUALIFIED.**	DOCTOR.....1 TRAINED NURSE/ MIDWIFE.....2 TRADITIONAL BIRTH ATTENDANT.....3 OTHER.....4 (SPECIFY) NO ONE.....5			
405 Who assisted with the delivery of (NAME)?  PROBE FOR THE TYPE OF PERSON AND RECORD THE MOST QUALIFIED.**	DOCTOR.....1 TRAINED NURSE/ MIDWIFE.....2 TRADITIONAL BIRTH ATTENDANT.....3 RELATIVE.....4 OTHER.....5 (SPECIFY) NO ONE.....6			
406 Did you ever feed (NAME) at the breast?	YES.....1 (SKIP TO 407)< NO.....2	YES.....1 (SKIP TO 408)< NO.....2	YES.....1 (SKIP TO 408)< NO.....2	YES.....1 (SKIP TO 408)< NO.....2
406A Why did you never feed (NAME) at the breast?*	INCONVENIENT....01 HAD TO WORK....02 INSUFFICIENT MILK..03 BABY REFUSED....04 CHILD DIED.....05 CHILD SICK.....06 OTHER.....07 (SPECIFY) (ALL SKIP TO 409)<	INCONVENIENT....01 HAD TO WORK....02 INSUFFICIENT MILK..03 BABY REFUSED....04 CHILD DIED.....05 CHILD SICK.....06 OTHER.....07 (SPECIFY) (ALL SKIP TO 409)<	INCONVENIENT....01 HAD TO WORK....02 INSUFFICIENT MILK..03 BABY REFUSED....04 CHILD DIED.....05 CHILD SICK.....06 OTHER.....07 (SPECIFY) (ALL SKIP TO 409)<	INCONVENIENT....01 HAD TO WORK....02 INSUFFICIENT MILK..03 BABY REFUSED....04 CHILD DIED.....05 CHILD SICK.....06 OTHER.....07 (SPECIFY) (ALL SKIP TO 409)<
407 Are you still breast-feeding (NAME)? (IF DEAD, CIRCLE '2')	YES.....1 (SKIP TO 409)< NO (OR DEAD).....2			
408 How many months did you breastfeed (NAME)?	MONTHS..... <input type="checkbox"/> UNTIL DEATH.....96 (SKIP TO 409)<			

\* For fieldwork beginning in 1988 or 1989, this date should be January, 1983 or 1984, respectively.  
 \*\* Coding categories to be developed locally and revised based on the pretest.

408A Why did you stop breastfeeding (NAME)?*	INCONVENIENT.....01 HAD TO WORK.....02 INSUFFICIENT MILK..03 BABY REFUSED.....04 CHILD DIED.....05 CHILD SICK.....06 CH HAD DIARRHEA..07 CH WEANING AGE...08 BECAME PREGNANT..09 OTHER.....10 (SPECIFY)	INCONVENIENT.....01 HAD TO WORK.....02 INSUFFICIENT MILK..03 BABY REFUSED.....04 CHILD DIED.....05 CHILD SICK.....06 CH HAD DIARRHEA..07 CH WEANING AGE...08 BECAME PREGNANT..09 OTHER.....10 (SPECIFY)	INCONVENIENT.....01 HAD TO WORK.....02 INSUFFICIENT MILK..03 BABY REFUSED.....04 CHILD DIED.....05 CHILD SICK.....06 CH HAD DIARRHEA..07 CH WEANING AGE...08 BECAME PREGNANT..09 OTHER.....10 (SPECIFY)	INCONVENIENT.....01 HAD TO WORK.....02 INSUFFICIENT MILK..03 BABY REFUSED.....04 CHILD DIED.....05 CHILD SICK.....06 CH HAD DIARRHEA..07 CH WEANING AGE...08 BECAME PREGNANT..09 OTHER.....10 (SPECIFY)
409 How many months after the birth of (NAME) did your period return?	MONTHS..... <input type="text"/> NOT RETURNED.....96	MONTHS..... <input type="text"/> NEVER RETURNED...96	MONTHS..... <input type="text"/> NEVER RETURNED...96	MONTHS..... <input type="text"/> NEVER RETURNED...96
410 Have you resumed sexual relations since the birth of (NAME)?	YES (OR PREGN.)...1 NO.....2 (GO TO NEXT COL)←			
411 How many months after the birth of (NAME) did you resume sexual relations?	MONTHS..... <input type="text"/> (GO TO NEXT COLUMN)	MONTHS..... <input type="text"/> (GO TO NEXT COLUMN)	MONTHS..... <input type="text"/> (GO TO NEXT COLUMN)	MONTHS..... <input type="text"/> (GO TO 412)

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP TO																					
412	CHECK 407 FOR LAST BIRTH: LAST CHILD STILL BREASTFED <input type="checkbox"/> ALL OTHERS <input type="checkbox"/>		418																					
413	How many times did you breastfeed last night between sundown and sunrise?	NUMBER OF TIMES..... <input type="text"/> AS OFTEN AS CHILD WANTED.....96																						
414	How many times did you breastfeed yesterday during the daylight hours?	NUMBER OF TIMES..... <input type="text"/> AS OFTEN AS CHILD WANTED.....96																						
415	At any time yesterday or last night, was (NAME OF LAST CHILD) given any of the following:*	<table border="0"> <tr> <td></td> <td>YES</td> <td>NO</td> </tr> <tr> <td>Plain water?</td> <td>PLAIN WATER.....1</td> <td>2</td> </tr> <tr> <td>Juice?</td> <td>JUICE.....1</td> <td>2</td> </tr> <tr> <td>Powdered milk?</td> <td>POWDERED MILK.....1</td> <td>2</td> </tr> <tr> <td>Cow's or goat's milk?</td> <td>COW'S OR GOAT'S MILK.....1</td> <td>2</td> </tr> <tr> <td>Any other liquid?</td> <td>ANY OTHER LIQUID.....1</td> <td>2</td> </tr> <tr> <td>Any solid or mushy food?</td> <td>(SPECIFY) ANY SOLID OR MUSHY FOOD.....1</td> <td>2</td> </tr> </table>		YES	NO	Plain water?	PLAIN WATER.....1	2	Juice?	JUICE.....1	2	Powdered milk?	POWDERED MILK.....1	2	Cow's or goat's milk?	COW'S OR GOAT'S MILK.....1	2	Any other liquid?	ANY OTHER LIQUID.....1	2	Any solid or mushy food?	(SPECIFY) ANY SOLID OR MUSHY FOOD.....1	2	
	YES	NO																						
Plain water?	PLAIN WATER.....1	2																						
Juice?	JUICE.....1	2																						
Powdered milk?	POWDERED MILK.....1	2																						
Cow's or goat's milk?	COW'S OR GOAT'S MILK.....1	2																						
Any other liquid?	ANY OTHER LIQUID.....1	2																						
Any solid or mushy food?	(SPECIFY) ANY SOLID OR MUSHY FOOD.....1	2																						
416	CHECK 415: WAS GIVEN FOOD OR LIQUID <input type="checkbox"/> NO FOOD OR LIQUID GIVEN <input type="checkbox"/>		418																					
417	Were any of these given in a bottle with a nipple?	YES.....1 NO.....2																						
418	At the time you became pregnant with (NAME OF LAST BIRTH), did you want to have that child then, did you want to wait until later, or did you want no (more) children at all?	THEN.....1 LATER.....2 NO MORE.....3																						

\* Coding categories to be developed locally and revised based on the pretest.

75

419 ENTER THE NAME, LINE NUMBER, AND SURVIVAL STATUS OF EACH BIRTH SINCE JAN. 1982\* BELOW. BEGIN WITH THE LAST BIRTH. THE HEADINGS IN THE TABLE SHOULD BE EXACTLY THE SAME AS THOSE AFTER Q. 402. ASK THE QUESTIONS ONLY FOR LIVING CHILDREN.

LINE NUMBER FROM Q. 212	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>
	LAST BIRTH NAME	NEXT-TO-LAST BIRTH NAME	SECOND-FROM-LAST NAME	THIRD-FROM-LAST NAME
	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>
420 Do you have a health card for (NAME)? IF YES: May I see it, please?	YES, SEEN.....1 YES, NOT SEEN.....2 (SKIP TO 422)<- NO CARD.....3	YES, SEEN.....1 YES, NOT SEEN.....2 (SKIP TO 422)<- NO CARD.....3	YES, SEEN.....1 YES, NOT SEEN.....2 (SKIP TO 422)<- NO CARD.....3	(GO TO 427) YES, SEEN.....1 YES, NOT SEEN.....2 (SKIP TO 422)<- NO CARD.....3
421 RECORD DATES OF IMMUNIZATIONS FROM HEALTH CARD.**	NOT GIVEN DA MO YR BCG 1 DPT 1 1 POLIO 1 1 DPT 2 1 POLIO 2 1 DPT 3 1 POLIO 3 1 MEASLES 1 (SKIP TO 423)	NOT GIVEN DA MO YR (SKIP TO 423)	NOT GIVEN DA MO YR (SKIP TO 423)	NOT GIVEN DA MO YR (SKIP TO 423)
422 Has (NAME) ever had a vaccination to prevent him/her from getting diseases?	YES.....1 NO.....2 DK.....8	YES.....1 NO.....2 DK.....8	YES.....1 NO.....2 DK.....8	YES.....1 NO.....2 DK.....8
423 Has (NAME) had diarrhea in the last 24 hours?	YES.....1 (SKIP TO 424A)<- NO.....2	YES.....1 (SKIP TO 424A)<- NO.....2	YES.....1 (SKIP TO 424A)<- NO.....2	YES.....1 (SKIP TO 424A)<- NO.....2
424 Has (NAME) had diarrhea in the last two weeks?	YES.....1 NO.....2 (GO TO NEXT COL)<- DK.....8	YES.....1 NO.....2 (GO TO NEXT COL)<- DK.....8	YES.....1 NO.....2 (GO TO NEXT COL)<- DK.....8	YES.....1 NO.....2 (SKIP TO 427)<- DK.....8
424A Now I have some questions about (NAME's) last episode of diarrhea. How many days ago did the diarrhea start?	DAYS..... <input type="text"/> DK.....98	DAYS..... <input type="text"/> DK.....98	DAYS..... <input type="text"/> DK.....98	DAYS..... <input type="text"/> DK.....98
424B CHECK 407: LAST CHILD STILL BREASTFED?	YES <input type="checkbox"/> NO <input type="checkbox"/> (SKIP TO 424C)			
424C Did you breastfeed (NAME) when he/she had diarrhea then?	YES.....1 NO.....2			

\* For fieldwork beginning in 1988 or 1989, this date should be January, 1983 or 1984, respectively.  
 \*\* Coding categories to be developed locally and revised based on the pretest.

79

424D When (NAME) had diarrhea then, was he/she given more, less, or the same amount to drink as before the diarrhea?	MORE.....1 LESS.....2 SAME.....3 DK.....8	MORE.....1 LESS.....2 SAME.....3 DK.....8	MORE.....1 LESS.....2 SAME.....3 DK.....8	MORE.....1 LESS.....2 SAME.....3 DK.....8
424E Was (NAME) given either a home solution of sugar, salt, and water to drink, or a solution made from a special packet? IF YES: Which?	HOME SOLUTION OF SALT, SUGAR, WATER.1 ORS PACKET SOLUTION.2 BOTH GIVEN.....3 NEITHER GIVEN.....4 (SKIP TO 424I)<	HOME SOLUTION OF SALT, SUGAR, WATER.1 ORS PACKET SOLUTION.2 BOTH GIVEN.....3 NEITHER GIVEN.....4 (SKIP TO 424I)<	HOME SOLUTION OF SALT, SUGAR, WATER.1 ORS PACKET SOLUTION.2 BOTH GIVEN.....3 NEITHER GIVEN.....4 (SKIP TO 424I)<	HOME SOLUTION OF SALT, SUGAR, WATER.1 ORS PACKET SOLUTION.2 BOTH GIVEN.....3 NEITHER GIVEN.....4 (SKIP TO 424I)<
424F The last time (NAME) was given (home solution/special packet), did he/she get better, worse, or was there no change?	BETTER.....1 WORSE.....2 NO CHANGE.....3	BETTER.....1 WORSE.....2 NO CHANGE.....3	BETTER.....1 WORSE.....2 NO CHANGE.....3	BETTER.....1 WORSE.....2 NO CHANGE.....3
424G How much of the (home solution/special packet) was (NAME) given every 24 hours?*	1/2 LITER.....1 1 LITER.....2 1 1/2 LITERS.....3 2 LITERS.....4 OTHER.....5 (SPECIFY) DK.....8	1/2 LITER.....1 1 LITER.....2 1 1/2 LITERS.....3 2 LITERS.....4 OTHER.....5 (SPECIFY) DK.....8	1/2 LITER.....1 1 LITER.....2 1 1/2 LITERS.....3 2 LITERS.....4 OTHER.....5 (SPECIFY) DK.....8	1/2 LITER.....1 1 LITER.....2 1 1/2 LITERS.....3 2 LITERS.....4 OTHER.....5 (SPECIFY) DK.....8
424H For how many days was (NAME) given (home solution/special packet)?	DAYS..... <input type="text"/> <input type="text"/> DK.....9B	DAYS..... <input type="text"/> <input type="text"/> DK.....9B	DAYS..... <input type="text"/> <input type="text"/> DK.....9B	DAYS..... <input type="text"/> <input type="text"/> DK.....9B
424I Was (NAME) given more, less, or the same amount of solid food as was given before he/she had diarrhea?	MORE.....1 LESS.....2 SAME.....3 SOLID FOODS NOT YET GIVEN.....4 DK.....8	MORE.....1 LESS.....2 SAME.....3 SOLID FOODS NOT YET GIVEN.....4 DK.....8	MORE.....1 LESS.....2 SAME.....3 SOLID FOODS NOT YET GIVEN.....4 DK.....8	MORE.....1 LESS.....2 SAME.....3 SOLID FOODS NOT YET GIVEN.....4 DK.....8
425 Was (NAME) taken to a private doctor, a hospital or clinic, a traditional doctor, or any other place during the last episode of diarrhea? IF YES: Where was he/she taken (the last time)?**	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5 (SKIP TO 426A)<	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5 (SKIP TO 426A)<	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5 (SKIP TO 426A)<	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5 (SKIP TO 426A)<
426 What treatments did (NAME) receive there (the last time)?**  CIRCLE ALL TREATMENTS MENTIONED.	INJECTION.....1 IV (INTRAVENOUS)....1 TABLETS OR PILLS....1 SYRUPS.....1 ORS.....1 OTHER.....1 (SPECIFY) NOTHING GIVEN.....1 (ALL GO TO NEXT COL)<	INJECTION.....1 IV (INTRAVENOUS)....1 TABLETS OR PILLS....1 SYRUPS.....1 ORS.....1 OTHER.....1 (SPECIFY) NOTHING GIVEN.....1 (ALL GO TO NEXT COL)<	INJECTION.....1 IV (INTRAVENOUS)....1 TABLETS OR PILLS....1 SYRUPS.....1 ORS.....1 OTHER.....1 (SPECIFY) NOTHING GIVEN.....1 (ALL GO TO NEXT COL)<	INJECTION.....1 IV (INTRAVENOUS)....1 TABLETS OR PILLS....1 SYRUPS.....1 ORS.....1 OTHER.....1 (SPECIFY) NOTHING GIVEN.....1 (ALL GO TO 427)<
426A Why was (NAME) not taken somewhere for treatment during the last episode of diarrhea?*	ILLNESS WAS MILD....1 MOTHER TOO BUSY....2 MOTHER WORKING.....3 NO FACILITIES AVAIL.4 OTHER.....5 (SPECIFY) (ALL GO TO NEXT COL)<	ILLNESS WAS MILD....1 MOTHER TOO BUSY....2 MOTHER WORKING.....3 NO FACILITIES AVAIL.4 OTHER.....5 (SPECIFY) (ALL GO TO NEXT COL)<	ILLNESS WAS MILD....1 MOTHER TOO BUSY....2 MOTHER WORKING.....3 NO FACILITIES AVAIL.4 OTHER.....5 (SPECIFY) (ALL GO TO NEXT COL)<	ILLNESS WAS MILD....1 MOTHER TOO BUSY....2 MOTHER WORKING.....3 NO FACILITIES AVAIL.4 OTHER.....5 (SPECIFY) (ALL GO TO 427)<

\* Develop response codes according to local instructions for mixing ORS.

\*\* Coding categories to be developed locally and revised based on the pretest.

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP TO
427	CHECK 424E.		
	HOME SOLUTION OR BOTH MENTIONED <input type="checkbox"/>	HOME SOLUTION NOT MENTIONED OR 0424E NOT ASKED <input type="checkbox"/>	428
427A	Where did you learn how to prepare the sugar, salt and water solution given to (NAME)?*	GOVERNMENT HOSPITAL.....01 GOVERNMENT HEALTH CENTER.....02 FIELD WORKER.....03 PRIVATE DOCTOR.....04 PRIVATE HOSPITAL/CLINIC.....05 PHARMACY.....06 TRADITIONAL DOCTOR.....07 OTHER.....08 (SPECIFY) MOTHER DID NOT ADMINISTER.....96 DK.....98	
428	Have you ever heard of a special product called (LOCAL NAME) you can get for the treatment of diarrhea?	YES.....1 NO.....2	
428A	Have you ever seen a packet like this before? (SHOW PACKET.)	YES.....1 NO.....2	428L
428B	Do you think this packet is used to <u>cure the diarrhea</u> , or that it is used <u>to prevent the child from drying out</u> ?	CURE DIARRHEA.....1 PREVENT DRYING OUT.....2 BOTH.....3 OTHER.....4 (SPECIFY) DK.....8	
428C	Have you ever prepared one of these packets for yourself or for someone else?	YES.....1 NO.....2	428M
428D	How much water did you use to prepare the packet (the last time)?**	1/2 LITER.....1 1 LITER.....2 1 1/2 LITERS.....3 2 LITERS.....4 OTHER.....5 (SPECIFY) DK.....8	
428E	Did you use boiled water, bottled water, or other water to prepare the packet (the last time)?	BOILED WATER.....1 BOTTLED WATER.....2 OTHER.....3 (SPECIFY) DK.....8	

\* Coding categories to be developed locally and revised based on the pretest.  
 \*\* Develop response codes according to local instructions for mixing ORS.

NO.	QUESTIONS AND FILTERS	CODING CATEGORIES	SKIP TO
428F	In what kind of container did you prepare the mixture of the packet and the water?*	COOKING POT.....1 EARTHEN JAR.....2 EMPTY BOTTLE.....3 CALABASH.....4 OTHER.....5 (SPECIFY)	
428G	Did you prepare a new mixture every day or did you use the same mixture for more than one day?	NEW MIXTURE EACH DAY.....1 USE SAME FOR MORE THAN 1 DAY....2 OTHER.....3 (SPECIFY)	
428H	Where can you get these packets?* PROBE: Anywhere else? CIRCLE ALL PLACES MENTIONED.	GOVERNMENT HOSPITAL.....1 GOVERNMENT HEALTH CENTER.....1 FIELD WORKER.....1 PRIVATE DOCTOR.....1 PRIVATE HOSPITAL/CLINIC.....1 PHARMACY.....1 SHOP.....1 TRADITIONAL DOCTOR.....1 OTHER.....1 (SPECIFY) DK..... 1	
428I	How much do (you think) the packets cost?*	COST..... <input type="text"/> <input type="text"/> <input type="text"/> FREE.....996 DK.....998	
428J	Do you have one of these packets in your house now?	YES.....1 NO.....2	→428L
428K	Can I see the packet?	SHOWS PACKET.....1 DOES NOT SHOW PACKET.....2	
428L	Which places can you go if you want to get a vaccination for a child?* CIRCLE ALL PLACES MENTIONED.	GOVERNMENT HOSPITAL.....1 GOVERNMENT HEALTH CENTER.....1 FIELD WORKER.....1 PRIVATE DOCTOR.....1 PRIVATE HOSPITAL/CLINIC.....1 PHARMACY.....1 SCHOOL.....1 SPECIAL CAMP.....1 TRADITIONAL DOCTOR.....1 OTHER.....1 (SPECIFY)	

\* Coding categories to be developed locally and revised based on the pretest.  
 \*\* Revise cost code according to local currency.

*DL*

429 ENTER THE NAME, LINE NUMBER, AND SURVIVAL STATUS OF EACH BIRTH SINCE JAN. 1982\* BELOW. BEGIN WITH THE LAST BIRTH. THE HEADINGS IN THE TABLE SHOULD BE EXACTLY THE SAME AS THOSE AFTER Q. 419. ASK THE QUESTIONS ONLY FOR LIVING CHILDREN. IF NO CHILDREN SINCE JAN. 1982, SKIP TO 501.

LINE NUMBER FROM Q. 212	[ ] [ ]		[ ] [ ]		[ ] [ ]		[ ] [ ]	
	LAST BIRTH		NEXT-TO-LAST BIRTH		SECOND-FROM-LAST		THIRD-FROM-LAST	
	NAME	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>	NAME	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>	NAME	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>	NAME	ALIVE <input type="checkbox"/> DEAD <input type="checkbox"/>
430 Has (NAME) had fever in the last four weeks?	YES.....1 NO.....2 (SKIP TO 433)← DK.....8	YES.....1 NO.....2 (SKIP TO 433)← DK.....8	YES.....1 NO.....2 (SKIP TO 433)← DK.....8	YES.....1 NO.....2 (SKIP TO 433)← DK.....8	(GO TO 501) YES.....1 NO.....2 (SKIP TO 433)← DK.....8			
431 Was (NAME) taken to a private doctor, a hospital or clinic, a traditional doctor, or any other place to treat the fever? IF YES: Where was he/she taken?*	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5						
432 Was there anything (else) you or somebody did to treat the fever? IF YES: What was done? CIRCLE CODE 1 FOR ALL MENTIONED.**	ANTIMALARIAL.....1 ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1	ANTIMALARIAL.....1 ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1	ANTIMALARIAL.....1 ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1	ANTIMALARIAL.....1 ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1	ANTIMALARIAL.....1 ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1			
433 Has (NAME) suffered from severe cough or difficult or rapid breathing in the last four weeks?	YES.....1 NO.....2 (GO TO NEXT COL)← DK.....8	(SKIP TO 501)← YES.....1 NO.....2 (SKIP TO 501)← DK.....8						
434 Was (NAME) taken to a private doctor, a hospital or clinic, a traditional doctor, or any other place to treat the problem? IF YES: Where was he/she taken?	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5	PRIVATE DOCTOR.....1 HOSPITAL/CLINIC.....2 TRADITIONAL DOCTOR..3 OTHER.....4 (SPECIFY) CHILD NOT TAKEN.....5						
435 Was there anything (else) you or somebody did to treat the problem? IF YES: What was done? CIRCLE CODE 1 FOR ALL MENTIONED.**	ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1 (ALL GO TO NEXT COL)	ANTIBIOTICS.....1 LIQUID OR SYRUP.....1 ASPIRIN.....1 INJECTION.....1 OTHER.....1 (SPECIFY) NOTHING.....1 (ALL GO 501)						

\* For fieldwork beginning in 1988 or 1989, this date should be January, 1983 or 1984, respectively.  
\*\* Coding categories to be developed locally and revised based on the pretest.

83

# Impact of the Direct Interventions

Anne Gadomski<sup>1</sup> and Robert Black<sup>2</sup>

## INTRODUCTION

During the past decade, we have witnessed a change in emphasis in international health programming, away from the costly development of tertiary and curative care structures to the development of primary care interventions. The latter, perceived as more cost-effective, were targeted at the major causes of childhood morbidity and mortality in developing countries, which are estimated to be responsible for 10.8 million of the 14 million deaths of children under five. According to 1987 UNICEF and WHO estimates, the major causes of mortality were diarrheal disease (5 million), acute respiratory infection (2.9 million), measles (1.9 million) and malaria (1 million). Malnutrition is a contributory cause of death in one-third of the 14 million (1).

The direct interventions include oral rehydration therapy and diarrheal disease control, acute respiratory infection control, immunizations, malaria control and nutrition programs. These strategies were not intended to replace development of comprehensive health care, but to function as stopgap measures to improve child survival while the more time consuming and costly development of a more comprehensive health care system took place. What impact have the direct interventions had? The answer to this question has been difficult to construct for the following reasons. In such a short period of time (five to ten years), it is difficult to measure the impact of these strategies as much of this time as been devoted to training and infrastructure development as well as to the implementation of these strategies. Additionally, measuring program effects is always confounded by other influences on health as well as by replacement mortality. Large scale program evaluation is often an expensive and resource consuming process that limits the number and quality of such evaluations. It can be said that the programs have yet to prove their effectiveness in long term mortality reduction.

Nevertheless, estimates have been made as to what impact these interventions have had on childhood morbidity and mortality in developing countries. This paper will describe the estimates that are available, review the performance and limitations of the direct interventions, and examine the prospects for future developments in these fields. In conclusion, comments on the appropriateness of the current and future strategies will be made in light of expected changing conditions in less developed countries (LDC's).

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## I. CONTROL OF DIARRHEAL DISEASES (CDD)

### A. Rationale for the Intervention

**Disease burden:** Based on a review of surveillance data in 1980, an estimated 744 to 1000 million episodes of acute diarrheal illness occurred, and an estimated 4.5 million deaths in under fives had occurred in developing countries. Children under five have an average of two to three episodes of diarrhea a year (2). Two to 20% of acute diarrheal disease episodes become persistent, chronic diarrhea with an increased risk of precipitating malnutrition (3). The Institute of Medicine (4) derived an estimate of 3.7 million deaths due to diarrhea in under fives based on the assumption that 25% of infant and child deaths in LDC are due to diarrhea. Of these diarrheal deaths, an estimated 524,000 are due to dysentery (5).

**Purpose of the intervention:** The CDD programme was initiated in 1980 by WHO to reduce mortality and morbidity due to diarrheal diseases and associated malnutrition in under fives. Control efforts include the training and promotion of appropriate management of acute diarrheal disease. Case management, termed oral rehydration therapy (ORT), is based on the use of oral rehydration solution (ORS) and/or appropriate home made fluids and continued feeding during diarrhea. CRT is expected to have a greater impact on mortality associated with watery diarrhea than on dysentery (bloody diarrhea), which may require specific antibiotic therapy. Prevention of diarrhea through health education efforts aimed at promoting breastfeeding and improving personal hygiene, food handling practices particularly with regard to weaning foods, and promoting the penning of animals (livestock, chickens, etc.) should be important components of the CDD program, but have not been fully implemented.

### B. Implementation

**The Model Program:** The model program promotes the use of fluids and foods at home early in the course of a diarrheal episode. If the diarrhea worsens, the program encourages bringing the child to the community health worker (CHW) or health facility for ORS. The program trains health professionals in the use of ORS and the promotion of ORT and breastfeeding, and attempts to increase the availability of ORS. Health education on prevention of diarrhea is limited.

**Coverage:** CDD programs are in operation in 96 countries, and in the planning stages in an additional 16. These 96 countries comprise an estimated 98% of the total population in developing countries (6). The quantity of ORS packets available in 1986 worldwide (330 million) was three times the quantity available in 1983. 1987 estimates suggest a leveling off. 55 countries during 1987 were producing their own ORS packets (6). UNICEF is the major external supplier of ORS distributing 65 million packets to 64 countries in 1986, and 58 million packets to 70 countries in 1987 (7).

WHO estimates that 59% of children in developing countries had access to ORS in 1986 (see FIGURE 1). Access implies that the provider of ORS lives within 5 km or one hour's walk (8).

An estimated 23% of children with diarrhea received ORT in 1986. ORT use rate is defined as the proportion of episodes of diarrhea in under fives treated with ORT (8). The trend for use of ORS and ORT follows the same upward trend as that for access to

ORS, as shown in TABLE 1 for the same time period.

The WHO data on access and use is estimated from data combined from three sources: information reported by countries to WHO, cross-sectional cluster-sample survey data, and ORS supply and distribution figures obtained from several international organizations (8). The Demographic and Health Surveys (DHS) national surveys, an AID-supported effort, are an additional source of information on ORT use. These surveys collect detailed demographic, reproductive and child health related information from a national sample of 4,000 to 12,000 women aged 15 to 49 (9). Survey results on the number of diarrheal episodes (mother's definition) in the past two weeks and what percent of children were treated with ORT are presented in TABLE 2 for the countries for which this data is available (10). The most recent (1986+) WHO ORT use rate estimates are also presented in the same table. These data are not intended for strict comparison due to the various ways the estimates have been derived (see table footnotes).

The WHO ORT use rates do not take into account the quality or effectiveness of the use of ORT. The effective use of ORS and ORT has been difficult to measure objectively in field settings, and global estimates are not available. The WHO is developing field methodology to estimate effective diarrhea case-management (8). Likewise, questions designed to elicit an estimate on the effective use of ORS (i.e. the respondent's knowledge about whether ORS is used to cure diarrhea or to prevent dehydration, how much water was used to prepare the ORS, etc.) have been included in some DHS surveys (11).

### C. Implementation Outcomes

Reduction in Mortality: Since an estimated 50-60 % of all diarrheal deaths are acute and probably due to dehydration, appropriate and timely rehydration of the child with diarrhea should have a considerable impact on diarrheal mortality. Reductions in diarrheal deaths of up to 50% with 80% coverage have been demonstrated in controlled community based oral rehydration therapy interventions (12). No information is available on how the intervention affected overall mortality.

A field trial, done in 1980, of ORT promotion in Dakahlia, Egypt, demonstrated a decreased mortality rate in children under five during the diarrheal season in the outreach villages (10.5/1000) versus the control villages (18.1/1000) (13). In 1983, AID-funded efforts were made to strengthen the CDD program nationwide by increasing ORT training of health personnel, increasing national production and distribution of ORS, establishing rehydration rooms, and instituting a mass media campaign. A follow-up study in 1986 of the control and outreach areas in Dakahlia demonstrated a further reduction in overall mortality in under fives, primarily due to a reduction in diarrhea-associated mortality as shown in TABLE 3.

Mortality had been declining prior to the initiation of the CDD program, however the accelerated rate of decline after 1983 was attributed to the CDD program. Measles vaccination may have contributed to this decline; but this is uncertain because rates of measles vaccination coverage had varied 41-85% since 1979.

According to WHO, the rate of use of ORS and ORT in 1986 (23%) may have prevented an estimated 700,000 deaths that year (6). This figure was derived by the following formula (3):

$$\begin{aligned} \text{Diarrheal Death Averted} &= (\text{ORT use rate}) (\% \text{ of Rx'd cases in which ORT used effectively}) \\ & \quad (\% \text{ of diarrheal deaths averted by effective use of ORT}) (\text{total diarrheal deaths in the absence of Rx}) \\ &= (.23)(.80)(.67)(5.7 \text{ million}) = 702,696 \end{aligned}$$

If the objectives of 80% access to ORS and 50% use of ORT can be attained by 1989, then an estimated 1.5 million diarrheal disease deaths among children in developing countries would be prevented each year (6).

In an effort to evaluate the impact ORT has had, hospital data from developing countries with CDD programmes were compared pre and post ORT initiation. Overall case fatality rates for diarrhea decreased by 67% to 95% with a median of 71% in 14 hospitals. Hospital admission rates for diarrheal disease in 14 hospitals decreased a median of 61%. Inpatient case fatality however did not demonstrate a clear diminishing trend, as 11 hospitals showed a significant reduction, 13 showed nonsignificant change, and two showed an increase. The case fatality trends must be interpreted in light of the reduction in total hospitals admissions such that only the most severe cases were admitted (see TABLES 4 and 5).

Indirect Effects and Improvements in Health: Diarrheal control can improve child health by curtailing the negative effect diarrhea has on growth and nutritional status. Diarrhea negatively impacts on nutritional status by decreasing intake, increasing losses in stools, decreasing enzyme activity, and increasing utilization of nutrients due to the catabolic effects of infection (15). Approximately 20% of the growth differential between children in selected developing countries and children in North America can be attributed to diarrhea (16). Fewer diarrheal episodes, particularly less dysentery and prolonged diarrhea, may mean less growth faltering and decreased risk of malnutrition (if other infections are controlled as well and food is available). Recent studies have added more support to the hypothesis that malnutrition is a risk factor for both an increased incidence, duration, and severity of diarrheal disease (17,18,19,20). Improving nutritional status could be a primary intervention for diarrheal disease control because decreasing the adverse effect of one episode of diarrhea on the child's nutritional status may decrease the incidence and severity of subsequent episodes of diarrhea (see section on nutrition).

#### D. Implementation Problems

These include limited access and availability of ORS, and knowledge about ORT. Although limited access and availability of ORS has been partially overcome by the promotion of home fluids, a reliable supply of ORS is still necessary. Inadequate training of health professionals in case management, inadequate supervision, lack of coordination with other programmes and use of health education, inadequate surveillance data and operational problems also plague progress in CDD (6). Incorporation of ORT into medical education has also been slow. WHO estimates that up to 30% of upper level health staff have been trained in some of the largest countries with CDD programs, however in 48 countries, only an average of 9% were trained (7).

From the community's standpoint, acceptance of ORS is compromised by the expectation that ORS is a cure for diarrhea when in fact it does not decrease stool output, but prevents dehydration. What the community wants and often expects from Western medicine is a cure. Disillusionment with ORT grows quickly as diarrheal stools keep coming. Prospects for an ORS that decreases stool frequency and volume (results that the

caretaker can see) will most likely increase the community acceptance of this intervention.

Educational efforts are complicated by social, behavioral and economic factors. For example, different messages about ORS and ORT given by multiple providers in the private and public sectors may lead to confusion in the community. Often there is lack of agreement on ORS formulations or ORT fluids amongst providers serving the same geographic area. Additionally, education pertaining to personal hygiene and food preparation designed to prevent diarrhea is less tangible than messages about ORT. The lack of visible and immediate results associated with these measures contributes to poor acceptance by community.

A substantial proportion of diarrhea-associated deaths are not due to dehydration and ORT is unlikely to prevent these. Nondehydration-related diarrheal mortality is associated with dysentery and prolonged diarrhea, about which less is known. Strategies are needed to address these subsets of diarrheal mortality, and these will require field testing (21).

Socioeconomic factors limit the impact CDD can have on child survival. For example, the opportunity costs associated with clinic visits (long waits, long distance to travel means time lost away from income or food generating activities) may lead to the late presentation of the child to referral clinic, and therefore more severe dehydration or dysentery occurs. Emphasis on early intervention with ORT in the home may circumvent this problem, but this requires intensive education and media exposure. Changes in feeding during diarrhea may be difficult to implement due to lack of time or resources with which to implement the prescribed change. Additionally, overcoming cultural practices (e.g. withholding certain foods during diarrhea) and/or habits involves behavioral modification for an end (e.g. weight gain) that is remote and difficult to make tangible over the short period of time.

From the donor or policy maker's perspective, accountability to the donor's constituencies leads to a preference for interventions that are quick to implement, have measurable inputs and outcomes, and dramatic effects over the span of the usual funding cycles (1-5 years). While ORT fits some of these criteria, it is limited in that it is essentially a curative intervention. It is representative of short-term interventions that are easier to implement and visible, but often at odds with the more long-term needs and less easily perceived wants of a community in a developing country.

#### E. Future Prospects

Prospects for improving implementation of existing interventions include ORS formulations such as cooked rice powder (22) and hydrolyzed wheat based ORT that reduce the volume and duration of diarrhea (23). Rice-based ORS is currently the standard form of therapy at ICDDR-B.

Prospects for better controlling the problem include the nutritional or dietary management of diarrhea, which is currently receiving much research attention and will be addressed in the Nutrition section of this paper. These interventions designed to break the vicious cycle between diarrhea and malnutrition should specifically target high risk children, especially those who are already malnourished.

Preventing dysentery morbidity and mortality requires identification of the child with dysentery, appropriate antibiotic administration and follow up, and dietary management. Implementation of treatment algorithms for the case management of dysentery and prolonged diarrhea (5,21) will require evaluation to assess what impact these will have on diarrheal mortality.

The protective effect of breastfeeding both on the incidence and severity of diarrheal diseases has been documented in several studies (24). In a controlled clinical trial, infants who were receiving ORS and breastfed through their acute diarrheal episode had fewer and smaller volume stools than those receiving just ORS(25). A prospective study of infant feeding practices in Peru and their relation to infection demonstrated that the incidence and prevalence rates of diarrhea in exclusively breastfed infants less than six months of age were lower than in infants of the same age who received other liquids in addition to breast milk (26). The authors estimated the potential impact of improved feeding practices in the study community and found that universally practiced exclusive breastfeeding for six months would theoretically reduce the incidence of diarrhea by one third and the prevalence by one half. Breastfeeding would be expected to impact diarrheal disease directly by its anti-infective properties, and indirectly by delaying exposure to pathogens in bottled milk, other liquids and solid foods offered to infants, and by improved nutritional status.

Hygiene education and handwashing may reduce diarrhea incidence rates by 14-48% (27). Enteropathogens may survive 3 hours or longer on contaminated hands, and handwashing is particularly relevant to avoiding infection with Shigella that have a very low infectious dose (200 organisms or less) (27,28).

The relative importance of pathogens that cause diarrhea have guided vaccine development efforts. In Bangladesh, rotavirus or enterotoxigenic E.coli were isolated in 70% of the dehydrating diarrheal episodes (29). Rotavirus vaccine development was given a high ranking and E.coli an intermediate ranking based on their potential benefits and likelihood of successful development under certain assumptions and resource constraints (4). The need for a multivalent rotavirus vaccine is slowing development of this vaccine (30). Since Shigella is an important cause of dysentery and prolonged diarrhea, contributes to the development of malnutrition (16) and has a high case fatality, it also has received a high priority ranking for vaccine development. An additional consideration in vaccine development is cost-effectiveness, e.g. cholera vaccine might not be cost effective given current low cost of treatment of a cholera episode versus the projected cost of delivering an effective vaccine (personal communication with David Sack, October 1988).

Prospects for achieving sustainable health improvements: Though it has been methodologically difficult to measure the impact of improved water and sanitation interventions on diarrheal disease, a review of this topic concluded that diarrheal morbidity rates were reduced by a median of 22-27% (53 studies), diarrheal incidence rates reduced by 16 -37%, and diarrheal mortality by a median 41% (only two studies) (31). The interventions included those that improved water supply or excreta disposal, which had a greater impact on diarrheal disease than improving water quality. In addition, improving water and sanitation is likely to improve health status in other ways.

## II. ACUTE RESPIRATORY INFECTION (ARI) CONTROL

### A. Rationale for the Intervention

**Disease Burden:** Mortality rates from acute lower respiratory infection (ALRI) range from 10.4 -30 per 1000 infants and 1.6 - 4 per 1000 children one to four years, based on prospective surveillance data from studies done in Narangwal, Matlab and Papua, New Guinea (2). ALRI has been estimated to cause 25 to 33% of all deaths of children under five (33). By applying estimated ALRI mortality rates to population distribution in developing countries in 1984, the estimated number of ALRI deaths is 13.5% of the 10.4 million infant deaths (less than one year of age), and 22% of 4.4 million child deaths (1 to 4 years of age), for a total of 18% for the under 5 years (4). WHO combined ARI/CDD home surveys have determined the following mortality rates (based on retrospective histories):

Fortaleza, Brazil	20 deaths/1303 0-4 year olds of which 30% were ALRI-associated.
Haryana State, India	258 deaths/15025 0-4 year olds of which 14.3% were ALRI-associated.
Sichuan Province, China	165 deaths/7126 0-4 year olds of which 36% were associated with pneumonia (the leading cause of death). Of the 59 pneumonia deaths, 38 were reported to have occurred at home (34).

Compared to diarrheal disease, much less is known about the etiology and extent of ALRI, particularly with regard to the relative importance of viral vs bacterial etiologies in ALRI mortality. This has been due to the greater difficulty of determining the etiology of ALRI and defining the illness, as well as to the relative inattention this area has received (probably because of these difficulties). The few studies that have utilized lung aspiration in hospitalized children have shown that the bacteria, Haemophilus influenzae and Streptococcus pneumoniae were isolated in half the cases that had a bacterial isolate while Staphylococcus aureus accounts for 17% (35). In Papua New Guinea, bacteria (S.pneumoniae and H.influenzae) were found to be the predominant causes of fatal pneumonias (36). Of the viruses isolated in hospitalized children, RSV, parainfluenza, influenza and adenovirus were the most prevalent (35). These are the same agents that account for most viral ALRI in developed countries.

**Purpose of the intervention:** Community based case management following the WHO algorithm, is designed to reduce ALRI mortality by providing timely referral of ALRI and antibiotic treatment. Measles and DPT vaccine do have a significant role in the prevention of ALRI, but this discussion will focus on ALRI case management, and the above vaccines will be discussed in the EPI section. Other primary preventive measures include health education.

### B. Implementation

**The Model Program** employs a CHW to identify the severity of ARI and to treat or refer based on observable signs that are employed in the WHO treatment algorithm. The algorithm allows the CHW to distinguish between pneumonia, otitis media, and other specific ALRI, and outlines appropriate treatment, supportive measures and need for referral. Respiratory rates are counted by the CHW in order to differentiate pneumonia from other ARI. A health worker is trained to administer antibiotics for pneumonia.

Health education pertains to the recognition of signs of pneumonia as this will aid in self referral to the CHW. The importance of immunization is also stressed in the program.

Coverage: Relative to the CDD program, the ARI program is a younger and much less extensively implemented. The first meeting of the WHO ARI Technical Advisory Group was in March 1983. As of December 1987, 29 countries, mostly in Latin America (14), had initiated ARI control programs and in 17 of these, the programs were operating. In Africa, Tanzania and Zimbabwe have programs operative in limited areas, while in Malawi and Swaziland, programs are being planned. In SE Asia, Indonesia has an operative program, while Burma and Sri Lanka are in the initial stages (34).

### C. Implementation Outcomes

Reduction in Mortality: In the Narangwal study, use of penicillin reduced pneumonia case fatality from 10.5 per 100 episodes to 2.2 per 100 episodes (37). This study also demonstrated that CHW's could be trained to administer antibiotics safely (38). Subsequently, five studies have shown that training of CHW's to follow the WHO algorithm is feasible, and that case management can have a significant impact on ALRI mortality (see FIGURE 2)(39).

In Jumla, Nepal, active ALRI case finding, and case management with cotrimoxazole were the sole interventions introduced in an area that had high infant mortality and malnutrition. Preliminary results suggest a 18% reduction in the total under five mortality rate and in ALRI-specific mortality compared to the control group (all figures are subject to further statistical review) (N. Daulaire, personal communication, 1990). In the Haryana, India study, active case finding among a cohort of low birth weight infants resulted in a marked reduction in ALRI case fatality from 25% in control areas to 9% in the intervention areas (40). Higher coverage of the intervention area with DPT and BCG may have contributed to this reduction as well.

In a pre and post evaluation of an active case finding program in Abbotabad, Pakistan, overall and ALRI-specific mortality in under fives were reduced by 55% and 52% respectively. However, immunizations, a food supplement program for pregnant women and a diarrheal disease control program were being implemented at the same time as the ARI case management program, but to an equal extent in the control and intervention areas. In the Kathmandu Valley study, immunization, case management of diarrhea and maternal education about ARI and breastfeeding were introduced in addition to ALRI active case finding and management, and mortality rates were compared pre and post intervention. Overall under five mortality and IMR were reduced by 40%, ALRI specific mortality (primarily non-measles associated pneumonia) of under fives was reduced by 60-70%, and case fatality for pneumonia treated by the study team was 1% (39).

Active ALRI case finding, referral and health and nutrition education about ALRI by CHW's in Bagamoyo District in Tanzania demonstrated within two years a reduction of overall under five mortality by 27% (from 40.1/1000 to 29.2/1000) and pneumonia mortality in the under fives by 30% (14.3/1000 to 10/1000)(41).

Indirect Effects and Improvements in Health: Several of the above studies demonstrate a reduction in total mortality, a reduction that is sometimes larger than that due to ALRI alone (39). The reduction in overall mortality is surprising because it implies

that replacement mortality is not overriding the impact of this particular intervention (39). It may also suggest that the antibiotic management may be having an effect on other illnesses, as in Nepal where there may have been a reduction in deaths associated with prolonged diarrhea. Reducing the ALRI disease burden is likely to prevent the nutritional losses associated with this illness (see Nutrition section).

#### D. Implementation Problems

Given the limited extent of the implementation of ARI control programs at this point in time, problems are less evident and have yet to arise. However, it can be said that the threat of antibiotic resistance is always present; how quickly this resistance may develop is unknown. If CHW's give shorter courses in order to conserve scarce supplies, as frequently occurs in malaria disease control, then this may hasten the development of resistance. Widespread availability of antibiotics has in the past contributed greatly to the development of resistance, e.g. chloramphenicol resistance in enteric organisms in India, or to tetracycline in cholera in Tanzania. Monitoring antibiotic sensitivity introduces the need for a reliable lab as well as reporting and surveillance activities.

While the training of health professionals and CHW's in ARI case management appears to be feasible, supervision and monitoring will be required to maintain interest and enthusiasm over time. Considerably more effort is involved in active case finding than passive, so the feasibility of this may be questionable. Passive case finding may be less effective than active searching for cases, but could be augmented by health education efforts designed to improve the recognition of ALRI disease severity. Relying on passive case finding may be equivalent to self-referral. Therefore, the same constraints that prevent timely visits to the primary health care center for other illnesses will prevent early identification and therefore prompt treatment of the child with ALRI. These constraints, described in the CDD section, include opportunity costs associated with clinic visits as well as accessibility of the CHW.

As is true of most of the direct interventions, an existing primary health care infrastructure can facilitate the development of the ARI program. It will be difficult to implement case management in areas where this infrastructure is poor or lacking, since effective case management depends on a reliable supply of antibiotics, adequate training and supervision of CHW's and on an operative referral network.

From the community's perspective in Jumla, acceptance of the ARI program has been high and may have led toward a demand for increased services. Whether this will be true in other settings has yet to be determined. From the donor's perspective, the lack of exposure to and experience with ARI programs has limited support for these programs. This lack of support for ARI programs, in spite of demonstrated effectiveness, may be changing in recent years.

#### E. Future Prospects

For improving implementation of existing intervention, many of the same training, supervision and management problems that have arisen in the CDD program could be anticipated in the implementation of ARI programs. Maintaining the supply of antibiotics could be facilitated if other existing channels for the provision of medicines i.e. CDD, Malaria, EPI or Essential Drugs are utilized.

For better controlling the problem: Improving nutritional status may reduce the severity of ALRI in developing countries. Feeding during the ARI illness may reduce the nutritional insult of the episode, which may leave the host better able to withstand the next round of infections.

While reducing the severity of ALRI depends on early identification and case management, reducing the incidence of ALRI will depend in part on vaccine development. WHO has assigned a high priority to the development of a vaccine for respiratory syncytial virus (RSV) and parainfluenza viruses (30), while the Institute of Medicine has ranked S. pneumoniae high, and H.influenzae (the Hib vaccine), RSV and parainfluenza intermediate (4). Hib vaccine currently is quite expensive and still undergoing development to improve its efficacy in children less than 18 months of age. The prospects for RSV and parainfluenza are more distant (30). Until vaccines can be developed and delivered for these agents, emphasis will have to be on case management when the disease occurs.

For achieving sustainable health improvements, socioeconomic development is necessary to improve housing, decrease crowding, improve ventilation of indoor fires, prevent air pollution and control smoking, all of which would be expected to reduce the rate of ALRI.

### III. EXPANDED PROGRAMME ON IMMUNIZATION (EPI)

#### A. Rationale for the Intervention

Disease Burden: EPI estimates that the immunizable diseases account for over 3.4 million deaths per year. Measles causes nearly 2 million deaths each year, more deaths than all the other EPI target diseases combined. It is highly contagious and nearly all (90%) non-immunized children will contract the disease (42). 70 million cases were estimated to occur as of 1987, and the above mortality estimate is obtained by applying a case fatality rate (CFR) of 2-4%. However, the CFR for children under five obtained from prospective community studies ranges from 2% (Bangladesh) to 34% (Guinea-Bissau), making the above estimate conservative (43). Delayed excess mortality, particularly in children who have had measles before one year of age may also contribute to the underestimation of the full extent of measles on child mortality (43).

Neonatal tetanus causes an estimated 800,000 deaths each year. Case fatality rate is nearly 85%. Pertussis causes an estimated 600,000 childhood deaths per year. 250,000 cases of polio still occur annually, resulting in few deaths but substantial disability (42). An estimated two million cases of tuberculosis in under fives occur annually, of which 60,000 have meningeal involvement with mortality ranging between 50 and 100 % (44).

Purpose of the EPI is to reduce morbidity and mortality from the above diseases by vaccination of under one year olds with three DPT, three OPV, one measles and one BCG vaccine dose, and to prevent neonatal tetanus by vaccinating pregnant women with tetanus toxoid.

#### B. Implementation

The Model Program vaccinates infants and pregnant women through the primary health care infrastructure. Exceptions to this have been accelerated strategies implemented

through vaccination campaigns (46). The advantages of the latter approach have been its high visibility, high coverage, low vaccine wastage, and short term efficiency particularly in areas where a PHC infrastructure is lacking. A dramatic decrease of polio incidence followed the introduction of mass immunization on two days a year in Brazil (47). Liberia is an example of combined PHC and campaign approaches, as it has one annual vaccination week that coincides with the end of the planting season (convenient for recipients) and precedes the measles season (epidemiologically wise). This effort serves to augment routine coverage by the fixed PHC centers by approximately 50% of annual vaccinations (48). Disadvantages of the campaign approaches are increased cost of maintaining a vertical program, poor sustainability, subsequent slippage to precampaign levels of coverage postcampaign, and diversion of resources away from supporting PHC (44).

Coverage: 97 countries have EPI programmes (42). In 1974, WHO estimates that less than 5% of infants were covered with 3 doses of DPT or polio vaccines. In 1982 the global immunization rate was between 10 and 15%. As of June 1987, the global immunization coverage with 3 DPT or polio was estimated at 53% (excluding USA and Canada) (42). The breakdown of coverage by WHO region by July 1988 is shown in TABLE 6, and is compiled from data reported by countries to the WHO, and is therefore limited by incomplete reporting, etc.

Review of country specific immunization coverage demonstrates a pattern of increased coverage in areas where a health infrastructure is already in operation, and socioeconomic development is progressing (see immunization coverage for AID emphasis countries available in Appendix A compiled from references 50,51). Given the large number of infants in China, India, Indonesia and Nigeria, improving coverage in these countries would have a significant impact on the global estimates (42).

### C. Implementation Outcomes

Efficacy: The vaccine efficacy figures that are used by the WHO are: measles vaccine 95%, pertussis 80%, OPV 95%. The clinical efficacy of OPV immunization starting at birth is safe and effective although the serologic response to the vaccine is somewhat less compared to that attained in older infants. Since for 10-40% of infants whose only opportunity for immunization is at birth, a single dose of OPV will offer some protection (52). BCG at birth protects against severe forms of tuberculosis, however tuberculosis control depends on case management and adequate therapy of infectious cases.

Effectiveness has been assessed via two indicators: immunization coverage and incidence of immunizable disease (based on reporting of diseases, and where there is no reporting, on survey data). Coverage data has been reviewed above. Combining coverage data with disease incidence curves, the effectiveness of immunization can be appreciated especially when high coverage is achieved or is sufficient to produce herd immunity, as shown in FIGURES 3 through 6: 1) Figure 3: Annual reported morbidity due to poliomyelitis, 1969-1984 in the Americas (42); 2) Figure 4: Indonesia Diphtheria mortality rates, 1978-1982 (53)[0-4 yr olds were targeted for immunization]; 3) Figure 5: Immunization Coverage and Incidence of Immunizable Diseases for Selected Developing Countries, 1974-1984 (54); and 4) Figure 6: Measles in Rwanda CCCD data (48).

**Reduction of Mortality:** With immunization coverage rates reported by June 1987, WHO estimates that one million deaths have been prevented in developing countries excluding China (42). The estimated impact of the EPI program as of 1987 is shown in more detail below. The figures are based on data reported by countries to the WHO and are a combination of data on coverage, incidence and case fatality rates to yield an estimate of deaths averted. The numbers of cases and deaths are not actual counts, but estimates, and are very sensitive to the case fatality rate used (personal communication with M Grabowsky, October 1988).

As of July 1988, the estimated number of cases prevented by poliomyelitis immunization in developing countries was increased to 217,000 (China excluded) (49). Likewise the estimated number of measles deaths per year prevented by immunization in developing countries went up to 978,000, for neonatal tetanus up to 248,000 and for pertussis up to 356,000(49).

The impact of measles vaccination on childhood mortality in high risk children has been disputed in the past because it was felt that replacement mortality would override the beneficial effect of vaccination (43). However, recent studies have demonstrated that measles vaccination appears to reduce mortality by a larger percentage than that expected from simply a reduction in measles deaths (43). For example, the risk of mortality in immunized children between 6 months and 8 months of age was 3% compared to 40% among non-immunized children in Guinea-Bissau (55). In Haiti, unimmunized children who had a birth interval less than 24 months to their younger sibling, came from lower SES, and had illiterate mothers had a 0.678 probability of being alive at age 39 months compared to 0.932 for measles-immunized children who shared the above profile (56).

The incidence of neonatal tetanus as well as neonatal mortality can also be effectively reduced by training traditional birth attendants (neonatal mortality reduction of 70% and 65% reduction in neonatal tetanus compared to control group), although immunization is more effective in specifically reducing neonatal tetanus (57).

**Indirect Effects** of vaccination may be improved nutritional status due to a lower burden of infectious disease as immunization is an effective means of breaking the vicious cycle and synergy of infection and malnutrition. Therefore, immunization may provide benefits beyond the control of immunizable disease. Immunization against measles and pertussis protects the child from two major causes of ARI mortality. Measles immunization also reduces mortality from diarrhea or pneumonia that frequently is associated with post-measles syndrome. Feachem estimates that measles vaccination may decrease diarrheal incidence by 2.2% and associated mortality by 16% (58).

The process of setting up the immunization infrastructure, from supplying and equipping the cold chain to training personnel, may serve as a foundation for a more comprehensive health service infrastructure, and a conduit for other supplies and services.

#### **D. Implementation Problems**

All vaccination programs require the maintenance of a cold chain because of the heat lability of vaccines. Maintaining the cold chain in the face of equipment failure and frequent power failures represents a major implementation problem. Reuse of needles and syringes to conserve resources has carried the risk of transmission of HIV and hepatitis B. In a sampling of fixed health centers in Africa, it was found that 58% of these centers

have used the same syringe for multiple injections (44). Live virus vaccination in potentially immunosuppressed individuals suffering from HIV infection has raised safety issues.

Management of the cold chain and supply of vaccines requires constant attention, training and supervision. Unreliable supply of vaccine may be a contributing factor to lack of community support and CHW loss of enthusiasm. The logistics of vaccine delivery are compounded by the fact that up to 80% of the population in developing countries live in difficult to reach areas (44). Reaching high risk groups in poverty areas and urban slums is confounded by social disorganization, lack of addresses, clinic registration, or community organization, and limited means whereby to mobilize even mass campaigns. Reluctance to immunize children with minor illness or malnutrition continues to be a problem; 50-80% of children seen in outpatient departments are in need of immunizations but do not receive them (42). Withholding immunization because of minor illness may be one of many factor contributing to the high dropout rates between the first and subsequent vaccine doses. The percent of children not returning for second and third doses of oral polio vaccine have been in the range of 20 to 40% for polio 2 and 50-70% for polio 3 in Guatemala and Liberia, according to DHS survey results (9).

Community acceptance still presents a problem, as many misconceptions and fears may be associated with injections. Adverse reactions to the first immunization with DPT, such as fever, may be misconstrued by the parent and lead to further mistrust. Fear of being infected with HIV may be a justifiable fear if needles and syringes are reused. Again, the opportunity costs of clinic visits or waits associated with vaccination campaigns may be a contributing factor to non-participation in a community.

From the donor or policy maker's perspective, the short-term, high visibility efforts are easier to support than longer term efforts (42). Long-term financial support to ensure sustainability of routine operations is lacking and mechanisms for dealing with recurrent costs are needed.

#### E. Future Prospects

For improving implementation of existing interventions: Because exposure to measles infection in some areas can occur before 9 months of age, measles immunization at 6 months of age with a booster at 9 months or earlier has been recommended. The Edmonston-Zagreb measles vaccine may immunize at 4-5 months of age (59), and may eliminate the need for a booster dose at 9 months. Clinical efficacy trials of this vaccine given before 9 months are needed(43).

Improvements in the immunogenicity of IPV (Salk injectable vaccine) against all three serotypes of the polio virus have rekindled the debate concerning the use of killed versus live attenuated polio vaccine. IPV requires two doses, at 3 and 6 months of age, can be given with DPT and is more heat stable than Sabin oral polio vaccine (OPV). However, its greater expense currently limits its widespread use. Development of an acellular pertussis vaccine may eliminate the side effects and adverse reactions experienced with the current vaccine.

For better controlling the problem: Strategies to improve coverage of difficult to access populations include i) the need to focus on every opportunity to immunize eligibles, such as vaccinating all susceptible children attending a health facility for other

reasons or being hospitalized, 2) offering immunizations at health facilities at times convenient for child caretakers, 3) opening multidose vials even if only one child is present for immunization, 4) limiting the contraindications to immunization as has been recently recommended by the WHO (60), 5) targeting malnourished children identified through other programs 6) educating the community on the benefits of immunization, 7) rallying political and community support, 8) channeling whereby members of the community are involved in the identification of susceptible and non-immunized children, 9) involving traditional healers and birth attendants, 10) providing outreach services and 11) using pulse immunization strategies (44).

Since an estimated 70% of children receive at least one dose (44), strategies focused on decreasing the attrition rates between first and subsequent doses would increase coverage. Area specific research on the reasons for dropout after first immunization may lead to new strategies designed to reduce the drop-out rate. Improving the impact of immunization may be possible by administering vitamin A at the time of vaccination, because vitamin A may have an adjuvancy effect. This is currently being explored (61).

The development of several new vaccines may lead to an expanding scope of diseases preventable through EPI, as well as an increased workload, change in schedules, etc. Some of the potential new vaccines have been mentioned in the CDD and ARI section. The Institute of Medicine has ranked diseases by their total disease burden and prioritized vaccines for development into three categories: high, intermediate and low as follows:

Improvements in primary health system infrastructure, training and management would improve the efficiency of the EPI program. In many areas, the support and trust of the community has yet to be gained, and efforts in community education, involvement and social mobilization may enhance the acceptance of immunization.

For achieving sustainable health improvements, vaccination should be integrated into PHC, which will require better management, training, supervision, and coordination with other PHC activities. Technical advances in cold chain to overcome power failures may facilitate operations, however alternative energy sources such as solar energy units would still require maintenance. In order to achieve financial sustainability, mechanisms for covering the recurrent costs of maintaining cold chain and EPI activities could include the following: user financing schemes such as the Bamako Initiative (1), self help measures, building recurrent costs into the programs, health insurance schemes, taxation, redistribution of health budgets to decrease the proportion spent on curative services in urban areas (44).

#### IV. MALARIA

##### A. Rationale for the Intervention

Disease burden: Malaria is endemic in areas inhabited by nearly 2200 million people, and half the world's population is at some risk of malaria (61) (see FIGURE 7). Given the magnitude of the problem, reliable data on the incidence of malaria are difficult to compile. Estimates range from 100 million to 300 million new clinical cases each year (all ages included) (63). It appears that the incidence of disease following the eradication era has either not changed or has increased. Based on cases reported to WHO (5-10

million), resurgence of the disease (2.3 fold increase in prevalence) (64) peaked in 1976, and then fell to pre-resurgence levels by 1982 (65). However, in some areas rates of disease continue to increase. For example, according to CCCD data, the incidence of malaria morbidity has quadrupled between 1980 and 1987 in Rwanda (48).

Several reports of increases in malaria mortality can be cited. For example, case fatality rates at 35 sentinel sites in Zaire have increased from 2 per 100 cases in 1984 to 5 per 100 cases in 1987 (48). Young children, as well as pregnant women, are especially susceptible to malaria particularly in *Plasmodium falciparum* endemic areas. An estimated one million infants and children die each year of "malaria associated with nutritional and other health problems" (66). Case fatality for the under five group is estimated to be 3.5% (4). Even though newborn infants are partially protected for the first few months of life by passively acquired maternal IgG, it has been presumed that all infants in highly endemic areas and 20% of infants in other endemic areas become infected in the first year of life (4).

The prevalence of malaria in sub Saharan Africa, the largest endemic focus, is believed to be unchanged over the past 10 years. Of the 421 million that live south of the Sahara, approximately 373 million live in malarious areas, and of these an estimated 224 million live in highly endemic areas. The estimates of the number of clinical cases range from 76 and 150 million in this area of the world (66). In Malawi, malaria deaths have steadily accounted for between 12 and 14% of the deaths among under five admissions to hospital from 1980 -1986, compared to 8-10% for deaths due to diarrhea over the same time period (48). Community studies in the Gambia have demonstrated that 15-25% of deaths among under fives were due to malaria (67).

Purpose of the intervention, in terms of **disease control**, is to prevent malaria mortality via a case management approach and prophylaxis of high risk groups that ideally is built into the primary health care system. In terms of **vector control**, the purpose is to prevent transmission of malaria via measures that will vary from region to region depending on the ecology of the predominant vector species. For example, in SE Asia and China, cutting back bush around villages will destroy larval habitats of an exophilic species of mosquito, *Anopheles dirus* and *A. baiabacensis* (63). Indoor spraying of residual pesticides will not affect exophilic (outdoor biting) mosquitos, so this is a strategy that would not be effective in areas where exophilic species predominate.

## B. Implementation

The model program: Disease control is comprised of case management using appropriate antimalarials, health education, surveillance systems for monitoring outbreaks and drug sensitivity. Chemoprophylaxis is recommended by the WHO only for pregnant women, although other sources advocate the use of chemoprophylaxis for small children, immigrants, laborers and non-immune visitors.

Vector control is comprised of those interventions that interrupt transmission: bed netting, window screens, insecticide spraying, drainage of breeding areas, larvicidal measures, construction of houses distant from breeding places, and siting domestic animal shelters between housing and breeding places so that mosquitos are diverted to animals (personal communication with David Clyde, October 1988). One of the main lessons learned from the "eradication era" was that malaria varies from one geographical area to

another, and that control efforts need to be tailor-made to the particulars of ecologic and socioeconomic environments.

The model program for malaria has been called 'integrated control' which implies stratification of the control measures used according to the main problems of the area in question. Usually, this results in a combination of residual insecticide spraying, larvicidal measures, and case management. Coverage of 90% or more of the households in a community is necessary for residual insecticide spraying to be effective.

### C. Implementation Outcomes

Efficacy of antimalarials depends on sensitivity of various parasite strains, severity of the illness (cerebral malaria), the subject's immune status (immigrants and young infants being considered malaria naive hosts, pregnant women have their immunity to malaria decreased) and compliance with prescribed dosage regimens.

Effectiveness: Recent malaria disease control efforts demonstrated that community-based treatment of clinical malaria and maloprim chemoprophylaxis of under fives in The Gambia reduced overall mortality, malaria-associated mortality and febrile episodes with parasitemia (68), as shown in TABLE 9. Treatment of clinical malaria alone compared to treatment plus chemoprophylaxis did not have any effect on mortality, a finding similar to studies done evaluating the Rural Health Development Programme in Saradidi, Kenya (69).

Whereas untreated mosquito nets do not appear to be effective in reducing the incidence of malaria under fives (70), permethrin treated bed nets are (71, 72). Morbidity surveillance of under fives in The Gambia revealed significantly ( $p < 0.05$ ) fewer febrile episodes with parasitemia in the permethrin treated bed net group (23) versus the placebo treated bed net group (34). A similar comparison of children in villages with and without treated bed nets in Papua New Guinea revealed a reduced incidence of malaria in 0-4 year olds, but not for older children (72). The main advantages of insecticide treated nets are that they are relatively inexpensive (US\$0.50 per treatment of net), retain their activity for 6-12 months, reduce malaria transmission by increasing vector mortality and decreasing contact with the host, and are popular because permethrin kills head lice and bed bugs as well (72). Bed nets in China cost the equivalent of US\$2.00 and last five years, versus DDT spraying which is 2.5 times more expensive than the use of impregnated bed nets. Repellents are effective at decreasing contact with biting insects but may not be effective in reducing disease transmission (63).

### D. Implementation Problems

The most threatening technical problems revolve around parasite and vector resistance. The development of parasite resistance to drugs is due to use of subcurative doses, and is most rapid when there are no vector controls in operation and when there is widespread use of a specific drug (65). The spread of chloroquine-resistant malaria is shown in FIGURE 8.

There are frequently more side effects of alternative drugs, which also require a longer treatment period and are more costly than chloroquine. Treatment of severe malarial anemia with transfusions has become problematic since blood screening for HIV is not available (73).

By 1983, 57 species of anopheles mosquitos were resistant to one or more insecticides, which undermined malaria control programs in one third of the 62 countries that had a program (66). Vector resistance is considered to be one of the contributing factors to the resurgence of malaria in the southeast Asia region (less so in Africa since disease control measures were prioritized). In 1984, about 33% of SE Asians at risk for malaria were protected by extensive spraying (74). Even if insecticide resistance did not occur, the outdoor habitats of some Anopheles species and the increased outdoor night time activities of human beings (communal TV's, etc.) provide for increased transmission of malaria.

Animal and human toxicity of insecticides in the past was frequently overlooked, until the environmental and animal toxicity of DDT became apparent. The advantage of DDT was that it was very toxic to mosquitos, and its effect lasted for six months whereas the duration of effect of other insecticides (malathion, dieldrin) is shorter, and is more toxic to animals and humans than DDT (66). Malathion, ten times more expensive than DDT, is toxic to humans (neurotoxin) whereas DDT is an environmental contaminant. The insecticide propoxur, used currently in Latin America and South America, is 39 times the cost of DDT and much more toxic to humans and animals (personal communication with David Clyde, October 1988).

It is logistically difficult for spray teams to reach more remote areas during the monsoon. Since seasonal transmission is tied to the wet seasons, access to these areas at these times would increase the effectiveness of malaria control.

In terms of the health system, integrating malaria control efforts and PHC activities has been problematic in terms of management, retraining and coordinating specialized malaria activities. Adding the specialized activity of either malaria disease or vector control to existing CHW or PHC systems often resulted in overburdening them.

Community obstacles to malaria control include the fact that many householders object to the smell of organophosphorus insecticides; malathion, in particular, smells bad. 60% of households in the Philippines reject insecticide spraying of their homes on account of the odor or because it involves intrusion into their homes by strange men (personal communication David Clyde, October 1988). Compliance with medication is low in some communities due to the bitter taste of chloroquine. Recent trials of coated chloroquine in Malawi have shown these tablets to be more acceptable (personal communication with Deborah Helitzer-Allen, October 1988).

The costs of initiating and sustaining an effective malaria control program has been a major implementation problem, that has become progressively worse with the increasing development of parasite and vector resistance. Premature withdrawal of international or bilateral assistance may have contributed to the deterioration of antimalarial programs in the 1970s and early 80s (66).

#### E. Future Prospects

For improving implementation of existing interventions, there must be a stratification of control efforts, with a combination of disease control and ecologically appropriate vector control measures, and integration into PHC. China and Thailand provide good examples of effective malaria control integrated into PHC (66). In Thailand, malaria control is carried out by mobile teams that include a medical officer and

microscopist, fixed clinic centers and insecticide spraying. In China, malaria control has been achieved via residual spraying (DDT), larvicidal measures, and bed nets. Interestingly, China's malaria control program was built into an existing PHC infrastructure, whereas Thailand's malaria vertical control program served as a foundation for the development of more comprehensive PHC. Since multipurpose worker may be overburdened with both malaria vector and disease control activities added to other PHC activities, it may be more practical to train a semi-specialized worker in the community to take responsibility for vector control activities and have the FHC provider take over disease control activities (personal communication with David Clyde, October 1988).

For better controlling malaria, the following intervention modifications could be made or added:

1) Spraying of insecticides would be more accepted by the community if it was done by members of the community. The spray containers now are small and light enough that spraying can be done by women and even school children.

2) Use of polystyrene beads to cover breeding sites have been in limited use in suitable foci in urban areas in India against A.stephansi, and have been tested in Tanzania (74). These expand and float on surface of water and are fatal to mosquito larvae and prevent egg laying. These layers may persist for years and may be best utilized in pit latrines, wells and water tanks.

3) Enhancing the cultural relevance of malaria education efforts, e.g., focus on the child at risk as opposed to mosquito images on health education posters as was done in Singapore (63).

Efforts in malaria vaccine development have been hindered by the parasite's ability to evade host immunity. Researchers have to do better than nature in stimulating solid immunity, since even the natural immunity that develops in hosts exposed to malaria for their entire lives is still not protective. Hopes for increased immunogenicity have led to trials of vaccines based on a conjugated synthetic circumsporozoite peptide. A transmission blocking vaccine being developed in Sri Lanka for P.vivax has potential community health value, but would not protect the vaccine recipient from disease. Prospects for a vaccine are currently rather remote (75).

For achieving sustainable health improvements, the following measures have been suggested: 1) housing and bedding modifications, 2) linking vector control to social and economic development i.e. raising fish in rice fields as a means of reducing larval populations of mosquitos, as was done in Indonesia, 3) community tree farming, of eucalyptus trees that soak up a lot of water [Income from fish cultures and forests provide an incentive for continued participation] (63), 4)landfilling, drain construction, improved sanitation.

Biologic controls were being developed and utilized prior to the introduction of DDT. With the development of insecticide resistance, some of these methods are being rediscovered (66): 1) larvicides, e.g., Temephos (Abate) layers out on the surface of water, has a long duration of effect and less toxic than DDT; 2) Bacillus thuringiensis is a commercially available bacterium that produces a protein that is toxic on ingestion to Anopheles larvae; 3) larvivorous fish, other predators of mosquitos; 4) release of sterile male mosquitos, limited by ability to produce large numbers of such mosquitos.

## V. NUTRITION

### A. Rationale for the Intervention

**Disease Burden:** According to UNICEF, malnutrition affects 40% of all children in developing countries and contributes directly or indirectly to an estimated 60% of all child deaths (1). A similar figure, six million or 45 % of all under five deaths, has been derived based on an average relative risk of mortality of 2.5 for mild/moderate protein-energy malnutrition (PEM) and 7.2 for severe PEM (personal communication with Kenneth Brown and D. Schroeder, September 1988). This average has been compiled from the following relative risks of mortality by nutritional status as determined in the following studies:

ESTIMATED RELATIVE RISKS OF MORTALITY BY NUTRITIONAL STATUS  
WEIGHT-FOR-AGE

Study	Ages (mos)	Duration of Study	Nutritional Status Category Mild/Mod PEM	Severe PEM
Kielman (76)	1-36	12	4.4 (60-79%/>80%)	10 (<60%/>75%)
Chen (77)	13-23	24	1.15 (60-74%/>75%)	3 (<60%/>75%)
Heywood (78)	6-30	24	1.8 (81-90%/>100%)	8.5 (<60%/81-90%)

The Kasongo Project team in Zaire found a weaker relationship between nutritional status (weight-for-age) and mortality, the relative risk for mild/moderate PEM was 1.8 (<80/>80%) and for severe PEM 3.3 (<60/>60%) (79). However, the age range for this study was broader including children from 6-59 months of age, which possibly diluted the relationship between malnutrition and mortality. Survival curves of children in Guinea-Bissau level out at age 48 months, suggesting that children under three years of age should be the priority group for growth monitoring projects as opposed to children under five years (80). Besides PEM, iron, iodine and vitamin A deficiency are felt to be widespread, and are linked to decreased growth and development and increased morbidity and mortality (81). Reliable estimates are lacking for the global scope of these problems.

**Purpose** of nutrition interventions are 1) to prevent low birth weight by improving maternal health and nutrition during pregnancy, and 2) to prevent childhood malnutrition by promoting breastfeeding and maternal nutritional status during lactation, improving weaning practices (appropriate time to initiate weaning, balanced diet, hygienically prepared foods), feeding during illness, and monitoring growth. Growth monitoring (GM) can function as an early warning device if the detection of growth faltering leads to prompt and appropriate nutritional intervention.

### B. Implementation

**The model program** is comprised of nutrition education, often centered around the growth monitoring session. More than 200 different types of growth charts are used in over 80 countries (82). GM is a tool to ideally detect lack of weight gain in two consecutive months, and lead to effective and age appropriate intervention (e.g. increased

frequency of breastfeeding for an under three month old or increased frequency of weaning foods for a six month old). Food and other nutrient supplementation of diet is frequently a component of nutrition programs, especially for pregnant women participating in prenatal programs. Vitamin A and/or iodine are supplemented according to regional needs, either directly or via fortification.

### C. Implementation Outcomes

**Efficacy:** Dietary management of diarrhea program entails the continued feeding and proper choice of foods for feeding during diarrhea to overcome the depleted caloric intake and temporary food intolerance associated with diarrhea (83,15). The efficacy of continued oral feeding during diarrhea has been studied in a controlled clinical setting in Peru. Children receiving full strength formula, comprised of casein, sucrose, dextrin with maltose and vegetable oil, maintained a continuously positive trend in weight gain during the diarrheal episode. Children who received half strength formula, ORS and/or intravenous therapy lost weight until the day after they too were started on full strength formula. No adverse effect of feeding on the duration or severity of diarrhea was found. Children who received formula from the beginning of their diarrheal episode gained approximately 140 g more than those who did not receive formula (84).

There is strong evidence supporting an inverse relationship between breastfeeding and overall mortality (85), and between breastfeeding and diarrheal morbidity (25) and mortality (85). A case control study of infant mortality in Brazil demonstrated that, after adjustment for confounding variables, completely weaned infants had 14.2 times the risk of death from diarrhea and 3.6 times the risk of death from respiratory infections than breastfed infants (86). Breastfeeding affects child survival not only through its nutritional impact and anti-infective properties but also through its birthspacing effect (87). Large scale attempts to promote breastfeeding in Egypt, Belize and El Salvador have been successful through policy changes in hospitals and ministries of health, education of health professionals and support for breastfeeding mothers (88).

Interventions designed to improve the preparation of weaning food include chemical and physical alterations to reduce the potential for pathogen multiplication during storage, and education regarding the cooking, reheating and storage of foods, domestic hygiene (27) and proper cleaning of feeding bottles. It is unlikely that any combination of factors will permit holding prepared weaning foods for longer periods without refrigeration, which implies that these foods require reheating or fresh preparation each time. Weaning foods that can be conveniently and hygienically prepared and at the same time provide optimal nutrient content would be the ideal (89). Weaning education can be effective in improving nutritional status and could reduce diarrheal mortality rates by 2-12% (90).

The effectiveness of growth monitoring programs has been difficult to evaluate since they are usually not isolated activities, and other services are usually provided at clinic sites. The indicators used to evaluate such programs include knowledge, attitude and practice (KAP) assessment and nutritional status.

Nutritional supplementation during pregnancy in controlled trials has resulted in small increases in birthweight (an average of < 100 grams). It is unrealistic to expect that nutritional supplementation during pregnancy can reverse the chronic malnutrition often

found in women in LDC's. In a longitudinal study done in Guatemala, nutritional supplementation during two consecutive pregnancies (>20,000 kcal) and interim lactational period (>40,000 kcal) increased the birthweight of the second offspring by an average of 300 grams compared to offspring of women receiving lesser caloric supplementation (91). While this study was somewhat biased due to self selection of the mothers that participated in the study, the authors report that these women came from the poorest and least educated segment of the study villages. Also the effect of supplementation was still seen after adjusting for birthweight of the first offspring, which would have controlled for other biologic and socioeconomic factors affecting fetal growth.

Indirect Effects: Malnutrition has been shown to be a predisposing factor for diarrhea, both for increased duration and case-fatality (17) as well as increased incidence of repeated episodes (18,19,20). Malnutrition is also a risk factor for ALRI mortality. Interventions that reduce the prevalence of malnutrition may indirectly reduce diarrheal disease and ALRI mortality, however the magnitude of this indirect effect would be difficult to estimate. Repeated infections are also a risk factor for malnutrition, and efforts designed to control the number and severity of infections may indirectly reduce the prevalence of malnutrition.

The overall impact of illness on weight gain, as determined by a prospective study in the Sudan, is 160 gram reduction in growth of 3 to 6 month old infants for a diarrheal episode, vs 95 grams for a cough and/or cold episode (20).

Since vitamin A deficiency in children has been associated with an increased risk of diarrheal and respiratory infection as well as an increased risk of mortality, an intervention that reversed vitamin A deficiency in a community may be expected to have an indirect effect on morbidity and mortality. Results from a field trial in Sumatra indicate that mortality rates are reduced by 30% among children under five years in the communities that received supplemental vitamin A (200,000 I.U. retinol palmitate every six months) (92). A randomized clinical trial of vitamin A supplementation of children admitted to hospital for measles resulted in a decreased mortality among the supplemented group (6 deaths/88 or 7%) vs the control group (12 deaths/92 or 13%) (93). However, the magnitude of the effect of vitamin A on mortality in uncontrolled settings is uncertain. The role of vitamin A supplementation in reducing childhood morbidity and mortality awaits further efficacy and effectiveness field trials (94).

#### D. Implementation Problems

Nutritional programs are curtailed by food unavailability, cultural practices, anorexia during illness and lack of nutrient dense, inexpensive and convenient weaning foods. Additionally, CHW's that implement growth monitoring are either volunteers (usually very young), in which case there is a high turnover, or are overburdened with other PHC responsibilities, in which case growth monitoring reverts into the mechanical activity of weighing the child and not an interactive session that is beneficial to the caretaker of the child. In these ways, growth monitoring loses its preventive function, and takes on a curative focus (95). Interventions are then made only for those children who are moderately malnourished, when it is perhaps too late to significantly curtail that child's risk of mortality. At the root of the problem may be inadequate training and compensation for CHW's. While malnutrition has been recognized as a predictor of

increased mortality risk, the risks of failing to gain weight are less clear. Resources available for intervention are limited and the impact of the different interventions on mortality are currently under study (96).

From the community standpoint, maternal health, nutritional status, education and workload are important factors in a mother's ability to care for the nutritional needs of her child, and these factors are easily overlooked in nutrition programs focused on the child. Mother's ability to implement the advice she receives from a CHW is frequently limited by other demands on her time (97). Mothers who work away from the home will not be able to breastfeed for longer durations, etc. Additionally, time spent in queues waiting for child weighing is a large disincentive for participating in GM projects, and discourages return visits. Supplemental food is often the lure for community participation in growth monitoring, and this is a costly aspect of a nutrition program.

Growth monitoring concretizes the growth of a child for those that can grasp the significance of a graph, growth chart or "road to health". However, even if the GM interaction is a positive one, the returns on implementing nutritional advice are slow to materialize and therefore less tangible, and involve more of a leap of faith for the individual mother. Additionally, the other constraints on a mother's time and resources may limit her ability to implement the advice she receives.

#### E. Future Prospects

For improving implementation of existing interventions, better integration with other programs, e.g. EPI, vitamin A distribution, and iodine deficiency control is necessary (61). Control of diarrhea due to E.Coli, Shigella and possibly other agents would improve the growth of children and therefore reduce the prevalence of PEM (16). Feeding back GM information to the community is a way of concretizing the "progress" the children of the community are making, and may serve as a stimulus for action and participation.

For better controlling the problem of malnutrition, it is necessary to reduce food borne diarrhea via the promotion of proper weaning practices and food hygiene, which includes sanitary preparation and storage of weaning foods. Selection of nutrient dense locally available weaning foods and feeding during illness and in recovery period to optimize "catch-up" growth are likely to have a significant impact if well integrated into community practices.

For achieving sustainable health improvements: Development efforts in agriculture and income generating projects help achieve sustainable nutritional improvements. The introduction of a more productive crop, such as that which converted India from a rice importer to a rice exporter, can help alleviate food unavailability. Socioeconomic development with more equitable distribution of goods and services is most likely to have the greatest impact on nutritional status.

## VI. CONCLUSIONS

Emphasis on the impact direct interventions have on mortality as separate entities loses sight of the interaction of risk factors for mortality. The benefits conferred by one intervention are quickly lost when a child succumbs to another disease for which no intervention was offered. The Narangwal studies demonstrated the impact of integrated

PHC services offered at the same point in time. Additionally, the implementation of the direct interventions has direct bearing on their effectiveness, and therefore their potential impact on child survival.

Given the ideal of an integrated packet of effectively implemented direct interventions, how might changing conditions in LDC's modify the impact of these interventions, and how might the direct interventions be adapted to these conditions? These changing conditions might well include "environmental resource degradation (e.g., Bhopal), resurgence of malaria, diseases of lifestyle and behavior (smoking, AIDS), and chronic disease" in addition to "the old business of poverty and its associated afflictions - infection, malnutrition, unwanted high fertility" [(comments of Lincoln Chen (98)]. Underlying these themes are the familiar ones of the Third World debt crisis, political instability, corruption and least aid getting to those that need it most. The effect of the 1982-83 drought in Africa and economic recession could have overridden any impact the direct interventions might have had. The growth of periurban and urban slums may make them less accessible, and probably increase the proportion of difficult to reach, high risk profile children. Increasing population pressure may lead to decreasing water quantity and quality, scarcer resources, less fuel, poorer housing. Poor water and sanitation may increase diarrheal disease incidence. Air pollution, active and passive smoking, and crowding may increase ALRI. Outreach efforts may be more difficult to implement unless they are truly community based.

The social and economic constraints of the poor limit the effectiveness of the direct interventions especially nutrition. Visits to health center lead to economic loss (time away from gainful or subsistence activity), especially when there are long queues, inconvenient clinic hours (hours chosen to maximize health worker efficiency vs recipient convenience). Prevention is often an ideal of public health that is a luxury in reality of most villages, as most recipients of health care come to the health center only when they are sick and can afford to.

Preventive care can be resisted by health professionals as well, especially those trained in the high tech curative paradigm. The lack of demand for preventive services may further reinforce a health care worker's prioritization of curative services. The private practice of health care providers often supercedes government obligation on account of reimbursement differential, which reinforces a curative focus.

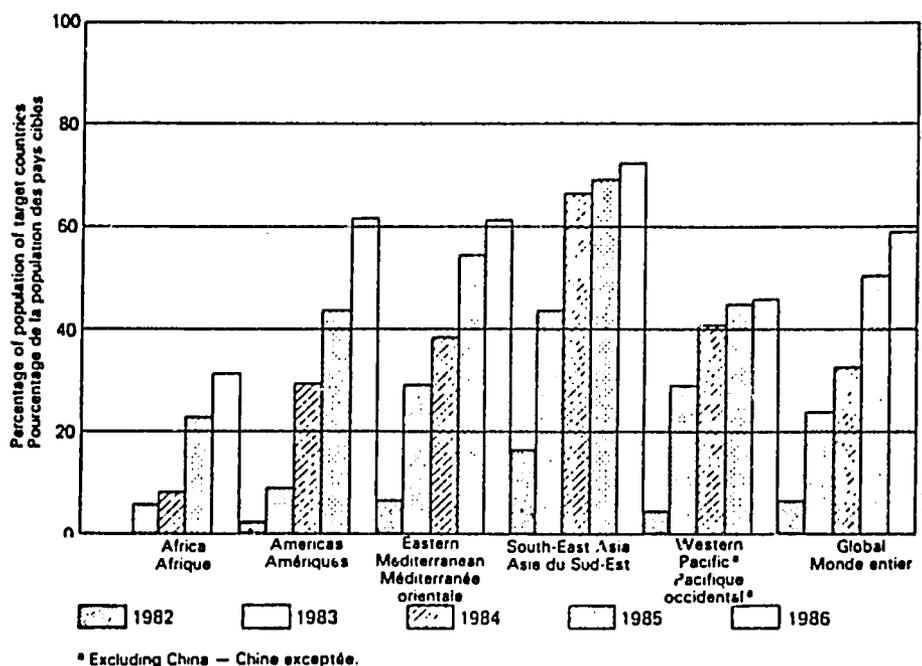
The direct interventions could be more responsive to these conditions in the following ways. Targeting of high risk populations and more innovative approaches to accessing the hard to reach populations are needed to improve the impact of the direct interventions. Making health centers more 'user friendly', culturally sensitive and convenient would aid in attendance and compliance. Compensating the CHW's for their time and effort would provide a more concrete and fairer incentive than community respect for the CHW's position. Developing health service infrastructure, i.e., better management, training and supervision, may lead toward more efficient programs that are better able to supply immunizations, ORS and antibiotics when the need arises. Tying income generating activities to malaria vector control efforts and to nutrition programs would greatly augment the participation in and the sustainability of these programs.

To increase the acceptability of the preventive paradigm and make it more culturally relevant, changes are needed in the medical curriculum in schools in LDC's.

This reorientation towards prevention and health promotion may have a long term effect on not only the major causes of childhood mortality in LDC's but also would help prepare LDC's for the era of chronic diseases and diseases of lifestyle. On the community level, the demand for preventive services could be augmented if there was some tangible result associated with these services. This requires imaginative techniques of health education and feedback of information to the community.

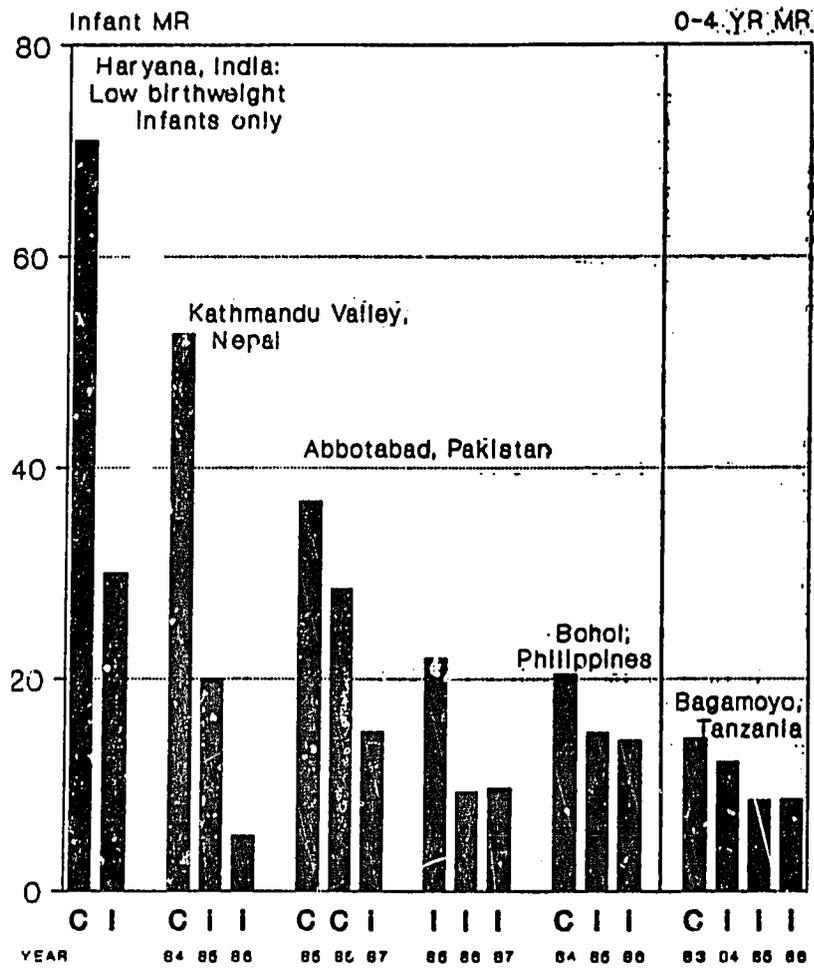
In conclusion, the direct interventions have had an impact on child survival in developing countries, and could have a greater impact if specific modifications are made to improve the effectiveness of these interventions.

FIGURE 1  
Diarrheal Disease Control: Access to ORS by WHO Region and globally, 1982 - 1986



from reference 6, p.78

**FIGURE 2**  
**Acute Lower Respiratory Infection-**  
**Specific Mortality Rates (MR)**



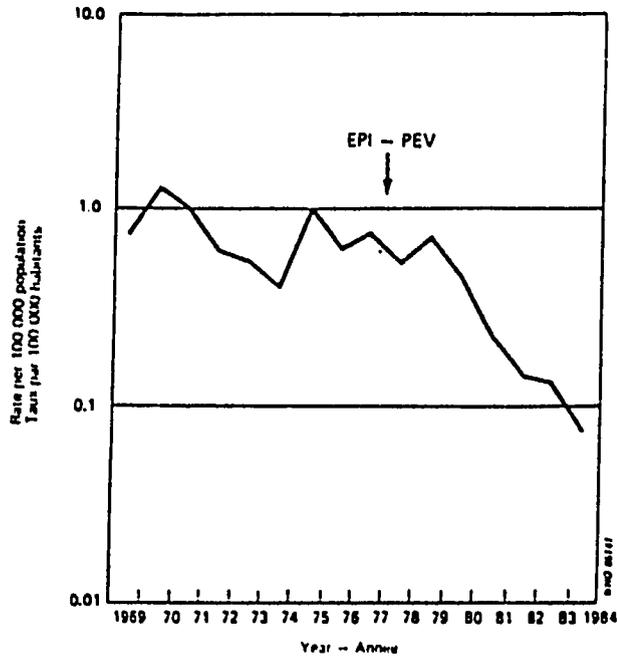
C = control area (or year)  
 I = intervention area  
 i = intervention in previous control area

\* Infant MR = deaths/ 1000 livebirths;  
 0-4 YR MR = deaths/ 1000 children <5 yr.

Figure taken from reference 34, p.14

Figure 3

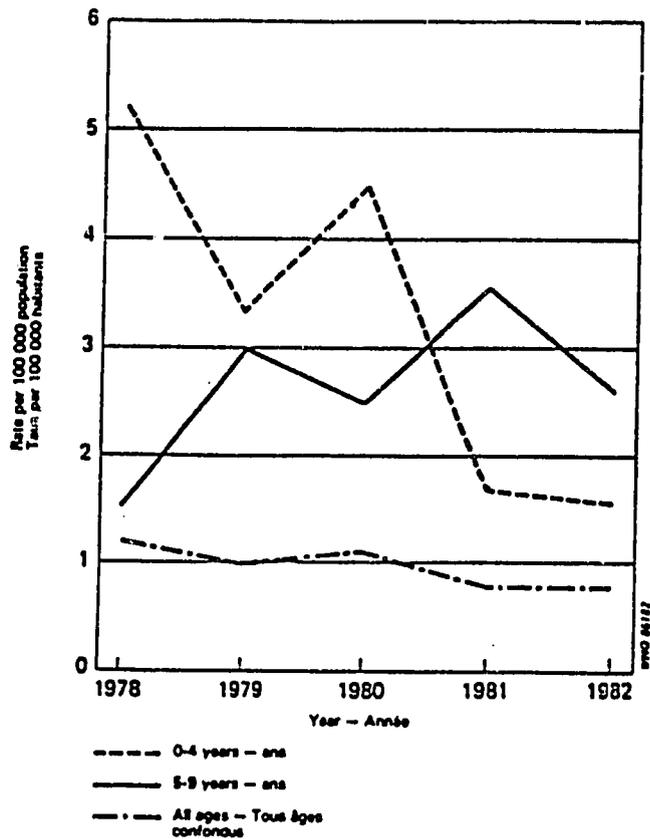
**ANNUAL REPORTED MORBIDITY DUE TO POLIOMYELITIS  
(PER 100 000 POPULATION), AMERICAS, 1969-1984**  
**INCIDENCE ANNUELLE NOTIFIÉE DE LA POLIOMYÉLITE  
(PAR 100 000 HABITANTS), AMÉRIQUES, 1969-1984**



Source: Progress report on the Expanded Programme on Immunization in the Americas, April 1985. — Rapport de situation sur le Programme élargi de vaccination dans les Amériques, avril 1985.

Figure 4

**AGE-SPECIFIC DIPHTHERIA MORBIDITY RATES, YOGYAKARTA (INDONESIA), 1978-1982**  
**TAUX DE MORBIDITÉ DIPHTÉRIQUE PAR ÂGE, YOGYAKARTA (INDONÉSIE), 1978-1982**



(Based on a report from the Ministry of Health, Indonesia — D'après un rapport du Ministère de la Santé, Indonésie.)

FIGURE 5

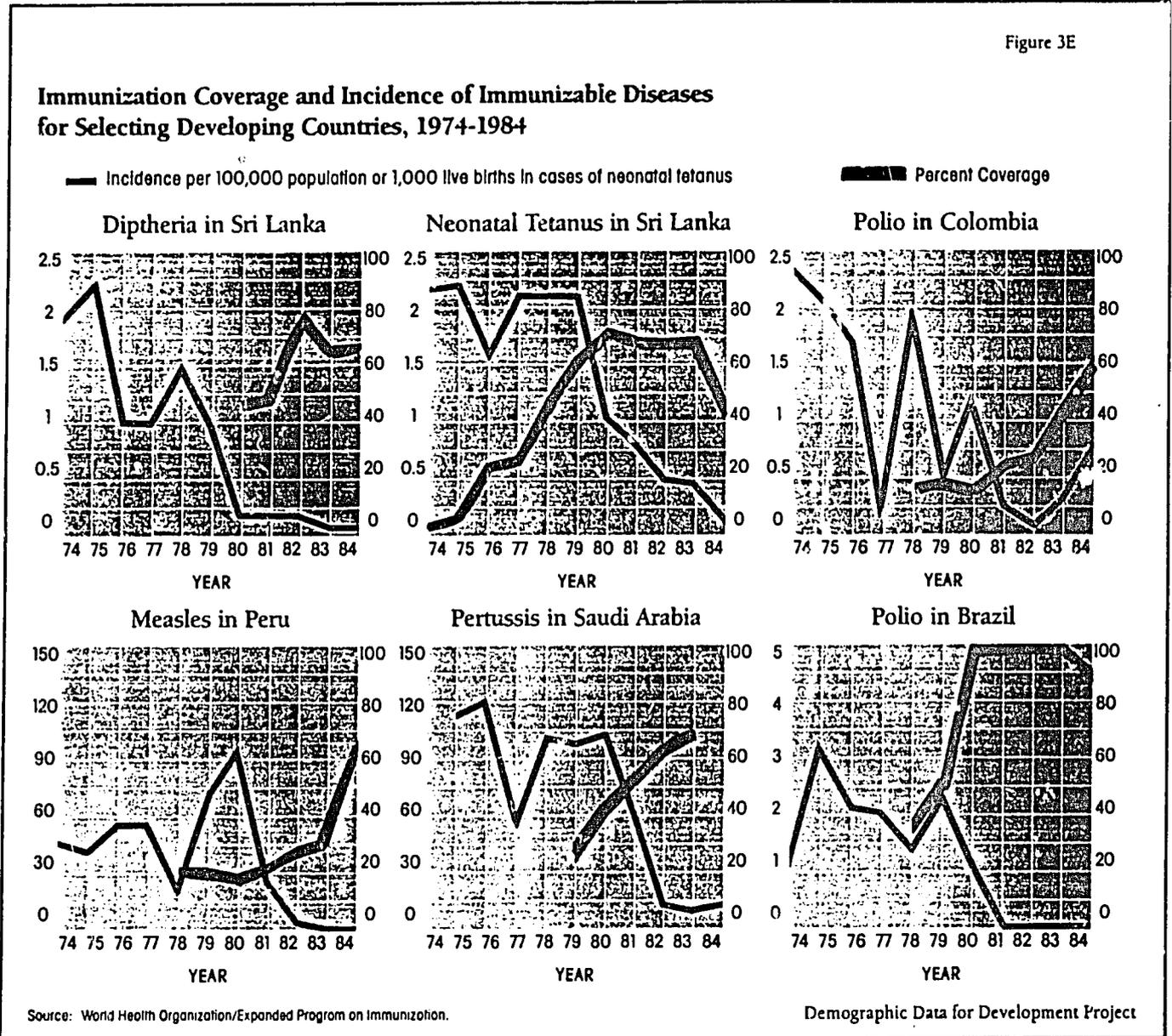
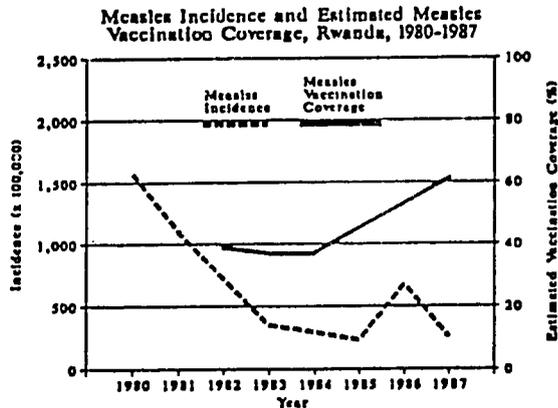


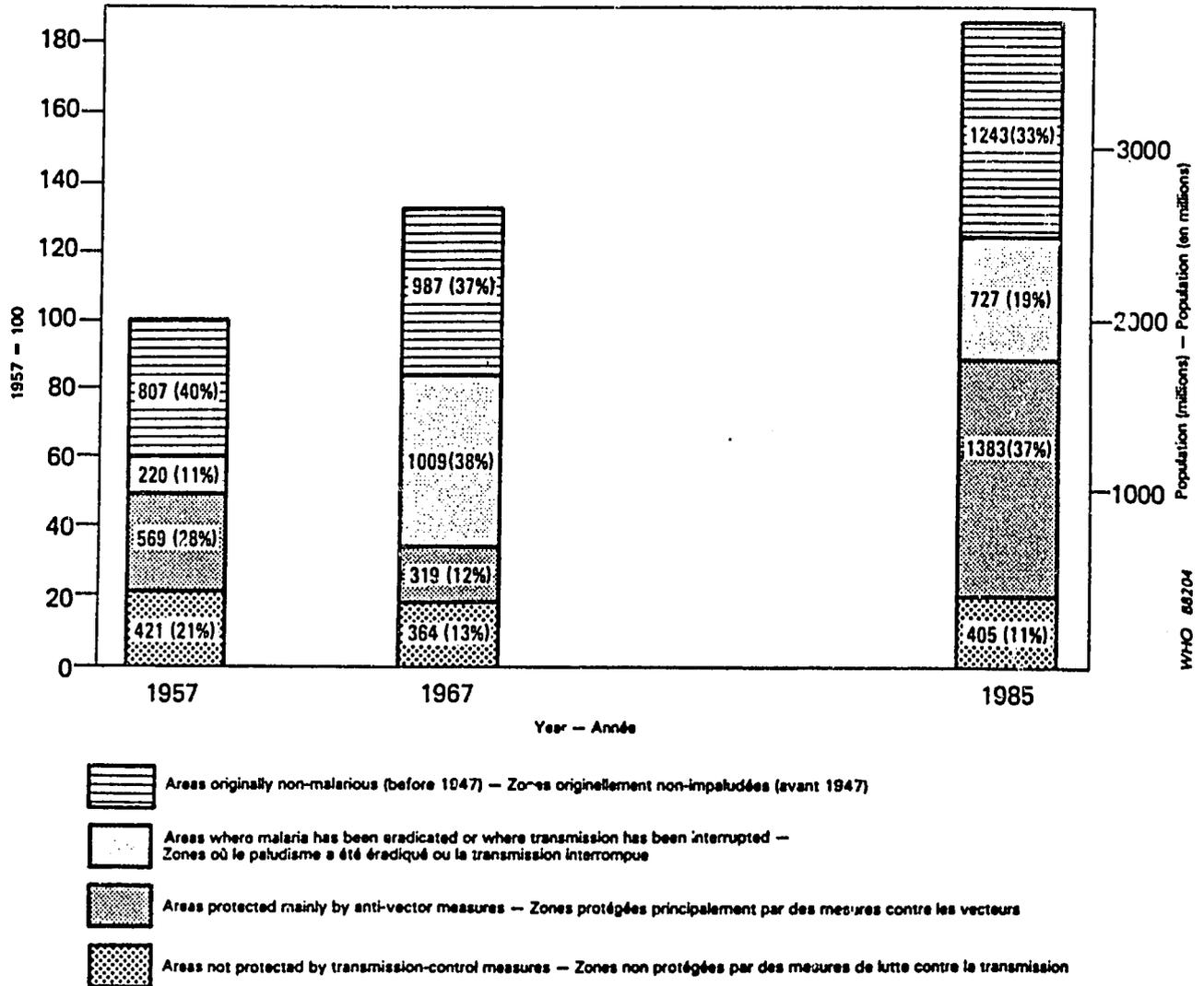
FIGURE 6



111

FIGURE 7

**GLOBAL TRENDS OF THE EPIDEMIOLOGICAL SITUATION OF MALARIA AND OF ANTI-MALARIA MEASURES  
IN TERMS OF THE POPULATIONS CONCERNED (EXCLUDING CHINA)**  
**ÉVOLUTION MONDIALE DE LA SITUATION ÉPIDÉMIOLOGIQUE DU PALUDISME ET DES MESURES  
ANTIPALUDIQUES EN TERMES DES POPULATIONS CONCERNÉES (NON-COMPRIS LA CHINE)**



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FIGURE 8

Spread of Chloroquine Resistant  
*Plasmodium falciparum*  
Malaria in Africa, 1978, 1983, 1987

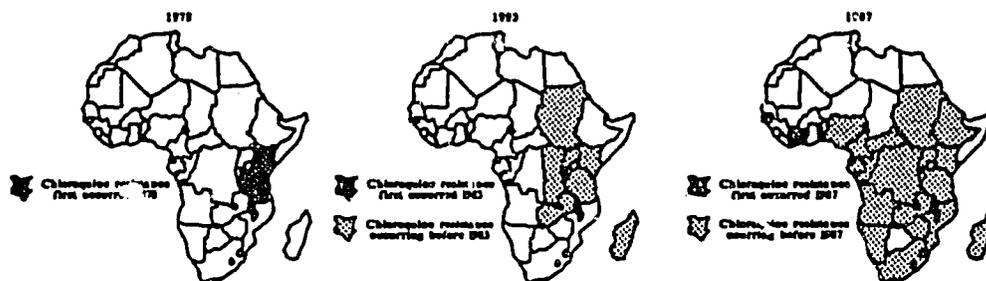


Figure taken from reference 48 p24.

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TABLE 1

Diarrheal disease control: estimated minimum use rates for  
ORS and ORT in children aged 0-4 years 1983 -1986 by WHO region and globally

Region	ORS USE RATE				ORT USE RATE		
	1983	1984	1985	1986	1984	1985	1986
AFRICA	1	3	5	7	4	8	12
AMERICAS	2	12	10	20	12	10	36
EAST MED	6	12	17	23	21	22	29
SE ASIA	9	9	12	13	14	25	26
WEST PACIFIC	9	13	20	17	27	32	29

from Wld hlth statist quart 41(2):79,1988

TABLE 2

DHS data on the percentage of children less than five years of age who have had diarrhea in the past two weeks and the percent of children treated with ORT (10) and WHO/CDD data on ORT use by countries for which DHS data is available:

Country	DHS	WHO@	
	Diarrhea* prevalence	% Rx'd w/ORT	ORT USE RATE
<b>AFRICA</b>			
Burundi (1987)	16	30	9^
Liberia (1986)	39 (4wk)	10	6^
Mali (1987)	35	2!	2^
Senegal (1986)	38	7	3^
Nigeria (1987) (DHS data from Ondo State)	5	24	18
<b>ASIA/NEAR EAST</b>			
Thailand (1987)	16	43	31
Sri Lanka (1987)	6	40	15^
Morocco (1987)	29	15	15^
<b>LATIN AMERICA</b>			
Brazil (1986)	17	12	40+
Columbia (1986)	19	42	7^
Dominican Rep (1986)	25	38	11^
El Salvador (1985)	36	26	15^
Guatemala (1987)	17	14	5.4#
Mexico (1987)	20	NA	75
Peru (1986)	32	4	5.5#
Trinidad & Tobago (1987)	6	41	56#

@ from Health Statistics Database, International Science and Technology Institute (ISTI), 10/19/88.

() year of DHS survey country report

\* % of under five year olds with diarrhea in the past two weeks, diarrhea is defined by the respondent usually the mother (DHS)

! ORS only

^ ORS and ORT reported estimates are the same

+ based on CDD household sample surveys

# midpoint between sum and greater of the ORS and sugar/salt solution rates is used as the ORT use rate

TABLE 3

Six month mortality in children aged 1-59 months in Dakahlia, Egypt

Death rates per 1000	Outreach villages		Control villages	
	1980	1986	1980	1986
Total	10.5*	6.0	18.1*	6.5
Diarrhea-associated	7.6	3.1	15.3	4.6
Non-diarrhea	2.9	2.9	2.9	1.9

\* Difference in rates between outreach and control significant  $p < 0.001$  in 1980.

Table modified from ref 13

TABLE 4

The impact of ORT on diarrheal disease admission rate and case fatality rates (CFR)

Country	Period pre-ORT post-ORT	Admission Rate (%)	Overall CFR (%)	Inpatient CFR (%)
<u>Angola</u>	1983	2.7	0.7	24.7
	1984-85	1.0**	0.1**	13.4*
<u>Indonesia</u>	1980-81	25.7	2.4	9.1
	1982-85	13.2**	0.7**	5.0*
<u>Nigeria</u> Kano:	1984	35.7	6.4	17.8
	1985	16.8**	2.1**	12.7*
Yola:	1984	43.5	5.9	13.7
	1985	2.1**	0.3**	12.8

\* signifies Chi squared value, 1 d.f. =  $p < 0.05$ \*\* signifies Chi squared value, 1 d.f. =  $p < 0.001$ 

Table modified from ref 14

TABLE 5

Summary of data from hospitals on the impact of ORT on diarrheal disease admission and case-fatality rates (CFR)

	#reports	#w/decrease	#w/increase	#w/no change	median change
Admission rate	14	13	1	-	61% reduction
Overall CFR	14	11	-	3	71% reduction
Inpatient CFR	26	11	2	13	41% reduction

Table modified from ref 14

TABLE 6

Estimated percentage of children immunized in the first year of life and percentage of pregnant women immunized against tetanus in developing countries by WHO region based on information available July 1988.

REGION	% of children immunized by 12 months of age				% of pregnant women immunized
	BCG	DPT3	Polio3	Measles	Tetanus II
African(1)	55	36	36	40	21
American(2)	60	50	77	56	0
SE Asia	47	49	43	21	38
W Pacific(3)	81	72	74	72	4
Global	59	55	58	50	17

(1) Excluding South Africa; (2) Excluding USA & Canada; (3) Excluding Australia, New Zealand, Japan

Table modified from reference 49

TABLE 7

Estimated Number of Cases and Deaths Prevented from Neonatal Tetanus, Measles, Pertussis and Poliomyelitis in Developing Countries (excluding China) June 1987.

CASES (thousands)	Neonatal			
	Tetanus	Measles	Pertussis	Polio
would have occurred w/o immunization	1,189	84,890	74,575	424
occurred w/ 1987 immunization	947	61,062	47,760	246
prevented by immunization	242	23,828	26,815	173
<u>DEATHS (thousands)</u>				
would have occurred w/o immunization	1,011	2,609	870	21
occurred w/ 1987 immunization	805	1,906	563	12
prevented by immunization	206	703	307	9

From reference 42

TABLE 8

Rankings of various vaccine candidates based on their potential benefits under a variety of assumptions and resource constraints:

HIGH	INTERMEDIATE	LOW
<u>S.pneumonia</u>	Hepatitis B	Hepatitis A
<u>Plasmodium spp.</u> (malaria)	<u>H.influenzae</u> type b	<u>N.meningitidis</u>
Rotavirus	<u>E.coli</u>	Yellow fever
<u>S.typhi</u> (Ty21a)	Sirep group A	Dengue
Shigella	<u>S.typhi</u>	Rabies(live)
	<u>M.leprae</u>	Japanese encephalitis
	<u>V.cholerae</u>	
	RSV	
	Parainfluenza	

Table modified from reference 4

TABLE 9

Overall mortality rates in children aged 1-4 years pre and post intervention in non-intervention and intervention villages in The Gambia Malaria Control Study.

<u>Study Group</u>	<b>Overall Mortality Rates (per 1000)</b>	
	<u>Preintervention</u>	<u>Postintervention</u>
Nonintervention villages	45(37/830)	49(44/904)
Placebo chemoprophylaxis and malaria case treatment	42(44/1043)	60(32/531)*
Maloprim chemoprophylaxis and malaria case treatment		27(15/545)*

\*ChiSquare = 5.9, 1d.f., p<0.01

Table modified from reference 68

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## Appendix A

### COMPILED DPT 3 IMMUNIZATION COVERAGE FOR AID EMPHASIS COUNTRIES

from references 50 and 51

COUNTRY	# of children 12-23 mos immunized if reached 80% coverage (000)	WHO# ESTIMATED COVERAGE		DHS* SURVEY COVERAGE	
		%	#	LOW %	HIGH %
<b>AFRICA</b>					
Kenya	901	75	845	-	-
Malawi	268	66	221	-	-
Mali	285	3	-	3.2	28
Niger	229	5	14	-	-
Nigeria	3688	21	968	3	14
				(Cndo State)	
Senegal	219	53	145	7	32
Sudan	723	14	127	-	-
Zaire	1028	32	411	-	-
<b>ASIA/NEAR EAST</b>					
Bangladesh	3134	5	196	-	-
Egypt	1180	80	1180	-	-
India	15926	53	10551	-	-
Indonesia	3655	48	2193	-	-
Morocco	549	53	364	32	84
Nepal	474	38	225	-	-
Pakistan	3029	56	2121	-	-
Yemen	242	11	33	-	-
<b>LATIN AMERICA</b>					
Bolivia	205	29	74	-	-
Ecuador	263	43	142	-	-
Guatemala	259	33	107	26	48
Haiti	200	19	47	-	-
Honduras	137	63	108	-	-
Peru	519	50	324	24	65

# Reference 50, based on WHO 1986 reported data

\* Low = % of children with vaccination cards x % received DPT immunization; High = % of children received DPT immunization of those who had immunization cards (assumes children without cards have same % coverage as those without cards)

## Appendix A

### COMPILED MEASLES IMMUNIZATION COVERAGE FOR AID EMPHASIS COUNTRIES

from references 50 and 51

COUNTRY	# of children 12-23 mos immunized if reached 80% coverage (000)	WHO# ESTIMATED COVERAGE		DHS* SURVEY COVERAGE	
		%	#	LOW %	HIGH %
<b>AFRICA</b>					
Kenya	901	60	676	-	-
Malawi	268	66	221	-	-
Mali	285	-	-	9.3	80
Niger	229	19	54	-	-
Nigeria	3688	32	1475	3.3	16
				(Ondo State)	
Senegal	219	70	192	13	57
Sudan	723	11	99	-	-
Zaire	1028	39	501	-	-
<b>ASIA/NEAR EAST</b>					
Bangladesh	3134	3	118	-	-
Egypt	1180	78	1150	-	-
India	15925	1	199	-	-
Indonesia	3655	47	2147	-	-
Morocco	549	48	330	30	80
Nepal	474	66	391	-	-
Pakistan	3029	41	1553	-	-
Yemen	242	13	39	-	-
<b>LATIN AMERICA</b>					
Bolivia	205	17	44	-	-
Ecuador	263	49	161	-	-
Guatemala	259	47	152	34	63
Haiti	200	21	52	-	-
Honduras	137	60	102	-	-
Peru	519	41	266	25	70

# Reference 50, based on WHO 1986 reported data

\* Low = % of children with vaccination cards x % received measles immunization; High = % of children received measles immunization of those with immunization cards (assumes children without cards have same % coverage as those without)

# APPENDIX A

## ESTIMATED IMMUNIZATION COVERAGE WITH BCG, DPT, POLIO, MEASLES AND TETANUS VACCINES BASED ON DATA AVAILABLE AS OF JULY 1988

Country	Newborns surviving to 1 year of age (millions)	Cumulative percentage of infants	Immunization coverage (%)				
			Children less than 1 year of age				Pregnant women
			BCG	DPT III	Polio III	Measles	
Developing countries ranked by surviving infants.							
1 India (7)	22.13	25	46	58	50	17	47
2 Indonesia (7)	5.05	30	68	48	45	46	26
3 Nigeria (6)	4.59	35	41	20	21	31	12
4 Bangladesh (7)	4.15	40	14	9	8	6	7
5 Brazil (6)	4.07	45	56	52	89	55	...
6 Pakistan (7)	4.03	49	72	62	62	53	27
7 Mexico (6)	2.68	52	54	34	96	60	...
8 Ethiopia (4 & 6)	1.99	54	11	6	6	10	5
9 Iran (7)	1.98	57	56	74	74	76	12
10 Philippines (6 & 7)	1.83	59	92	73	73	68	49
11 Viet Nam (7)	1.78	61	59	51	54	42	...
12 Egypt (7)	1.78	63	72	81	81	86	1
13 Thailand (7)	1.44	64	61	48	47	34	38
14 Turkey (7)	1.41	68	34	71	70	50	...
15 Zaire (6)	1.29	67	52	32	33	39	27
16 South Africa	1.28	69	...	...	...	...	...
17 Burma (7)	1.17	70	45	23	13	14	26
18 Kenya (7S)	1.13	71	86	75	75	60	37
19 Tanzania (5 & 6)	1.07	72	93	69	65	76	60
20 Rep. of Korea (6)	0.95	73	47	76	80	89	...
21 Sudan (7)	0.90	74	46	29	29	22	12
22 Algeria (7S)	0.90	75	95	66	66	59	...
23 Colombia (6)	0.88	76	69	57	65	56	...
24 Morocco (7)	0.85	77	87	78	78	76	33
25 Argentina (6)	0.75	78	89	67	79	87	...
25 countries	70.07	78	52	49	51	37	25
Other developing countries	19.56	22	62	50	50	45	20
Sub-total developing countries (excluding China)	89.63	100	54	49	51	38	24
China (7S)	19.94	18	85	75	77	77	...
Total developing countries (including China)	109.57	100	60	55	56	46	20
Total industrialized countries	18.11		59	63	71	79	...
Global Total	127.68		59	55	58	50	17

(4) 1984 coverage data

(5) 1985 coverage data

(6) 1986 coverage data

(7) 1987 coverage data

(s) survey data

... no information available

128

# Indirect Health Interventions with Reference to Family Planning and Breastfeeding

John Hobcraft<sup>1</sup>

## Introduction

The distinctions between direct and indirect child health interventions are often hard to make. There is little doubt that immunisation is a direct intervention and few would question that maternal education was an indirect health intervention. Oral rehydration therapy is widely viewed as one of the key direct interventions in reducing diarrhoeal deaths. Yet, if the wider goal of wiping out diarrhoeal disease is to be achieved, ORT is at best an indirect intervention; suitable direct interventions include personal and public hygiene measures and specific education.

The criteria for assessing the impact of direct and indirect health interventions can also differ. Many indirect interventions are broad spectrum ones, which can have an effect upon many causes of death and may well have wider implications for morbidity or health. Female education clearly falls within this category, since it can operate through many pathways to achieve better health for children. Female education can alter the extent of health knowledge, can affect access to existing services, can change personal and environmental hygiene practices, can alter feeding patterns, and can change the treatment of a variety of diseases. Immunisation against a specific disease often serves only to alter morbidity or mortality from that disease, although some synergistic interactions may occur. Is a specific intervention, such as a particular immunisation to be judged for its effectiveness by its impact on that disease alone, or on a cluster of related diseases, or by the overall reduction in child mortality, or by some even wider measure of altered health status in the society?

A proper assessment of effectiveness also requires us to estimate the costs of providing the service, which can be extremely difficult to do. Measuring the direct cost of an immunisation programme may be feasible, although proper costing of time spent by clinic staff might involve addressing issues of underemployment. However, any immunisation programme also involves opportunity costs, since the resources used might have been employed on some other health-inducing activity. How can these costs be quantified? The difficulties become even greater if we are trying to assess the costs of maternal education, or of a family planning programme. The decisions and priorities involved in expanding education may have nothing directly to do with health. Do we then regard education as a free health input? If so, it must be astonishingly cost effective.

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<sup>1</sup> London School of Economics. Paper prepared for meeting on "Child Survival Programs: Issues for the 1990s". I should like to acknowledge partial support for this work under grant number G/00232122 from the Economic and Social Research Council of the United Kingdom.

Or should we try to attribute some portion of the education budget to health interventions? How might this proportion be determined? Would it be the proportion of time spent directly on teaching health? Or is the learning of individual agency an important health input?

One of the many problems in making any such assessment is our striking lack of knowledge about the relative quantitative importance of the key pathways through which maternal education operates to reduce child mortality. We have moved on little in this respect since Caldwell's<sup>2</sup> enumeration of many of the possible pathways. Similarly, family planning programmes are often funded for reasons predominantly associated with controlling population size, although the health and human rights rationales are once again being stressed. What proportion of the family planning budget should be regarded as a health input? Does the mental health and psychological well-being of the mother feature as a relevant health output? How does this affect the apportioning of the inputs?

These are all fundamental questions which have not begun to be answered. Without some attempt to answer the questions raised in the preceding paragraphs we cannot make informed judgments about where to spend health budgets in order to achieve the greatest returns for a fixed amount of money. Straightforward assessments of such priorities are likely to be further distorted by choices of external donor agencies, which alter the true cost assessment within a country, by introducing apparently 'free' financing which is often tied to a particular intervention. A relationship between partners is called for.

A further major difficulty in assessing the impact and effectiveness of any health intervention involves the interaction of different components. Knowledge can often not be put to use without the means to access services, which can include actual availability of services, travel time and costs, foregone earnings, means to pay for use of services, and the ability to make access decisions without constraints from others. Some of these constraints form necessary preconditions for gains to result from knowledge, others act as deterrents. Similarly personal hygiene, sanitation, and clean water supply all interact in the faecal-oral transmission of diarrhoeal disease. These frequent and wide-ranging interactions are additional to the synergies among diseases, which can dramatically alter the effectiveness of particular interventions in differing environments.

It is with these complications in mind that we embark upon the difficult task of discussing the role and importance of some indirect interventions for infant and child health, particularly mortality.

### **Family Planning, including Birth Spacing.**

An indirect intervention in child (and maternal) health which is the focus of much recent attention is family planning. The probable health effects include the longer-term impact of the reduced flow of births upon the provision of health services, alterations in the social composition of births, the consequences for individual child care within the

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<sup>2</sup> J.C.Caldwell (1979) Education as a factor in mortality decline: An examination of Nigerian data. Population Studies 29:395-413. For a review of the limited progress on these issues, see H.Ware (1984) Effects of maternal education, women's roles, and child care on child mortality. In W.H.Mosley and L.C.Chen (Eds.) Child Survival: Strategies for Research (supplement to Population and Development Review Vol.10).

family, and the more direct links between family formation patterns and child survival<sup>3</sup>.

Most family planning programmes in the third world have been predominantly driven by goals of population control. As a result, their major emphasis has been upon reducing fertility, rather than upon reducing the incidence of high-risk pregnancies. Consequently, there is almost no experience of intervention with a strong health focus, for example on the avoidance of teenage births or short birth intervals. Thus, any attempt to evaluate how far existing family planning programmes have served to reduce mortality through improving family formation patterns is almost bound to understate what might be achieved.

Programmes which emphasise population control have often had a sizeable, sometimes dramatic, impact upon levels of fertility in a society. It is self-evident that a reduction in population growth through fertility control is achieved by reducing the flow of new entrants to the population. This means that the same total expenditure on maternal and child health can achieve greater inputs per birth or per child. Other things being equal, reducing the rate of growth of births from, say, three per cent per annum to one per cent per annum for a ten year period could lead to about a 22 per cent increase in health expenditure per child. Of course, other things rarely are equal and at least one possibility is that the inputs required for the successful family planning programme reduced available resources for child health; the gains might also be used elsewhere.

Reductions in fertility also have other health consequences. All serious analysts of the correlates of child mortality are aware of likely changes in population composition over time, especially when fertility levels are changing. It is not uncommon, but by no means universal, for the early stages of fertility decline to mean a relative reduction in the proportion of births to advantaged groups. This unfavourable change tends to raise overall levels of child mortality and leads to potential bias in naive analyses of excess mortality risks for high-order births. As the demographic transition reaches its final stages, it is quite common for the fertility reductions to be continuing among the disadvantaged groups after rates for the advantaged groups have stabilised; this leads to a gradually more favourable composition of births, with consequent advantages for child mortality. It is unlikely, however, that such changes in socioeconomic composition would lead to major changes in child mortality levels.

Reductions in completed family size have direct consequences for the health of the mother. Maternal mortality risks probably rise with increasing birth order, but more important is the reduced number of occasions upon which the woman is subjected to these risks. Since maternal survival is almost certainly helpful to the chances of child survival, this probably has some indirect effects upon the survival chances of children too.

Lowered individual fertility may also have an indirect impact on child survival chances and on the health and development of the child. The smaller family allows more resources to be allocated per child, including maternal care and attention, food, schooling, and costs of the access to health services. There may be other less tangible benefits from greater emotional bonding with the parents.

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<sup>3</sup> See, for example, J.Trussell, J.Potter, and J.Bongaarts (1988) Does family planning reduce infant mortality? An exchange. *Population and Development Review* 14(1):171-190. See also J.Hobcraft (1987) Does family planning save children's lives? Background Paper for International Conference on Better Health for Women and Children through Family Planning, Nairobi, 5-9 October.

## Changes in the timing of births

None of the effects of family planning programmes upon child health so far considered have paid any direct attention to the avoidance of high-risk pregnancies. Limiting family size on its own serves to reduce the incidence of births at high maternal ages and high parities. Since such pregnancies place both the mother and the child at greater risk of death, there are some gains to be made.

The recent upsurge of interest in possible health effects of family planning has been much concerned with possible benefits from altering the timing of births, rather than simply the quantity. Births to teenage mothers and births at short intervals have frequently been identified as being at high risk of death and attempts to alter the timing of births thus advocated. Claims for the possible overall mortality gains from the avoidance of these high risk pregnancies have ranged from zero<sup>4</sup> to thirty<sup>5</sup> per cent reductions in infant or child mortality. Since these results have generated quite heated debate, we proceed to examine some of the issues in more detail.

## Quantifying the gains from improved birth timing

In order to focus discussion, we shall concentrate on the differences between Bongaarts and Hobcraft in their presentation and interpretation of the magnitude of these effects.

Both make considerable use of summarised regression results from a major study covering thirty-four developing countries<sup>6</sup>.

Hobcraft refers to sizeable excess mortality for first births and for births to teenage mothers, which both experience an increase of risk of death before age five of about one-third, compared with well-spaced second or third births, whose preceding sibling had not yet died, of mothers aged 25-34. Further, a teenage first birth experiences an excess risk of just over 80 per cent compared with the same reference group. Births of order seven and above experience an excess risk of some 20 per cent, but those to mothers aged 35 and over have minimal excess risk once other factors are controlled. First births were at especially high risk in the Middle East and North Africa, with a two-thirds, rather than one-third, excess risk.

In contrast Bongaarts chooses to focus his attention upon infant mortality only, which serves to lower the effect of improved birth timing. The excess risk he takes for

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<sup>4</sup> See J.Bongaarts (1987) Does family planning reduce infant mortality rates? Population and Development Review 13(2):323-334.

<sup>5</sup> The figure of about thirty per cent as the likely maximal gains for some countries (e.g. Costa Rica or Jordan) are given by J.Trussell and A.R.Pebley (1984) The potential impact of changes in fertility on infant, child, and maternal mortality. Studies in Family Planning 15(6):267-280. Similar figures also appear in J.Hobcraft loc cit in note 3. Both of these papers go to great lengths to emphasise that such figures are the maximum that might be achieved, but the figures have sometimes been taken out of context to suggest that such gains might be universally achievable.

<sup>6</sup> See J.N.Hobcraft, J.W.McDonald and S.O.Rutstein (1985) Demographic determinants of infant and early child mortality: a comparative analysis. Population Studies 39(3):363-385. Averaged results for all 34 countries and for regional grouping are given by Hobcraft (1987) loc cit in note 3.

first births is 62 per cent, about twice the value for experience up to age five. Moreover, Bongaarts fails to recognise that the relative risks for first births (and other crucial groups) in the regression analyses are contrasted with a reference group for which the preceding birth survived, in addition to the controls for birth order and birth-spacing (and maternal education and sex of child). The survival of the previous birth was interpreted as an indication of the 'family environment' effect by Hobcraft. Since this indicator is only available for births after the first, the first birth relative risk includes the impact of these high-risk families. In a high mortality environment, the first birth excess risk might well be reduced from 62 per cent to about 34 per cent, if a rough and ready allowance is made for this<sup>7</sup>, while being about 47 per cent in a lower mortality environment. The corresponding figures for mortality up to age five would be 36 per cent uncorrected, perhaps 12 per cent in a high mortality environment and 24 per cent in a low mortality situation. The more elaborate simulations given by Hobcraft referring to the impact of differing family formation patterns upon survival patterns in a family of six children took due account of these features.

The two studies also differ in their treatment of excess mortality risks associated with short birth intervals. Hobcraft concluded that a badly spaced prior birth (taken as an interval of less than two years between births) raised the average chance of dying in infancy by about 60 to 70 per cent and the chances of dying before age five by around 50 per cent. If the preceding child died, this raised the risk of infant death by 80 or 90 per cent, virtually regardless of the length of the preceding interval, and the overall chance of dying before age five by at least 70 per cent. On the other hand Bongaarts arbitrarily reduces the effect associated with a short interval to a 30 per cent excess (incidentally citing a 52 per cent excess, that for survival to age five, rather than the 66 per cent figure for infancy given by Hobcraft). Bongaarts again fails to allow anywhere in his analysis for the 'familial' effects stressed above. Similar adjustments to those indicated in footnote 6, but with somewhat higher overall levels of mortality for the poorly spaced births, might raise an adjusted excess risk for infants to around 113 per cent with high mortality levels and about 90 per cent with moderate to low levels<sup>8</sup>.

Hobcraft also considers the risks associated with a rapid subsequent birth, although Bongaarts avoids having to allow for this by confining his analysis to infant mortality and hence must understate (at zero) any potential gains from improving spacing of subsequent births. For example, the excess mortality risk between ages one and five associated with the next birth occurring within twelve months is 70 to 80 per cent.

Hobcraft uses the results from the regression models in some elaborate simulations,

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<sup>7</sup> This is obtained as follows. The relative risk of infant death in a poor family environment is obtained by dividing that when one child died by that where one child survived in the measured effects for preceding birth intervals. Where this interval was less than two years, the relevant values were 3.23 and 1.66, giving an excess risk of 95 per cent; where this interval was two to four years, the values were 1.92 and 1.06, giving an excess risk of 81 per cent. We take 90 per cent as the excess risk associated with this group. If 22 per cent of children die before age 5, the extra risk from familial background is 0.209 (=  $0.22 \times 0.95$ ) and the adjusted (and truer) excess risk for a first birth becomes 34 per cent (=  $1 - 1.62/1.209$ ). The other adjustments are similar.

<sup>8</sup> For infants, the relative risk in infancy for a poorly spaced previous birth which survived was 1.66 and that for one which died was 3.23. Taking 30 per cent of these births as dying, we get an overall relative risk of 2.13, or 113 per cent ( $0.7 \times 1.66 + 0.3 \times 3.23$ ). Fifteen per cent is taken as a moderate level of mortality by age five for this disadvantaged group. The figures provided here are meant to illustrate the orders of magnitude involved rather than provide definitive answers, which would be harder to obtain.

which kept track of survival status of preceding births and of the other relevant factors which were controlled in the regression analyses. In order to highlight the potential gains from changing family formation patterns, rather than the overall level of fertility, he considered four ways of having six births. The family building strategies involved either early or late first birth and repeated good or poor birth-spacing. Compared with the most favourable pattern (one with a later first birth and all births well-spaced), a teenage start with subsequent good spacing raises the number of infant deaths by 15 per cent; a later start, with persistent poor birth-spacing generates 80 per cent more deaths; and a teenage start combined with poor spacing raises the risk of death by 120 per cent. This worst combination, in a moderate mortality environment where 6.7 per cent of low-risk births (not first births, not to teenage mothers, well-spaced before and after, and with the preceding child not having died) die by age two, results in fully 26 per cent of the most disadvantaged families of six experiencing two or more child deaths (before age two), compared with only eight per cent for the families with the most advantaged pattern of childbearing. It was and must be stressed that these results refer to illustrative family formation patterns which are extremes. However, they serve to illustrate the possible maximum gains which might be expected at the family level from changes in the timing of births. They are upper limits for two reasons: firstly, as already indicated, they contrast extremes of family building patterns which may only rarely arise in reality; secondly, they assume that all the excess mortality risks shown in the regression analyses are causal, which is almost certainly incorrect<sup>9</sup>.

Hobcraft also tried to set upper and lower bounds on the possible child mortality gains at the societal level which could result from improving the timing of child-bearing. The upper bounds were obtained by asking what might happen if patterns of child-bearing of one society could be transformed to those of another. The most favourable pattern of child-bearing among the 18 countries considered was for Korea, with Lesotho second. The least favourable pattern was identified as occurring in Jordan, with Colombia and

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<sup>9</sup> It was for this reason that Bongaarts chose to reduce the excess risks associated with a short preceding interval. Our difference is that all excess risks thus identified are in my view likely to be overstated. If any one is to be adjusted downwards, all should be; the problem is that we really have no way of assessing the relative magnitude of the causal effects compared with unmeasured other correlates. Bongaarts (see notes 3 and 4) argues that spacing effects are special in this respect. Trussell (p.176) says

"Bongaarts states that Hobcraft's estimated excess rate of 52 (*sic*) per cent is too high for a variety of reasons that I fully accept, the most important of which is the absence of controls for the effect of breastfeeding in the Hobcraft analysis".

Yet the only comparative study to examine this issue carefully concluded

"A rather surprising result is the lack of strong evidence to support the hypothesis about the interrelations between pace of childbearing, breastfeeding and mortality. We expected breastfeeding to be an important mediating mechanism through which the pace of childbearing affects mortality risks. If so, the estimated effects of length of preceding birth interval and timing of following conception should be considerably attenuated once the effects of breastfeeding are controlled. A comparison of Table 1(a) and (b) shows that this does not occur to any significant extent" (A.Palloni and S.R.Millman (1986) Effects of inter-birth intervals and breastfeeding on infant and early childhood mortality. Population Studies 40(2):223).

While the Palloni and Millman study was only concerned with Latin American countries, where the duration of breastfeeding is relatively short, it certainly undermines the confident assertions of Bongaarts. Palloni and Millman pay close attention to data errors and are cautious about their results. We would echo such caution and concur in regarding the effects as biased upwards, but perhaps no more than other ones.

Costa Rica close behind. If child-bearing patterns in the Latin American and Caribbean countries considered and in Jordan could be transformed to those of Korea, the results suggested potential reductions of mortality for children under five in excess of 20 per cent and as high as 30 per cent for Jordan. Even Senegal, the country with the third most favourable pattern of child-bearing, might show a ten per cent reduction in child mortality with a shift to the family building patterns of Korean women.

The maximum potential consequences of worse family building patterns were also assessed. For example, if traditional child-spacing restraints in Lesotho were to collapse and patterns came to match those of Costa Rica or Jordan, this could raise child mortality by up to 30 per cent.

Bongaarts addressed this issue in a different way. Given that he used the raw regression coefficients, except for the reduction in the effects of poor child-spacing, he was also generally dealing with maximal effects. However, the means for assessing change was curious. In essence, it was assumed an inevitable consequence of increased family planning in sub-Saharan Africa or South Asia would involve adoption of the poor spacing patterns of Latin America, since these regions respectively dominate his groups I and III. By adopting this scenario, Bongaarts more or less ensured that increased use of family planning could not be associated with improved mortality. He was almost replicating the worst case scenarios of transition for sub-Saharan Africa mentioned above.

There is nothing that has yet happened in Korea or Sri Lanka to suggest that spacing patterns inevitably get significantly worse with increased use of family planning. On the contrary, Hobcraft showed that Korea still had the most favourable timing pattern of births and both societies have considerably reduced the incidence of teenage births with increased use of family planning. Similarly, there is clear evidence that the major fertility transition in several Latin American societies has not been accompanied by much change in childspacing patterns<sup>10</sup>.

Both Hobcraft and Bongaarts set out to address the question of real gains from altered child-bearing patterns in individual societies, although once again the style of analysis and conclusions differed. Bongaarts took the experience of Matlab Thana in Bangladesh as a 'laboratory experiment' of a 'highly successful' family planning programme, citing the increase in contraceptive prevalence from less than 10 per cent in 1976 to 34 per cent in 1981 and the lack of change in infant and child mortality over this period. Yet what effects were to be expected in this environment? Bangladesh already had extremely good birth spacing, which has traditionally been achieved through very prolonged lactation, so that the only health question likely to be of interest on this score is simply whether this favourable spacing pattern is sustained with increasing use of contraception or whether the position worsens, as could happen. The main potential area that Hobcraft would identify for birth timing to affect child health in Bangladesh would be through delaying first births. There is nothing to suggest that this is a major goal of the programme, nor that major changes are taking place on this score in the Matlab area. For these reasons, the Matlab experience does not constitute any evidence against the possibility of gains from altering the timing of births; Bangladesh would be one of the

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<sup>10</sup> See J.Hobcraft and J.McDonald (1984) Birth Intervals. WFS Comparative Studies 28 International Statistical Institute. In our view, the WFS data given there do constitute fairly reliable evidence on time-series of birth interval distributions, which Bongaarts states "are not readily available" (p329, reference given in note 4).

last places to try to demonstrate any such effects.

In contrast, Hobcraft used real experience to pose the question as to how much unintended gain might have arisen from altered child-bearing patterns in countries with major changes in the use of family planning over the period covered by the data from the World Fertility Survey. Since no programme in the countries considered has set out specifically to alter child-spacing patterns as a major focus and very few have emphasised avoidance of teenage births, any gains in the timing of births were likely to be largely fortuitous. The concomitant part of any gains in infant and child mortality would thus represent an indication of the minimum that might be attained by a family planning programme, although we note the difficulty inherent in partitioning change by the simple standardisation procedures used. For two countries, Costa Rica and Trinidad and Tobago, those which experienced the greatest fertility declines over the period considered, the gains attributable to improved child-bearing patterns over a ten year period amounted to a 15 per cent decline in child mortality. These gains account for about a third of the very substantial reduction in Costa Rica and without these gains the child mortality level in Trinidad and Tobago may well have risen slightly over that period. These are striking gains when it is borne in mind that improvements in child-bearing patterns might only ever be expected to reduce child mortality by at most 30 per cent.

### **Programmatic implications**

Improving the pattern of childbearing is clearly not a panacea for reducing child mortality. However, there are sufficient indications that some gains might be made to warrant experimentation. The accumulated evidence of the past few years, which demonstrates very considerable recorded excess risk for poorly spaced births and for births to young mothers, cannot be dismissed on the basis of the flawed studies of Bongaarts. This is not to argue that major returns will necessarily flow from interventions, which requires them to be efficiently and effectively implemented. Moreover, it is clear that interventions through family planning must be targeted to the conditions of the society in question.

For much of Latin America and the Arab World, there is evidence to suggest that a major emphasis upon improving child-spacing for health purposes might improve overall mortality levels significantly, quite probably by 15 to 20 per cent, if the record of Costa Rica and Trinidad and Tobago is indicative, and possibly by as much as 30 per cent. For much of Asia and sub-Saharan Africa, the key element with regard to spacing is to ensure that programmes emphasise the importance of traditional spacing practices for child health. In both situations, there are strong arguments in favour of placing emphasis on child-spacing for health reasons.

Another key aspect of timing of fertility, in terms of identifiable high risk births, is the timing of the first birth. Altering behaviour in this regard is probably more a matter for fundamental social change than for family planning programmes. Rising female education can play a key role in delaying too early an initiation of childbearing, in addition to its many other indirect effects on child health.

The importance of teenage births as a risk factor for the survival has been called into question recently, on the basis of a few studies in highly developed societies<sup>11</sup>. To dismiss the very powerful evidence for excess risk in the third world on the basis of experience in situations where good compensatory care is widely available is akin to throwing out the baby with the bath-water<sup>12</sup>.

As with most areas of health, there are two approaches to intervention: preventive and curative medicine. In this case the two approaches translate into avoiding the high-risk pregnancy in the first place and into reducing the risks attached to those pregnancies by selective intervention. Just as curative medicine alone will not prevent the incidence of diarrhoeal diseases, so it is reasonable to encourage mothers to avoid high-risk pregnancies. Where they do not succeed in avoiding the pregnancy, medical intervention can come to the rescue. Early child-bearing in the U.S. occurs in a population which reaches reproductive maturity at an earlier age than do women in many third world countries. Thus a pregnancy at age 18 in Bangladesh is more likely to be associated with increased directly age-related risks to the mother and child than one to an 18 year-old in the U.S. Even the poorest mothers in the U.S. probably have access to better compensatory care than a typical Bangladeshi mother.

The issues of prevention versus subsequent direct health intervention also arise in the context of child-spacing. However effective a policy may be in this respect, some women will inevitably conceive at short intervals. This risk is probably particularly high where the previous child died, which is also a clear indicator of excess risk of death for the outcome of the current pregnancy. In such circumstances interventions will always be desirable. To provide a concrete example, in Jordan about a third of all births beyond the first birth occur at intervals of less than 18 months after the previous birth and these children face two-and-a-half to three times the risk of death in the first five years of life experienced by children born at intervals of two to four years. Such differences for a very large group of births cannot conceivably be dismissed as entirely or even predominantly due to prematurity or other factors. Delaying these pregnancies by even a few months might well serve to reduce child mortality considerably.

## Breastfeeding

Breastfeeding can play both a direct and indirect role in improving survival chances of children, although the gains are difficult to quantify and likely to interact in a host of ways with other health factors<sup>13</sup>. Breast-milk (and perhaps particularly colostrum) has important immunological properties, which can help the child to fight infection. Breast-milk is an uncontaminated source of food, although poor nipple hygiene can

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<sup>11</sup> See A.T.Geronimus (1987) On teenage childbearing and neonatal mortality in the United States. Population and Development Review 13(2):245-279. C.Makinson (1985) The health consequences of teenage fertility. Family Planning Perspectives 17(3):132-139.

<sup>12</sup> Undoubtedly some of the observed excess risk for births to teenage mothers is attributable to social disadvantage which would not necessarily be altered by delaying the first birth.

<sup>13</sup> For a detailed review of these issues see S.L.Huffman and B.B.Lamphere (1984) Breastfeeding performance and child survival. In Mosley and Chen op cit in footnote 2.

partially negate this advantage in terms of protection from exposure to infection. Breast-milk is a rich and well-balanced form of food and is well tolerated by the infant. Prolonged breastfeeding also serves to delay the resumption of menses and to reduce the probability of conception even after the resumption of menstruation<sup>14</sup>. Lactation is thus, at least on a population basis, an important factor in delaying the next conception, which has health consequences for the child being fed and for the survival of the next birth. As a consequence of its important role in birth spacing, lactation also inhibits overall levels of fertility, with effects upon the total flow of births and therefore on the total numbers of children for whom health services have to be provided.

The actual impact of breastfeeding on child survival is difficult to measure for several reasons, many of which are associated with reverse causation. A child that is very sickly at birth may not survive long enough to be put to the breast, especially in those societies where colostrum is not regarded as suitable for a baby. Moreover, if the mother is seriously unwell for reasons related to childbirth, the child will not be breastfed, will be more likely to have suffered birth complications, and will almost certainly be placed at greater risk of death subsequently, especially if the mother dies. At later stages in a child's life, cessation of breastfeeding may result from illness well before the death of a child. These difficulties mean that it is essential not to contrast survival chances of those children who were never breastfed with those who were, since the never breastfed group are often highly selected for poor survival chances for reasons unrelated to choices about breastfeeding. For this reason, a number of analysts have looked at the effects of breastfeeding status at one point in time upon later survival chances. This approach has the advantage of being analytically simpler than treating breastfeeding status as a time-varying covariate, as well as partially overcoming the problems of reverse causation. Breastfeeding does have sizeable effects upon the survival chances of children, as shown by DaVanzo *et al*<sup>15</sup> and by Palloni and Millman<sup>16</sup>.

Few studies, if any, have examined the total impact upon survival chances to, say, age five of early versus later termination of breastfeeding. For example, it would be helpful to know by how much the survival chances to age five of those children who were still alive at, say, age six months were affected by their breastfeeding status at age four months or even at age six months. It is important to know these longer term effects because weaning is hazardous for the child whenever it occurs, although there is some evidence that the risks decrease with increasing age. Thus the child that is weaned earlier may indeed experience higher mortality risks in the months of weaning, but may then have a smaller compensatory mortality advantage when the children who were breastfed for longer are being weaned.

A further problem is, of course, the use of breastfeeding status as though it were a switch. In most societies mothers do not wean abruptly, except perhaps when they

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<sup>14</sup> See A.M.John, J.A.Menken and A.K.M.Alaudin Chowdhury (1987) The effects of breastfeeding and nutrition on fecundability in rural Bangladesh: a hazards model analysis. *Population Studies* 41(3):433-446. D.Guz and J.N.Hobcraft (forthcoming) Lactation and fertility: a comparative analysis. *Population Studies* 43.

<sup>15</sup> J.DaVanzo, W.P.Butz and J.-P.Habicht (1983) How biological and behavioural influences on mortality in Malaysia vary during the first year of life. *Population Studies* 37(3):381-402.

<sup>16</sup> *Loc cit* in note 9.

discover they are pregnant again. An abrupt weaning can have major health consequences for the child<sup>17</sup>. There are variations in the extent of supplementary feeding and of the use of (often contaminated) water for drinks. Weaning is often a prolonged and gradual process and the differences in health protection between a child being breastfed several times a day, with some supplementation, and one being occasionally put to the breast as a pacifier may be greater than the contrast between the latter case and a fully weaned child.

### Some Other Indirect Factors

Although our brief was to dwell mainly upon the indirect effects of family planning and breastfeeding upon child survival and health, there are many other important indirect factors which are strongly associated with child health and perhaps of greater consequence in any debate concerning direct versus indirect interventions. Due to space limitations, it is only possible to touch upon these issues here and some selectivity is inevitable. Nevertheless, since the issues are important, we must not lose sight of them in the broader context of assessing interventions.

We are beginning to accumulate evidence concerning the clustering of infant and child deaths within families<sup>18</sup>. This has important implications for the methods of analysis used in studying the correlates of infant and child mortality, since most conventional statistical testing becomes biased as a result. But much more important are the issues of identification of such high-risk families and the development and targeting of appropriate and effective interventions.

There is now overwhelmingly strong evidence that maternal education serves to reduce infant and child mortality, as do several other measures of socioeconomic status, most notably husband's education and occupation<sup>19</sup>. This is clearly an indirect intervention, but a fuller knowledge of the pathways through which it has an effect would be useful for targeting programmes, e.g. community programmes aimed at use of soap or at basic health knowledge. If, alternatively, the main pathway turned out to be through increased individual autonomy this would be harder to stimulate with a cheap, direct, substitute intervention. Much more attention also needs to be paid to the interaction of education with the health environment and with service provision in determining health outcomes for children.

In a wider societal context, much emphasis has often been given to issues of political will, mass mobilisation and concern with equity of health provision, of income,

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<sup>17</sup> See, for example, the classic study by H.Leridon and P.Cantrelle (1971) Breastfeeding, mortality in childhood and fertility in a rural zone of Senegal. *Population Studies* 25:505-533. These abrupt weaning effects are an important factor in the impact of a too rapid subsequent pregnancy (or birth) upon the survival chances of the previous child.

<sup>18</sup> The 'familial' effects identified by Hobcraft *et al* (see note 5) are indicative of a wider problem. For example, Monica DasGupta has indicated (personal communication) that about 60 per cent of infant and child deaths in the Khanna resurvey study are attributable to only ten per cent of the families.

<sup>19</sup> See J.Hobcraft, J.Mcdonald and S.Rutstein (1984) Socioeconomic factors in infant and child mortality: a cross-national comparison *Population Studies* 38:193-223; J.C.Caldwell and P.Caldwell (1985) Education and literacy as factors in health. In S.B.Halstead, J.A.Walsh and K.S.Warren (eds) *Good Health at Low Cost* Rockefeller Foundation. See also footnote 2, for references to Caldwell and to Ware in this context.

and of opportunity, with some emphasis upon female autonomy<sup>20</sup>. Indeed, it could perhaps be argued that the success of China in this respect was fundamental to the change of climate which led to the Alma Ata declaration. As an example of horrifying levels of inequality, we might instance Mexico in the 1960s, where a child born to an uneducated mother, whose current husband was uneducated and in a household with no lavatory (a common situation in rural Mexico) was about **19 times** as likely to die between ages one and five as was a child born to a highly educated mother and father in a household with access to a lavatory (a common situation in urban Mexico)<sup>21</sup>. The debates about inequality, education, social networks and political also loom large in much recent literature on developed countries<sup>22</sup>. The larger debate concerning the relative roles of medicine, nutrition, public health intervention and socioeconomic change is also relevant here<sup>23</sup>. This affects judgements concerning important, and widely debated, issues about vertical or horizontal organisation of health programmes and about community-based provision.

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<sup>20</sup> See Halstead *et al loc cit* in footnote 19; A.T.Flegg (1982) Inequality of income, illiteracy and medical care as determinants of infant mortality in underdeveloped countries *Population Studies* 36(3):441-458; J.C.Caldwell (1986) Routes to low mortality in poor countries *Population and Development Review* 12(2):171-220.

<sup>21</sup> See J.Hobcraft (1984) Use of special mortality questions in fertility surveys: the World Fertility Survey experience. In United Nations *Data Bases for Mortality Measurement* UN ST/ESA/SER.A/84. Some caution is, of course, required in interpreting this result. We make no claim that the factors identified here are causal, but they were clearly powerful correlates of true risk factors.

<sup>22</sup> See M.I.Roemer (1985) Social policies and health care systems: their effects on mortality and morbidity in developed countries. In J.Vallin and A.D.Lopez (eds) *Health Policy, Social Policy and Mortality Prospects* Ordina for IUSSP. L.A.Sagan (1987) *The Health of Nations: True Causes of Sickness and Well-being* Basic Books. P.Townsend, N.Davidson and M.Whitehead (eds) *Inequalities in Health: The Black Report and The Health Divide* Penguin Books.

<sup>23</sup> See the classic work on England by McKeown and colleagues (T.McKeown (1976) *The Modern Rise of Population* Edward Arnold). For a different perspective see S.H.Preston (1976) *Mortality Patterns in National Populations* Academic Press. These themes have been addressed in the third world context by W.H.Mosley (1985) Will primary health care reduce infant and child mortality? A critique of some current strategies, with special reference to Africa and Asia. In Vallin and Lopez *loc cit* in note 22.

# The Interim is Over: Implications of the Changing Cause-Structure of Mortality for the Design of Health Interventions

Douglas Ewbank<sup>1</sup> and Susan Zimicki<sup>2</sup>

Countries with relatively high levels of child mortality generally also have low levels of personal income and limited resources that can be allocated to the health sector. Even with foreign aid, governments of these countries must make difficult decisions regarding which kinds of health services they are going to provide. Health planning therefore involves balancing the short-term need to save lives with the longer-term goal of universally available comprehensive health care. This balance is affected by the choices made with regard to a) the kinds of medical personnel that are trained and the topics stressed in their training, b) the types of drugs that are imported or produced in large quantities and provided free or at subsidized cost, c) the types and quantities of medical equipment purchased or produced, and d) the kinds and sites of health facilities that are built. The context in which these choices are made is strongly affected by planners' sense of health priorities.

Walsh and Warren's influential paper (1979) recommended selective primary health care as an interim strategy for health planning. They recommended that in the short-term priorities for candidate diseases be set according to their prevalence, mortality and morbidity, and the existence of cost-effective treatments or preventive measures. Thus acute diarrheal diseases and measles were given high priority, because of their high incidence, high mortality and the existence of feasible means of control, while diseases such as amoebiasis, for which control is difficult, were assigned to the low-priority group.

The current emphasis of international donors on a small number of "packages" that have been implemented in a wide range of developing countries is an extension of this approach. The packages -- the most obvious examples are those pertaining to vaccination and control of diarrheal diseases -- focus on technologies that are recommended because donors feel that a) they are effective in preventing or treating diseases which are common, serious health problems in most high-mortality countries, b) they can be successfully implemented in low-income countries without long-term capital investments in high-level

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training, equipment or facilities, and therefore, c) they can have a quick, demonstrable impact on morbidity and mortality.<sup>2</sup>

This very focused, short-term and generalized approach, with its emphasis on low-cost technology transfer, has been successful in that vaccination coverage levels and availability of oral rehydration solutions have greatly improved during the past decade (UNICEF, 1988). However, this approach has tended to concentrate benefits in groups with priority diseases (for example children, to the detriment of their mothers and fathers) and has often displaced emphasis from development of a health infrastructure that could sustain truly comprehensive care (Habicht and Berman, 1980; Rifkin and Walt, 1986).

With the growing success of these programs and awareness of the limitations of this approach, the attention of countries and donors has been drawn to designing successor programs. The list of candidates includes malaria prevention and treatment, treatment of acute respiratory infections, prenatal care, growth monitoring, and breastfeeding or other nutrition interventions. It is unlikely that any of these programs will be as universally applicable as those centered on vaccination and control of diarrheal diseases. We therefore propose that choices of successor programs for child survival be made with more careful consideration of local cause structures of mortality, that is, the relative importance of causes of death.

With respect to the short-term, this increased attention to geographic and other differentials will indicate adjustments to be made in current program priorities. For the longer term, our approach examines changes in cause structure that are likely to occur as the result of programs. Consideration of these changes indicates how the infrastructure must be developed in order to sustain current program successes and lay a foundation for future programs. In most cases, the diseases which are likely to predominate are not ones which are best addressed by the delivery strategies now recommended for vaccination and diarrheal disease control programs.

Clearly, many other factors must be considered before priorities for health programs are set, including locally perceived needs, political concerns, and the level of development of the health system. We are convinced, however, that a thorough examination of the cause of death structure and assessment of the ways it is likely to change will lead to recommendations that are balanced and realistic.

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<sup>2</sup> It is important to note that although these programs are often given priority, aid is not generally limited to such a narrow range of programs nor solely to programs within the health sector. For example, there has been continuing and significant attention to development of safe water supplies. It is also worth noting that in the case of vaccine-preventable diseases, the extraordinarily low cost of the vaccines to groups such as UNICEF and PAHO reflects the perception by international organizations that priority should be given to these diseases (Robbins and Freeman, 1988).

## **Use of the Child Mortality Rate to Examine the Cause Structure of Mortality**

While the level of infant mortality (i.e., mortality during the first twelve months of life) has been extensively used as an indicator of a country's overall level of mortality and morbidity, in this paper we will focus on mortality under age 5. During the first few months of life, mortality is dominated by "endogenous" causes -- low birth weight, birth trauma, congenital defects and neonatal tetanus. However, during the rest of the infant year the causes of death are basically the same as those at ages 1 through 4 years.<sup>3</sup>

Because neonatal mortality is responsible for much of infant mortality, relying on the causes of death among infants can be misleading. For example, tetanus will appear more important if infant mortality patterns are used to determine priorities than if under-five mortality patterns are used. A similar distortion can occur in the priority given to measles, since a large proportion of measles deaths occur after the first year of life. For example, in the Machakos area of Kenya, 6.2% of the infant deaths were to measles, substantially less than the 22.3% ascribed to diarrhea (Omandi-Odhiambo et al., 1984). However, if we look at all deaths under age 5, the proportions of deaths to measles and diarrhea are more similar (15.2% and 20.0%).

### **Implications of the Cause of Death Structure for Planning in the Short Term**

Given the cause of death structure in most developing countries and the present choice of cost-effective preventive measures it is not surprising that many high-mortality countries have decided to give priority to the vaccine-preventable diseases and acute diarrhea. However, a careful examination of variations in the cause of death structure of these countries suggests that countries might wish to give top priority to a few of the diseases targeted by these programs.

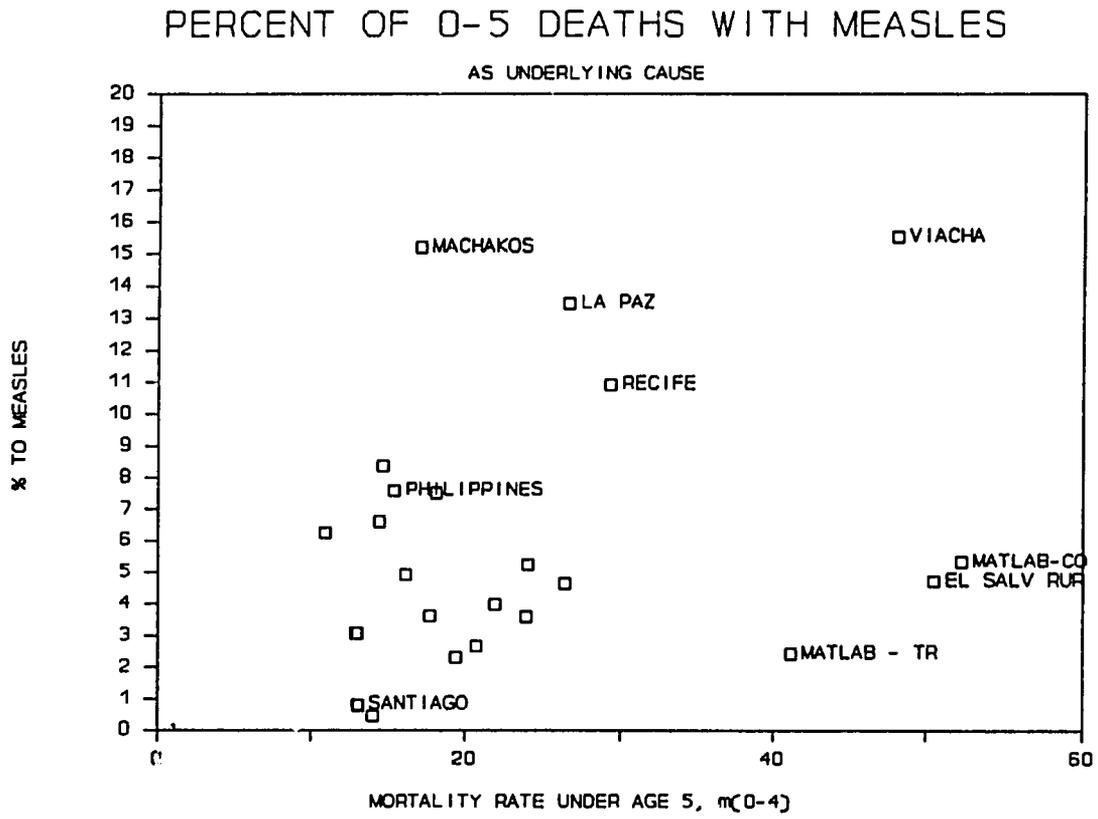
#### Vaccine-preventable diseases

Many vaccination programs are designed to begin with BCG vaccination at birth then proceed through the series of DPT and polio and finally end up with measles at about 9 months of age. Tetanus immunization is generally encouraged during the last months of pregnancy. Although this sequence is undoubtedly appropriate for individuals, it is not clear that this sequencing should set priorities within programs. In particular, in many countries measles and tetanus are substantially more important than the other vaccine-preventable diseases and probably deserve increased priorities within vaccination programs. For this reason, it is useful to examine in detail the relative importance of measles and tetanus and to consider ways in which vaccination programs might adjust the standard program design to take account of the relative importance of different vaccine-preventable diseases.

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<sup>3</sup> Moreover, from the point of view of measuring the effect of packages, small-sample estimates of 0-4 mortality are more reliable than estimates of infant mortality, particularly early infant mortality (Ewbank, 1984).

Figure 1



**Measles.** There is substantial variation in the relative importance of measles as a cause of death in children less than 5 years old. Figure 1 shows that the proportion of deaths under age 5 ascribed to measles in Latin American countries in the 1960's (Puffer and Serrano, 1973), in the Philippines (National Census and Statistics Office, 1985), in Matlab, Bangladesh (Shaikh et al., 1984), and in the Machakos District of Kenya (Omandi-Odhiambo et al., 1984). This proportion ranges from greater than 15% in Machakos and Viacha to less than 3% in two areas where vaccination coverage was high, Chile and the Matlab treatment area. However, despite substantial vaccination against measles in Machakos (probably with a high vaccine failure rate [Ewbank et al., 1986]) measles was responsible for a far higher proportion of deaths (15%) than in the Latin American populations with a similar level of mortality but lower levels of vaccination. The comparison becomes even more stark if we consider West African populations. Pison and Bonneuil (1988) report that in a population in eastern Senegal measles accounts for 31% of the deaths of children age 6 months to 9 years. In an urban area of Guinea-Bissau in 1979, a year when there was a severe measles epidemic, 57% of deaths of children aged 6 months to 35 months were caused by measles (Aaby et al., 1983). During the following two years, when about 67% were protected by vaccination, measles was still responsible for 9.5% of the deaths in this age group.

Given the high mortality reported for measles in parts of West Africa it is hard to believe that any single health intervention could reduce mortality as much as vaccination against measles.<sup>4</sup> If we use the proportion of deaths due to measles to help set priorities, we would expect that health programs in Africa would include substantially increased efforts to vaccinate children against measles. These efforts might include special education campaigns aimed specifically at increasing awareness of the need for measles vaccination at an early age. Moreover, in some areas a substantial portion of measles incidence (and mortality) occurs in children less than 9 months of age, that is at ages earlier than the recommended age for vaccination (Taylor et al., 1938). While the recommended strategy is to vaccinate children as soon as possible after they reach 9 months old, a 2-dose vaccination schedule with the first dose given when children are 6 months old or use of a vaccine which could be given to younger children might be appropriate.

**Tetanus.** Neonatal tetanus may deserve increased attention in areas where it is a major cause of death. There is substantial variation between areas in the importance of neonatal tetanus. Although tetanus mortality is less than 5 per thousand live births in many areas, there are well-documented cases of populations in which the rate is 20 or more deaths per thousand (Stanfield and Galazka, 1984). In high-prevalence areas, health planners should seriously consider emphasizing maternal vaccinations. This is especially important in countries where a large proportion of deliveries occur at home and few women receive antenatal care. In populations where tetanus is a major cause of death and coverage of antenatal care is inadequate (i.e., fewer than 2 visits by the end of the second trimester of pregnancy), an alternate strategy that should be seriously considered is

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<sup>4</sup> A successful intervention against malaria might have a similar impact, but is far more complex to implement.

immunizing all women of childbearing age (Schofield, 1986). For example, programs might consider providing tetanus immunization to mothers who bring children for their standard vaccinations. Two doses of toxoid provide reasonable protection against neonatal tetanus for the next pregnancy, while four or five doses probably provide protection for the entire reproductive span (Wassilak and Brink, 1986).

### Diarrheal diseases

Although diarrheal diseases are common causes of death in all high-mortality populations, there are important differences in the relative importance of acute and chronic diarrhea. For example, in the absence of health interventions in Matlab Bangladesh 44% of the deaths at ages 1 to 4 were due to diarrhea, with about half of these (22%) due to acute diarrhea (Chen et al., 1980). In the Gambia, even after a great reduction in deaths to measles, only 17% of deaths of children less than 7 years old were due to diarrhea (including "malnutrition and/or chronic diarrhea") and only 8% were due to acute gastro-enteritis (Greenwood et al., 1987).<sup>5</sup>

There are also differences in the facilities available for treatment of diarrhea. Data from the Matlab area provide insights into the relative advantages of clinic-based treatment and home use of oral rehydration solutions. At the time when home-based use of oral rehydration solutions was introduced, most cases of severe watery diarrhea were already properly treated in the diarrhea treatment center. Mortality was not further reduced by home-based use, but clinic attendance fell by 30%, with most of the decline due to milder cases being treated solely at home (Zimicki et al., 1984). Previous experience in Bangladesh had showed that adequate treatment of acute diarrhea in easily accessible clinics can reduce mortality substantially (Chen et al., 1980). However, this study demonstrated that even where adequate clinic treatment is accessible appropriate home management (including ORS, SSS or home-available fluids) can reduce the patient load at clinics. In areas where clinics are not easily accessible, home use of rehydration solutions should be a central element of acute diarrhea management.

Thus, for acute diarrhea the priority given to home management will be affected by the prevalence of the disease and the accessibility of clinics. In addition, the priority given to different types of home management (ORS, SSS, home-available fluids and dietary management) should be based on the strength of the distribution system for packets, the home availability of sugar and salt, and cultural practices which will enhance or hinder various types of education efforts.

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<sup>5</sup> The cause of death information from The Gambia was obtained during a year when there was a serious meningitis epidemic, which was responsible for 9% of the deaths of children less than 7 years old. Thus, the proportions of deaths due to all other causes in that year underestimate the true contributions of those causes.

## **Causes of Death That are Apt to be Important for Mid- to Long-Term Planning**

There are several important causes of death that are not currently priorities in health planning in most developing countries. Two examples are respiratory diseases and malaria. The reason for their relative neglect has had less to do with their prevalence than the perceived cost-effectiveness of the available technologies and disagreements over the appropriate strategies for their control. However, as mortality declines, there has been a shift in the relative importance of various diseases, and such "difficult" diseases are now the leading causes of death in some areas. Their rise to prominence has prompted a new look at possible ways of preventing or treating them. It is clear that most of these diseases will not be addressed by the type of specialized campaigns often used for vaccination and oral rehydration.

The effect of programs on the cause structure of mortality is apparent in the data for several populations. For example, in the Gambia, an extensive vaccination program reduced measles to only 3% of deaths of children less than 7 years old (Greenwood et al., 1987). In the absence of vaccination it had previously accounted for a substantially greater proportion, as virtually all unvaccinated children get the disease, and in this population about 5% of children die within a month of having measles, and up to 15% die within 9 months (Robertson et al., 1985). After the vaccination program reduced measles mortality, other diseases became the major causes of death: acute respiratory disease (15%), malaria (14%) and diarrheal disease (17%, with 8% due to acute diarrhea and 9% to chronic diarrhea).

A second example comes from the Matlab area of Bangladesh, where extensive programs to reduce mortality caused by acute diarrhea and measles brought about substantial changes in the cause structure of mortality in addition to lowering overall mortality. Chen et al. (1980) used 1975-1977 cause of death and hospital attendance data to estimate that in the absence of programs measles would have been responsible for 13% and acute diarrhea for 22% of deaths among 1 to 4-year-old children. After the programs (1982-83) these two diseases were responsible for 4% and 14% of deaths respectively (Zimicki, 1988). As these causes declined in importance, respiratory mortality increased from 13% to 20%.

As these two examples demonstrate, respiratory disease is one of the major candidates to be a focus for successor programs. There are substantial areal variations in the proportion of child deaths ascribed to respiratory infections, reflecting not only variations in the prevalence of other diseases (e.g., tetanus, measles, and malaria), but also differences in the mortality to this one cause. For example, in a study of regional differences in mortality in Kenya (Ewbank et al., 1986), respiratory diseases have about 3 to 4 times higher mortality in the highlands of Central Province than in the lowlands of Coast Province. A similar difference is apparent in Puffer and Serrano's work (1973) which shows that respiratory mortality is substantially more important in Bolivia than in

any of the other study areas.<sup>6</sup>

In areas such as these, where as much as 30 to 40% of the under-five mortality can be ascribed to respiratory infections, substantial mortality reduction will require programs to deal with this problem. Some portion of this mortality may be eliminated as a consequence of measles and pertussis immunization; however, acute respiratory infections accounted for about 15% of all deaths in children less than 7 years old in the Gambia even at high vaccination coverage levels for both pertussis and measles (Greenwood et al., 1987). Even though proposals for large-scale curative programs are controversial, since they would require diagnosis of pneumonia and dispensing of antibiotics by peripheral workers, identification and treatment of serious cases is an intervention that will probably be necessary for further reduction in mortality.

The case of malaria is similar. It is clear that mortality is not apt to decline substantially in malarious areas unless this problem is addressed. In the Gambia, for example, where average levels of vaccination for all antigens are between 80 and 90% for children one year and older, mortality is still very high (more than a quarter of all children die before their fifth birthday), with malaria responsible for 14% of the deaths of children before age 7 (Greenwood et al., 1987). The spread of chloroquine-resistant *Falciparum* malaria may even increase the number of deaths due to malaria.

The AIDS epidemic will also have significant implications for health planning even if it remains concentrated in relatively small areas. Piot et al. (1988) have recently estimated a 50% congenital infection rate in infants born to seropositive mothers. Thus in areas where 5% of women of childbearing age are seropositive the number of deaths under age 5 due to AIDS will be about 25 per thousand, similar to the number of deaths due to tetanus in high-prevalence tetanus areas. The implications for existing programs are the need for a greater emphasis on sterile injections techniques, recommendation of condoms for contraception, eliminating unnecessary transfusions (for example, for children with malaria), and provision of safe supplies of blood.

It is also important to consider the children of AIDS-infected mothers who are not themselves infected; they have a high likelihood of becoming orphans and of being treated as outcasts. It has been shown in several populations that children whose primary caretakers are not their own mothers are at increased risk of malnutrition (Bledsoe et al., 1988; Rawson and Berggren, 1974) and missed vaccination (Yoder, 1988). Thus this group becomes an important special target group for vaccination and other first-step health programs.

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<sup>6</sup> It seems likely that much of this variation can be explained by environmental factors (e.g., smoke in houses in colder areas or the effects of altitude) rather than differences in prematurity. For example, Puffer and Serrano report that the prevalence of low birth weights was actually lower in La Paz than in many of the other areas they studied although respiratory deaths were higher.

## **Developing Health Systems That Will be Appropriate in the Mid- to Long-Term**

One criticism of the programs developed following the approach suggested by Walsh and Warren is that they may have a short-term impact but are incompatible with achievement of the goal of comprehensive health care (Rifkin and Walt, 1986). This is especially true of campaign approaches to increasing vaccination coverage. However, short-sighted planning is not a necessary result of the use of cause of death statistics in setting priorities. In fact, the dynamic view of the cause of death structure discussed above supports health delivery strategies which are more consistent with the development of a comprehensive health care system.

Table 1 presents a list of major types of interventions and the causes of mortality that they address. The first three groups of interventions -- oral rehydration, vaccination and nutrition -- are stressed in current child survival programs. The other interventions (vitamin A, child spacing, anti-malaria, anti-acute respiratory infections, chronic diarrhea, and antenatal care) are packages which are candidates for emphasis in the next stage of programs to reduce mortality. For each package we have listed the main technologies that might be included. The list of technologies is meant to encompass all technologies that are both possible and practical; thus technologies of varying effectiveness are included. For example, for anti-malaria programs we have included vector control programs, although the main type of vector control program that has been used (spraying) does not appear to be a generally viable alternative for large areas.

The last four columns of Table 1 indicate the delivery strategies that are feasible for each technology. The three types of delivery strategies are:

- a) Campaign teams - including such special workers as EPI vaccination teams and antimalarial spraying campaigns,
- b) Clinic workers - including all regular employees of primary health care units and private physicians who care for women and children, and
- c) Home-based delivery - including home-prepared oral rehydration solutions and foods as well as the use of items purchased outside the primary health care system (e.g., condoms, antimalarials, and commercial oral rehydration solutions).

For clinic workers, we distinguish between workers who provide services solely within clinics and those involved in outreach services in order to make it clear that there is an alternative between the extremes of special campaign teams and in-clinic workers. It is important to note that many of the home-based activities require extensive education efforts which can be short-term (e.g., radio campaigns) or can be carried out in clinics. The table indicates only who provides the services, not the associated education programs.

An examination of Table 1 shows that very few of the technologies that are likely to be prominent in the next stage of the mortality decline are amenable to strategies based

Table 1

PACKAGE	CAUSE	TECHNOLOGY	TYPE OF STRATEGY			
			Campaign Teams	Clinic Workers Out-reach	Clinic Workers In-Clinic	Mothers
Oral rehydration	dehydrating diarrhea	home-available fluid oral rehydration solution appropriate feeding (DMD)		X X X		X X X
Vaccination	neonatal tetanus	immunization	X	X	X	
	measles	"	X	X	X	
	pertussis & diphtheria	"	X	X	X	
	tuberculosis	"	X	X	X	
	polio	"	X	X	X	
	non-neonatal tetanus	"	X	X	X	
Nutrition	malnutrition diarrhea	growth monitoring <sup>1</sup> nutrition education dietary management of diarrhea breastfeeding	X	X X	X X	X X
Vitamin A	diarrhea respiratory disease	capsule distribution	X	X	X	
Child spacing		contraception (breastfeeding)		X	X	X <sup>2</sup> X
Malaria	malaria	treatment vector control	X	X X	X	X <sup>3</sup>
ARI	pneumonia bronchitis	recognition + antibiotics		X	X	
Chronic diarrhea	malnutrition	recognition + antibiotics proper feeding		X X	X X	X
Antenatal care	perinatal	access to maternity clinics risk detection + referral iron and folic acid supplementation		X X	X X	

<sup>1</sup>Although growth monitoring is often considered to be an important part of primary health care, it is not actually an intervention. It is a method of identifying high risk children.

<sup>2</sup>Some contraceptives (eg, condoms and in some countries pills) are available commercially.

<sup>3</sup>Presumptive use of chloroquine in the home is probably common in many countries, but is not generally, a component of health programs.

on large campaigns or to unsupervised home use. This contrasts with vaccinations, which can be delivered using a campaign strategy, and oral rehydration solutions, which mothers can be taught to prepare and use in unsupervised conditions. However, experience with home-based oral rehydration solution strongly suggests that substantial continuing education efforts are necessary to sustain high levels of appropriate use in the home (Chowdhury et al., 1988). This experience is probably directly relevant to programs that include interventions such as improving child feeding practices and use of antimalarials.

It is clear, therefore, that health programs designed for the next stage of the mortality decline will require strong primary health care units, probably with effective out-reach efforts. The integration of health services around a primary health care unit has the added advantage that these programs can be designed to ensure sustainability and offer the flexibility needed to adjust priorities to fit local conditions.

There are important reasons why many of these programs are tied to clinics. First, campaigns are suitable only for conditions which have a relatively high incidence or prevalence. Second, campaigns are more feasible for prevention programs than for programs to treat acute illnesses. For example, if acute respiratory disease could be prevented through vaccination, campaigns would be one possible method for service delivery, but treatment for acute respiratory infections must be readily available whenever a case occurs. Third, there are few generally accepted program strategies that involve unsupervised home-based use of preventive or treatment measures, partly because these strategies are only reasonable for common illnesses and partly because of the nature of many of the technologies and the consequences of their misuse. These factors suggest that in order to prepare the health system to deal with the next stage of expansion and the next set of priorities, the current programs should be designed so that they strengthen primary health care services.

The same conclusion emerges if we examine vaccination and oral rehydration programs in detail. A short-term perspective can lead to the conclusion that special campaigns or intensive efforts by specialized teams are necessary to achieve the most immediate, greatest impact on mortality. However, a more careful analysis which looks to the mid- to long-term makes it clear that eventually these programs will have to be incorporated into regular primary health care programs if they are to be sustainable. In some cases, during the early efforts to implement a series of new interventions, there may be a need for extensive out-reach and education or even special campaigns. However, as the program matures and the technology is accepted by the majority of the population, the efforts must become routine.

One example of this occurred in an oral rehydration program in Egypt. During the early stages of a test program, the area where special outreach efforts were made achieved substantially higher rates of use of oral rehydration solution and lower diarrhea mortality than a control area. However, three years after the start of the national program, oral rehydration use and diarrhea mortality were very similar in the two areas even though special outreach efforts had been continued in the test area (National Control of Diarrheal

Diseases Project, 1988). This suggests that although campaigns can have a faster impact, similar success can be achieved through regular health programs. In the mid- to long-term, the reliance on primary health care units will probably be more sustainable and will prepare a basis for future program efforts.

### **Other Program Changes Required as Mortality Declines**

As the general level of mortality declines, it becomes increasingly important to identify those subgroups in the population which are at highest risk and which are not receiving services. When under five mortality is very high, many children are at high risk and need basic services. As the level of mortality declines, there are often pockets of high mortality which remain.<sup>7</sup> These pockets might include rural areas which are distant from services or very low income urban neighborhoods some of which may include many migrants (Rip et al., 1987). In addition there are often social, economic or cultural groups which are missed by general programs, for example, the poorest, illiterate groups, or fostered children. As mortality declines it becomes increasingly cost-effective to mount special efforts to identify and serve the high risk groups. In addition, finding these groups is justified by concerns for equity and the universal availability of a minimum standard of living.

### **Conclusion**

International assistance programs such as the Child Survival Program of USAID are centered on a few standard packages of programs. This approach is based on the fact that there are a number of causes of death that are important in many populations with high mortality and that can be reduced using relatively simple technologies. However, there are differences among high-mortality countries in the relative importance of acute diarrhea, vaccine-preventable and other causes of death. In particular, malaria may deserve high priority in the health strategies of a number of countries. In addition, in almost all high-mortality populations a substantial proportion of child deaths are due to respiratory disease; in areas where this proportion is particularly high, respiratory disease should be given high priority in health programs in the near future. These differences in the cause of death structure should be taken into account when setting the priorities for health programs.

In addition to the choice of the main packages of programs, variations in the relative importance of different causes might lead to altering the priorities given to various parts of each package. For example, in countries where measles is particularly important, vaccination programs should be designed to achieve the highest possible coverage of measles vaccination at the earliest appropriate age. Similarly, in populations where

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<sup>7</sup> For example, in one study from the Gambia four of the six children who died of measles "came from one hamlet which had a poor vaccination record" (Greenwood et al., 1987).

neonatal tetanus is very important, the vaccination program must find a workable strategy for immunizing a large proportion of women of childbearing age.

Careful examination of the cause of death structure and consideration of how it is likely to change will lead to two other conclusions. First, as vaccination and diarrheal disease programs succeed in reaching high coverage rates, there are still pockets of social or residential groups which are missed by the programs. Therefore, as these programs succeed, there is an increased need to target missed groups. Second, the success of current programs will increase the importance of other causes of death. The programs that will be among the next set of priorities are more likely to depend on strong primary health care units. In addition, the continued success of vaccination and diarrheal disease programs will probably also depend more on primary health care units. Thus, an examination of the major causes of death and the technologies available for preventing or treating them suggests that there is a growing need to strengthen general primary health care programs.

We have examined the implications of the cause of death structure for planning child health programs. This is only one of many considerations which should guide the setting of priorities; cultural, historical, political and financial considerations are also important. However, we feel that a careful examination of the causes of death and the available range of programs to address them leads to recommendations for health planning which are consistent with the development of comprehensive health care.

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# Infant Mortality Rates and Cause Attributable Profiles: Some Implications for Primary Health Care Design

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## INTRODUCTION

"Selective Primary Health Care," as defined by Walsh and Warren (1) emphasizes the use of specific technologies for maximal and rapid reduction of infant and child mortality, and at relatively low cost (2). Oral rehydration therapy and vaccination (particularly against measles and neonatal tetanus) are considered to be particularly effective and most widely practiced; antibiotic treatment of pneumonia, vitamin A supplementation, growth monitoring and nutrition education, and birth spacing are other interventions whose values to large populations are still under study.

Selective primary health care (SPHC) is severely criticized for different reasons:

- 1) It is said that SPHC diverts resources and does little to build sustainable primary health care at the community level; moreover SPHC is classically "top-down," creating dependence on outside organization and supply, uncontrolled by families themselves (3). The "military paradigm of EPI" (4), for instance, may preclude a respectful approach to health education and community diagnosis -- replaced, instead, by "manipulative social marketing" (5).
- 2) Others allege that SPHC focuses too narrowly on infectious disease control, ignoring (and thus, perhaps, abetting) powerful social, economic and nutritional antecedents to illness (6).
- 3) Finally, SPHC is not yet proved to have a lasting impact on mortality; a child saved from one bout may only live long enough to succumb to another not addressed by SPHC ("replacement mortality") (7).

This study seeks to sharpen the discussion. Developing countries are not all the same. They vary widely in such indicators as infant mortality, prevalence and severity of illness, resources and infrastructure. These variations may predict the impact of SPHC.

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We hypothesize that infant mortality rates and the profile of causes of death of infants are linked; and that if discernible patterns exist they may suggest refinements to the SPHC approach which take into account the criticisms noted above.

## **METHODS**

Our analysis of infant mortality rates and infant mortality causes is based on published data.

Two cross-sectional data sets were examined. The first was derived from Preston, Keyfitz, and Schoen (8) who compiled life tables and causes of death from national data of 48 countries (13 Third World) between 1946-1964, and longitudinal analysis of mortality in England and Wales from 1861-1964 (males only). The second data set was obtained by a search of the literature for studies of infant mortality linking rate and causes. Some came from individual survey and research studies discovered through Medlars and Index Medicus going back to 1972; others had already been compiled in World health organization collections of vital statistics. A full listing of these sources is available from the authors. 117 reports were identified, of which 17 were excluded from analysis for having either a) more than 50% of deaths unassigned by cause, or b) listing fewer than four causes of death.

Appendix 1 shows how the data were tabulated from this second set. Up to now we have done only the most rudimentary metaanalysis: means calculated for all cross-section data, curves fitted to longitudinal data, stratification of IMRs (high, medium, low) with display of their corresponding cause-attributable profiles. We have not, for instance, attempted to weight the profiles by respective sample sizes, nor calculated confidence intervals and tests for significance of differences.

We are aware, of course, of all the limitations of vital data and cause-specific mortality reports; whether these faults are subdued or magnified by our metaanalysis is not known, but we may appeal our conclusions from the data to "what makes sense."

## **FINDINGS**

Figure I, from Preston, et al, shows expectation of life at birth (a surrogate measure for IMR) and the average % of each age-cohort dying from specific causes; not surprisingly, neoplasms form a greater proportion of death at higher ages, infectious diseases at lower ages. A suggestive hump in the curve for diarrhea is magnified on Figure II; as IMR falls, deaths from diarrhea first increase proportionally, then decrease. Figure III shows a similar finding in a study by Mata in Costa Rica: as IMR fell, mortality from diarrhea in infants became increasingly correlated with IMR (9).

Figure IV shows the longitudinal data from England and Wales from 1861-1964, with the chance of dying by age one. Even as total IMR fell, the absolute contribution of diarrheal deaths rose and then fell.

We grouped data from 100 populations by rates of IMR which are high (IMR = 120-240, N=16), medium (IMR = 60-119, N=60) and low (IMR = 24-59, N=24). For each group the average contribution of each cause of death was determined. Figure V

shows the cause-specific patterns in absolute values; Figure VI shows them in proportional terms. The two figures appear to show the following:

Deaths from diarrhea increase absolutely and as a proportion of total deaths as IMR falls from the "high" to the "medium" level.

Neonatal tetanus deaths fall sharply with decreasing IMR, disappearing at the "low" level.

Measles, as a cause of infant mortality, is greatest at the "high" IMR level.

At "high" IMR, infectious and parasitic illness contributes substantially and probably represents malaria to a large degree.

Perinatal and respiratory deaths stay relatively constant in proportion to the high and medium levels of IMR.

At lower levels of IMR the total number of causes contributing substantially to death also declines. (Table I)

## **DISCUSSION**

If the distinction between categories of IMR and their corresponding cause-attributable profiles are valid, we may infer a more considered approach to SPHC. Theoretically, one prefers district-level data for district-level planning. What we show here are greatly aggregated data. But since health care planning takes place at national levels, these data provide for several inferences.

In high IMR areas, vaccinations against measles and neonatal tetanus are particularly important; independent evidence now suggests that there is a sustained reduction in infant mortality with these two interventions. Antimicrobials for treatment of pneumonia, malaria, and dysentery should be made regularly available at the community level. Vitamin A treatment of measles patients, and home-based ORT are possibly effective in addition. These steps alone are the beginnings of a basic primary care program that includes: home-care; village-based health workers who treat illness with a few basic drugs by protocol, and who prepare the community for the national EPI.

In medium IMR areas, a full-fledged national ORT program with stress on infant nutrition will be likely to cut IMR significantly (10) and a solid EPI will reduce morbidity considerably. More attention to causes and prevention of low-birth weight are also likely to pay-off in lower perinatal and respiratory deaths (in particular, birth spacing and extra food for pregnant women). Measures are needed to prevent bottle-feeding and use of contaminated weaning foods.

In low IMR areas a much greater emphasis on prenatal care is necessary to improve perinatal survival. Rush (11) estimates that higher birth weights will affect IMR more substantially at low IMR than medium IMR. Health education, better hygiene and water supply, good primary, secondary, and tertiary care will drive down the residual

## IMR.

This study also suggests that "replacement mortality" is a lesser factor when there are fewer conditions to do the replacement, but that diarrhea is the predominant "replacing" disease as IMR falls. Preston indicates this as well (12). In a way this is good news because as diarrhea becomes a larger component of mortality it presents more often as acute watery diarrhea, hence more susceptible to ORT, and less often as chronic diarrhea and dysentery (13).

It is not a coincidence that the falling IMR and changing cause-attributable profiles correlate with the strength of the health system and the ability of the citizenry to demand and choose equitable and quality care, although we cannot conclude a casual relationship. A nation's capacity to effect the different interventions is also related to its resources, political will and how much families know (and how much women know, in particular). PHC or SPHC alone seem unlikely to improve these attributes, unless one can show that citizens, politicians and donors recognize a falling IMR (and improved health) and that this awareness fuels more concern about the social and economic causes of poor health. UNICEF believes this to be true and promotes National Vaccination Days to this end. The dialogue around Child Survival has certainly created more awareness of social development issues.

Table 2 shows the USAID Child Survival emphasis countries in descending order of IMR, and with total populations given. Knowledgeable inspection of this table informs us how, as a first approximation, these countries may approach SPHC based on IMR, available resources, and the data presented in this paper. Country and district-specific profiles could suggest more accurate allocation of resources.

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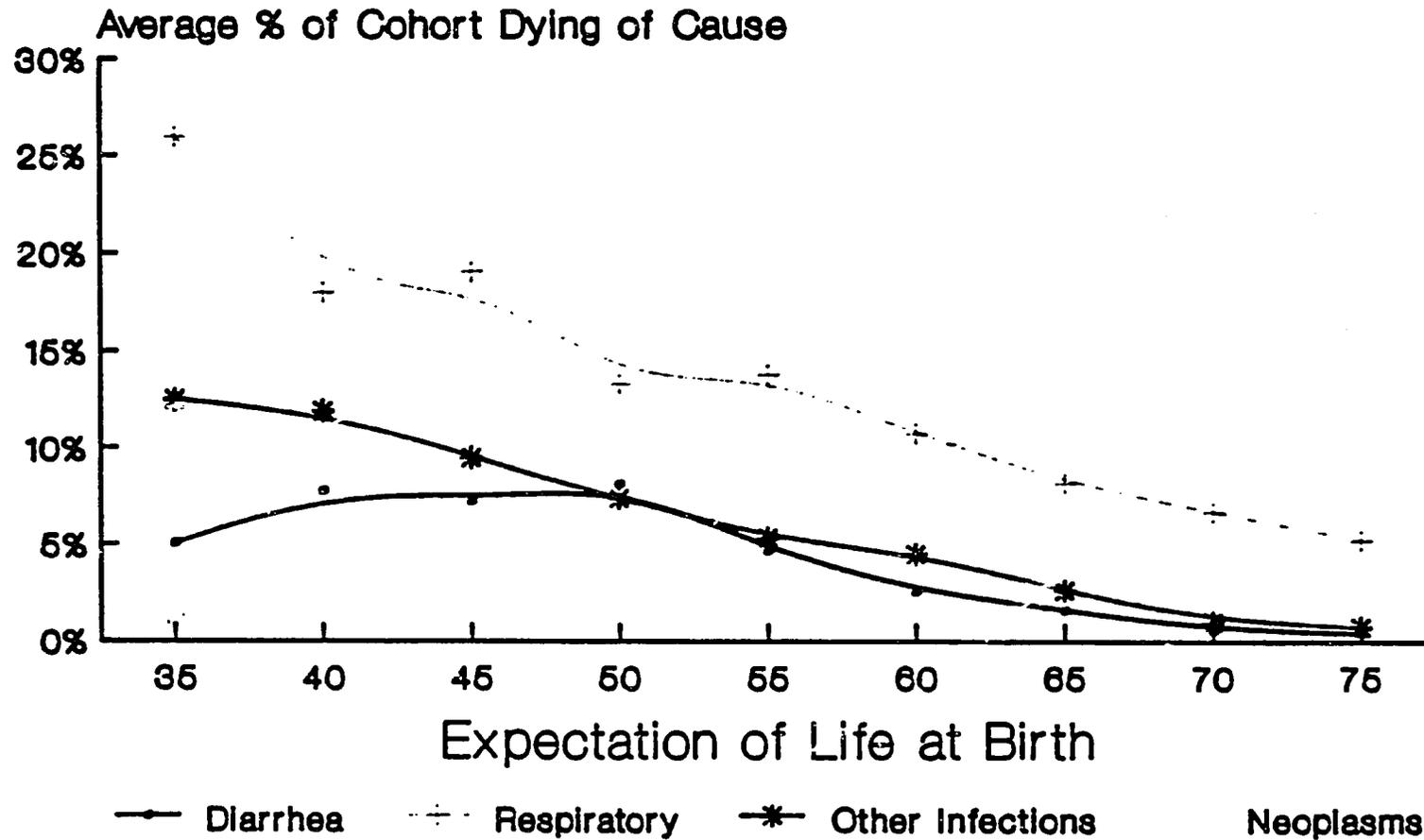
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13. Compare, for instance, Bangladesh and Egypt; IMR in the former is nearly twice that of Egypt, diarrhea causes about 15-20% of IMR (compared to 50-60% in Egypt), and about 30% of diarrhea in children in Bangladesh is chronic or dysenteric (compared to about 10% in Egypt).

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FIGURE 1

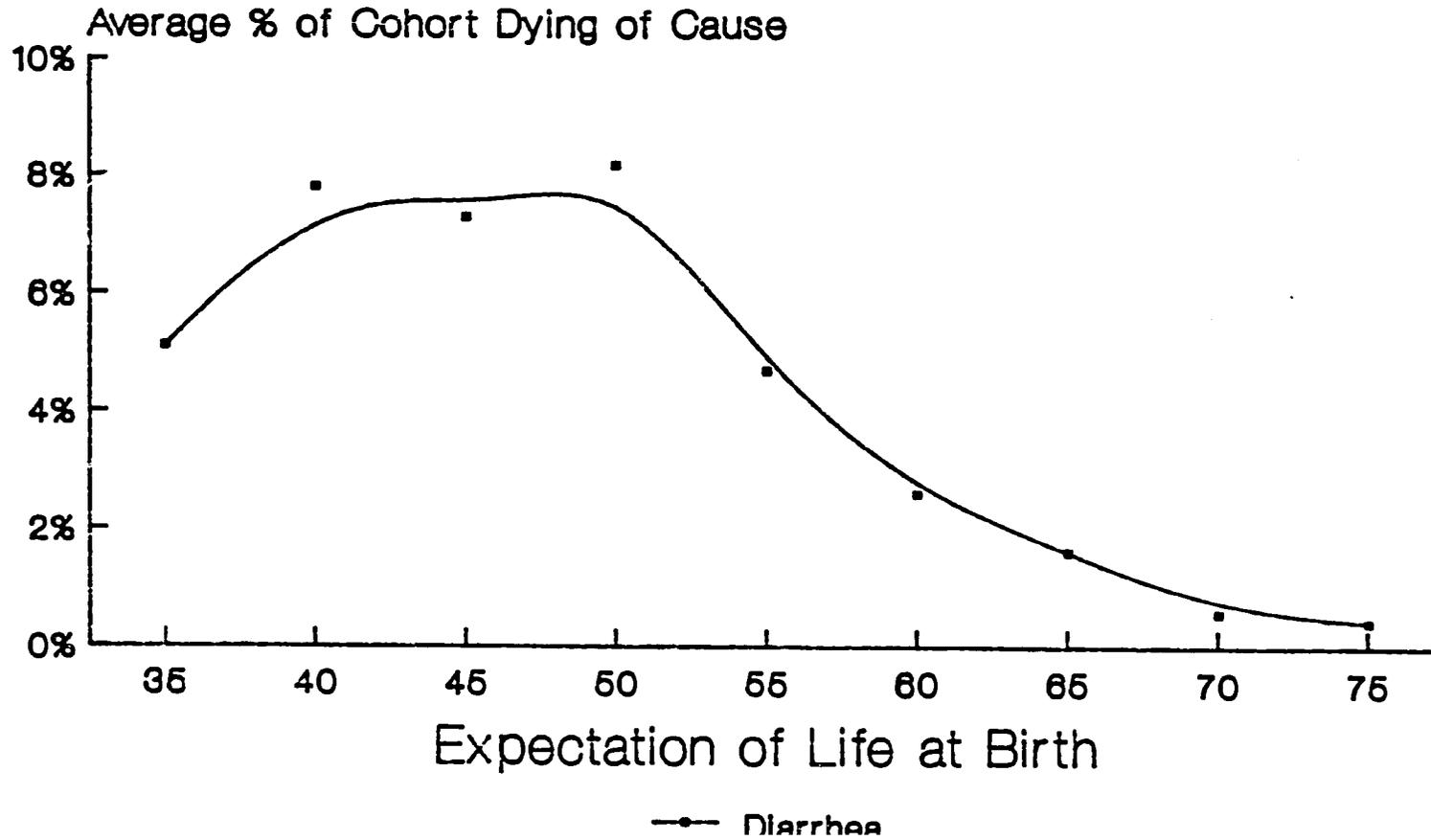
# Cause of Death of Newborns By Expectation of Life at Birth



Preston, Keyfitz and Schoen, 1972.  
Cause of Death: Life Tables for  
National Populations

FIGURE 2

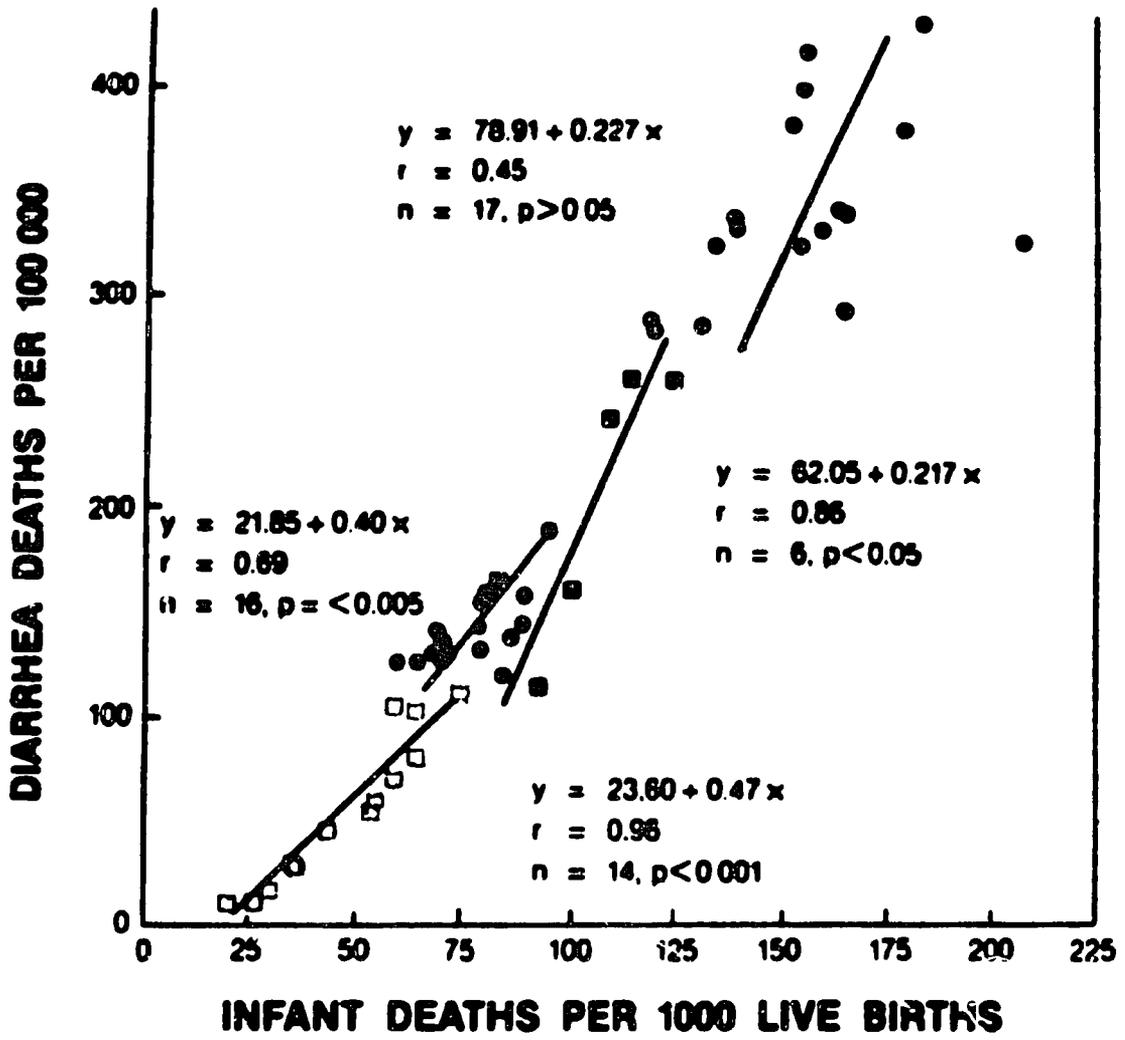
# Cause of Death of Newborns By Expectation of Life at Birth



Preston, Keyfitz and Schoen, 1972.  
Cause of Death: Life Tables for  
National Populations

1972

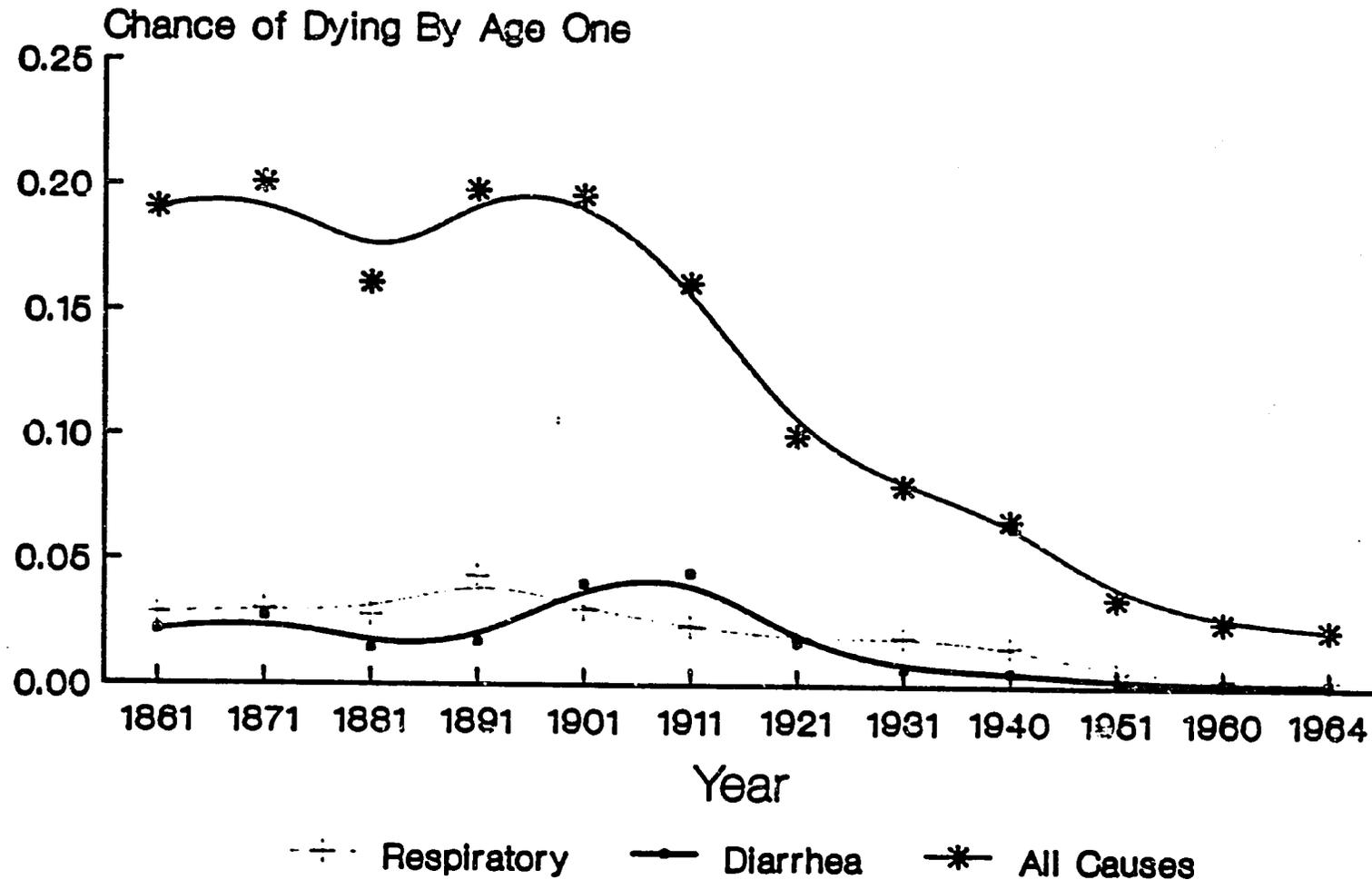
FIGURE 3



764

FIGURE 4

# Infant Deaths, England & Wales, 1861-1964 Cause Specific Life Table Analysis



Preston, Keyfitz, and Schoen, 1972

105

FIGURE 5

# Infant Mortality Profiles: Cause of Infant Death at Varying Levels

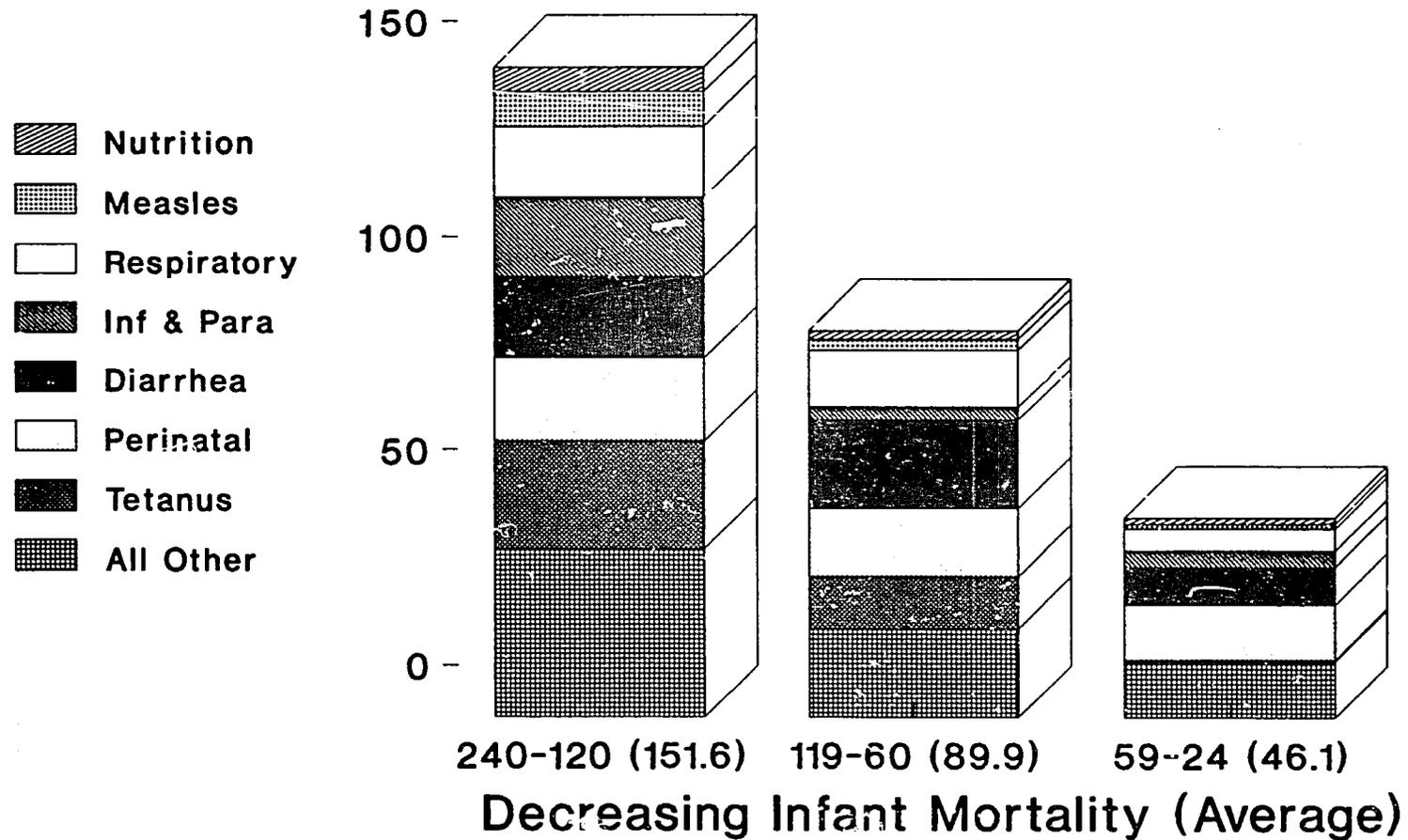
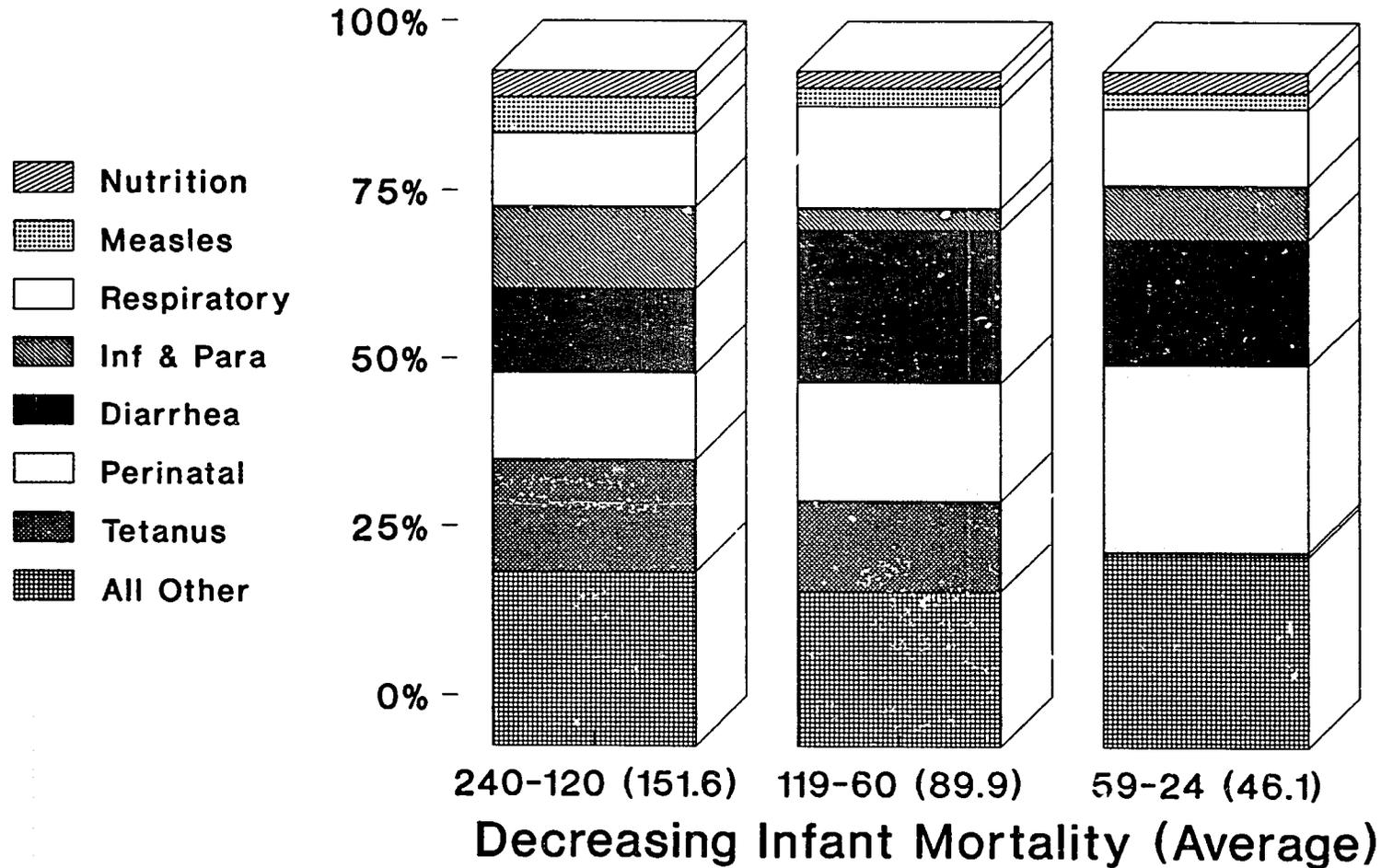


FIGURE 6

# Infant Mortality Profiles: Cause of Infant Death at Varying Levels



167.

Table 1

**Infant Mortality Rates and Total Populations of  
the 22 Child Survival Emphasis Countries**

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<u>Country</u>	<u>Infant Mortality Rate</u> (per 1000 live births)	<u>Total Population</u> (in millions)
Mali	175	8.1
Malawi	157	6.9
Niger	140	6.1
Senegal	137	6.4
Nepal	134	16.5
Bangladesh	124	101.1
Haiti	123	6.6
Yemen Arab Republic	120	7.3
Bolivia	117	6.5
Pakistan	115	100.4
Sudan	112	21.6
Nigeria	110	95.2
India	105	758.9
Zaire	103	29.9
Peru	94	19.7
Egypt	93	46.9
Morocco	90	21.9
Indonesia	79	166.4
Honduras	76	4.4
Kenya	76	20.6
Ecuador	67	9.4
Guatemala	65	8.0

Source: State of the World's Children 1987, UNICEF

# Demographic Models for Child Survival: Implications for Program Strategy

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## INTRODUCTION

The child survival revolution being promoted by the international health community has heightened our awareness that far more can be done to promote the health and welfare of children in the developing world. The essence of this strategy is to provide to every family some information and a few highly effective technologies. A fundamental premise is that substantial improvements in health can be achieved even in poor countries with the appropriate allocation of the limited resources available to the health sector. Indeed, this strategy does have a substantial empirical basis when one looks at the accomplishments of such low income countries as China, Sri Lanka, or the State of Kerala in India.

The child survival program strategy has been the subject of increasing debate in the international health community in recent years (Nabarro and Mosley, 1988). While all parties support the principle of redirecting the limited resources available from urban-based hospitals to community-oriented programs, there has been considerable disagreement about the relative importance of different elements of the basic program strategy, particularly in different social and economic settings (Taylor and Jolly, 1988). As originally proposed, the core interventions in the child survival program strategy included growth monitoring, oral rehydration, breastfeeding promotion, and immunizations (given the acronym GOBI). In recent years, particular emphasis by a number of major donors has been given to the support of immunizations and oral rehydration programs as the key underpinnings of the child survival revolution. Much of the criticism relates to the priority that should be given to promoting these technologies versus allocating resources to a broader range of primary health care interventions such as antenatal and childbirth care or water and sanitation programs.

The goal of policymakers is to make the optimum strategic choices under given resource constraints. The scientific approach to this problem would seem straightforward—assess the magnitude of various health problems and the effectiveness and cost of various interventions and select the best alternatives. This, in fact, was the basic approach of Walsh and Warren in their study which developed the concept of selective primary health care (Walsh and Warren, 1980). While the arguments put forth in that paper were scientifically sound based on an understanding of the biological processes of individual

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diseases, it is the premise of this paper that this approach, which has been widely used by the international community, is flawed because it fails to take into account how multiple disease processes operate. What we shall consider here are the program and policy implications coming from a shift of our analysis from a medical-clinical viewpoint to a public health-demographic perspective.

In this paper we will look at alternative demographic models of child survival and discuss their implications for program strategies. We propose that a more realistic model must incorporate both the concepts of competing risks and acquired frailty. A first approximation of a mathematical formulation of this model is given to illustrate the implications of using this model for testing alternative program strategies.

## ALTERNATIVE CONCEPTUAL MODELS

### Basic (Medical) Model

Figure 1 illustrates the basic model that is most often (implicitly) used by health professionals in considering alternative disease control strategies.

In these conceptual and all the following models, the following notation is used:

S	=	Survivors
D	=	Deaths
I	=	Incidence of a disease
C	=	Case fatality rate of the disease
Z	=	level of frailty as a consequence of the disease (among survivors)

Suffixes f, ff refer to progression of states of frailty among survivors.

Suffix r refers to the non-frail (robust) among survivors.

Suffixes a, b, refer to diseases a and b.

The basic model begins with a group of newborns (S) exposed to disease a. The deaths due disease a are a function of the incidence rate times the case fatality rate. All survivors leave the childhood age group with no distinction made among those who did and did not contract diseases.

The implications of this model are: every disease is considered separately; equal weight is given the curative and preventive interventions; and diseases of low mortality but high morbidity/disability (such as parasitic infestations, anemia, etc.) will largely be ignored as priorities for interventions.

Table 1 illustrates the typical use of this basic model in formulating child survival program priorities (Rohde, 1983). Each major cause of death is taken separately. Assumptions are made about the effectiveness of specific interventions, and the deaths prevented for each specific disease are equated with lives saved. As observed by multiple critics of this approach, no account is taken of the fact that in poor countries many of the same children are exposed to all of these conditions concurrently, producing increasing disability and death.

### Competing Risks Model

Figure 2 illustrates an alternative model which takes into account competing risks (Mosley, 1986). For the purposes of clarity of this presentation, the two competing diseases are shown as occurring sequentially. This can occur, for example, when a child has neonatal tetanus followed later by an episode of pneumonia or diarrhea. More often, however, populations of children are exposed simultaneously to multiple disease posing risks to survival (the mathematical model presented later takes into account these simultaneous risks).

One major implication of this model is that survival following any specific intervention relates not only to the effectiveness of the intervention, but is also strongly conditioned by the burden of all other diseases in the population. Because the problem of competing risks in high mortality populations can substantially reduce the effectiveness of disease-specific technologies in promoting child survival, one is forced to re-evaluate the appropriateness of selective intervention strategies (Chen, 1981). This is the concern that has recently been raised by Greenwood and colleagues (1987) who observed continued high mortality among immunized children in The Gambia.

A second implication, which follows the first, is that under high mortality circumstances, more broadly based interventions which reduce the risk of several diseases simultaneously may prove to have greater lifesaving potential. This, in fact, was demonstrated by Rahman (1982) in rural Bangladesh who compared the lifesaving potential of maternal tetanus immunizations with utilization of trained traditional midwives. As shown in Table 2, while tetanus vaccine was clearly more effective in selectively reducing the incidence of neonatal tetanus, the traditional midwives actually saved more lives because they also prevented neonatal deaths due to other causes.

### Frailty Model

While model 2 is more realistic in taking account of competing risks to survival, it is inadequate because it assumes that there is only one demographically significant consequence of a disease--that is death. In fact, the morbidity associated with most childhood diseases can result in the child being more frail for a period of time and, therefore, at higher risk of dying of all other causes (Cole and Parkin, 1977; Mata, 1978). Not infrequently this metabolically induced frailty can be aggravated by traditional practices by families such as withholding food during episodes of diarrhea or febrile illnesses. More significantly, frailty can also build up in a population of children through chronic low grade disease conditions or nutrient deficiencies that are not recognized as direct "causes" of death such as malaria parasitemia, hookworm infestation, or vitamin A deficiency.

In Figure 3 the competing risk model is expanded to incorporate frailty. This indicates that following disease a, the survivors are partitioned into two subgroups--a subgroup which was unaffected ( $S'$ ) and a subgroup which had a non-fatal episode of disease a producing more frail survivors ( $S''$ ). Under these circumstances, disease b will produce more deaths because the more frail subgroup will have a higher case fatality rate (inflated by a factor  $Z$  representing the effects of frailty). Again, following disease b we

see further partitioning of the survivors into: those who were spared both diseases a and b ( $S''$ ); those who were affected by either disease a or b but survived ( $S''_1$ ) whom we assume now have the same level of frailty; and those who survived cases of both disease a and disease b and are now assumed to have even greater frailty ( $S''_2$ ).

The empirical support for this model is abundant (Cole and Parkin, 1977; Solimano and Vine, 1980; Martorell and Ho, 1984; Rowland, et al., 1988; Rowland, et al., 1977). Studies in Guatemala by Mata (1978, p. 279) of the growth of children from birth through age 3 years documented the progressive retardation of growth (an index of frailty) associated with a multiplicity of respiratory and enteric infections and parasitemia is apparent. Noteworthy, every episode of significant disease in this child was treated with the best Western medicine as a part of the study. This is no doubt promoted survival but had little effect on the increasing frailty.

The implications of the frailty model as a framework for program strategy are quite different from either the basic or the competing risk model. First, the selection of priority disease problems for intervention programs must consider not just their direct contribution to death (reflecting the case-fatality rate) but their total contribution related to the production of frailty. When this is done, the priorities may shift to conditions making the largest contribution to frailty since these inflate the case-fatality rates of all other diseases. Vitamin A deficiency in some populations may be an example which fits this criterion (Sommer, 1983).

Second, lifesaving curative interventions which do not diminish the morbidity associated with the disease are likely to be relatively ineffective in promoting child survival as well as an inefficient use of resources if the disease is recurrent (as with diarrhea). Mata's experience with the ineffectiveness of curative interventions in Guatemala (Mata, 1978, p. 165-166), which has been matched by others in the Gambia and Uganda (Rowland, et al., 1977; Cole and Parkin, 1977), illustrates this point. At the population level, the wide use of oral rehydration solution without other nutritional supplementation measures may be expected to have this result. By contrast, the dietary management of diarrhea, if it actually reduces attendant morbidity, should have a measurable impact.

Third, specific interventions which reduce the incidence of highly prevalent diseases can be expected to show a substantial demographic impact because of the joint effects on mortality and frailty. Although the data are not incontrovertible, the model would imply a large effect on measles immunization in a high mortality population because of the elimination of the associated metabolic consequences of this disease (Aaby, 1988).

Fourth, broad based preventive measures like water programs, personal hygiene, and breastfeeding promotion which reduce the incidence of multiple diseases simultaneously will show a substantial demographic impact on overall survival, even if the direct effects on individual diseases are modest (Winikoff, 1980; Esrey, 1988; DaVanzo, 1988). This is because of the combined impact of reducing both the production of frailty and competing risks (Henry, 1981).

Finally, given point 4 above, combined curative-preventive approaches are generally likely to be most effective and most efficient as survival promoting strategies. prevention will reduce incidence, enhancing the efficiency of the curative intervention,

while treatment can reduce the case fatality rates, enhancing the lifesaving effectiveness of the preventive measures.

## PROXY MEASURES FOR FRAILTY AND ITS IMPACT ON MORTALITY

There is a substantial body of evidence that diseases and nutrient deficiencies produce immunologic and physiologic derangements at the individual level which are associated with heightened susceptibility to disease and death (Solimano and Vine, 1980). Important for the development and testing of a demographic model, however, are the availability of measurable indicators at a population level of the operation of frailty to produce mortality differentials. There are, in fact, a number of qualitative and quantitative indicators which can provide the basis for empirical validation of the model.

Anthropometry, which compares the physical growth of children relative to their expected growth based on reference standards, provides the most widely tested measure of frailty. Prospective studies of children in Bangladesh, India, and New Guinea have documented the increasing risk of death with greater degrees of growth faltering (based on weight-for-age) (Mosley 1984). Evidence that his higher risk of death with growth faltering relates to an increase in the severity of disease (therefore impacting on case fatality rate) rather than on influencing the incidence comes from a prospective study by Black and colleagues (1984) in rural Bangladesh. While they found no change in the diarrhea incidence by nutritional status, there was a definite prolongation in duration of the diarrheal episode.

Vitamin A deficiency is another cause of increased frailty in populations (Sommer, et al., 1983). Physical markers for this condition include night blindness, Bitot's spots, or more severe manifestations of xerophthalmia. A report by UNICEF summarizes several studies by Sommer and colleagues in Indonesia which document the relationship between clinical signs of vitamin A deficiency, mortality, and diarrheal or respiratory disease morbidity (UNICEF, 1986, p. 23)

It has been well recognized that a number of chronic diseases have a profound affect on the risk of morbidity and mortality in older populations. The most obvious is diabetes. Among children, conditions of significant concern would be chronic parasitic infestations. Many of these like hookworm, giardia, and ascaris are rarely direct causes of death though they can contribute to increasing frailty (Bradley and Keymer, 1984). More important is malaria which, in endemic areas may only rarely be counted as a direct cause of mortality, but, because of its metabolic effects (fever, anemia, immunosuppression, etc.) it can contribute significantly to mortality from all other causes. Payne, et al. (1976) have documented the impact of a malaria control program in rural Kenya on overall infant mortality.

As noted earlier, frailty can begin the prenatal period resulting in low birth weight. In this context, it is important to distinguish between two causes of low birth weight--those related to premature termination of pregnancy and those related to infants who are born small for gestational age. Mata (1978, p. 134) carefully studied the relationship of gestational age to birth weight for 430 children born in Guatemalan villages, and then (Mata, 1978, p. 157) showed the relationship between low birth weight, gestational age, and the risk of neonatal mortality. Noteworthy, it was particularly those preterm, low

birth weight infants who are at highest risk of neonatal mortality. Of greater interest is Mata's observation that this frailty associated with fetal growth retardation persisted throughout the four years of longitudinal observation of these children (Mata, 1978, pp. 158-159, 190).

In the next section we describe preliminary work on developing a macro model for frailty. At this stage the model considers only infant mortality, and only low birth weight, respiratory disease, diarrhea, measles, malaria, neonatal tetanus, and accidents as factors affecting frailty and mortality.

### Description of the Macro Model of Frailty and Cause-specific Mortality for Infants

In this macro model of infant mortality, we postulate that the risk of death for an individual varies according to his/her frailty expressed as a relative risk (compared to the population average), multiplied by the average incidence and case fatality of specific diseases in the population. We consider two age groups: 0-5 months and 6-11 months. Table 3 gives the postulated determinants of frailty and the causes of death considered in each age interval. In the model, frailty at age 0 is a function only of birth weight. Frailty at 6 months of age is determined by birth weight (among survivors) and by the proportion of days ill with diarrhea, respiratory disease and malaria in the age group 0-5 months.

With respect to mortality, diarrhea and respiratory illness are the causes of death in the first age group that are affected by frailty while tetanus and accidents are assumed independent of frailty. In the age group 6-11 months, diarrhea, respiratory disease and measles affect mortality both directly through case fatality and indirectly by increasing the risk of death from other causes. To develop the equations of the model, we introduce the following abbreviations and notation:

<b>B</b>	=	birth weight
<b>T</b>	=	tetanus
<b>R</b>	=	respiratory
<b>M</b>	=	measles
<b>P</b>	=	malaria
<b>A</b>	=	accidents
<b>I</b>	=	incidence
<b>C</b>	=	case fatality
<b>z</b>	=	frailty (relative risk of death with the population average as referent) associated with a particular disease
<b>Z</b>	=	overall frailty from combining separate frailty components
<b>a</b>	=	age group (0 = 0-5 months; 1 = 6-11 months)
<b>j</b>	=	frailty group (j=1,2,3)

Thus:

$I(T,0)$  = incidence of tetanus among infants 0-5 months of age, and,  
 $C(D,1)$  = case fatality of diarrhea among infants 6-11 months of age.

For each disease, age group and frailty group  $j$ , there is an associated frailty and proportion in the group. Thus,

$z(D_j,1)$  = frailty of those in group  $j$  of diarrheal disease at age 6 months;  
 $p(D_j,1)$  = proportion of the population age 6-11 months in frailty group  $j$  due to diarrheal disease in the previous age group.

At the outset since "observed" mortality data are used for inputs, the frailty values for each disease are defined so that the weighted average of frailty is 1.0

$$(e.g. \sum_j z(D_j,0) * p(D_j,0) = 1.0)$$

Combining frailty and causes of death in the age group 0-5 months, and following standard procedures for relating mortality and survivorship, the proportion of infants surviving to 6 months is given by:

$$\exp\{-[Z(0)*W(0) + Y(0)]\}$$

where  $Z(0) = \sum_j z(B_j,0) p(B_j,0) = 1.0$

and  $W(0) = I(D,0)*C(D,0) + I(R,0)*C(R,0)$   
 and  $Y(0) = I(A,0)*C(A,0) + I(T,0)*C(T,0)$

Birth weight frailty is assumed to affect survival in the age group 6-11 months as well. However, the distribution of frailty at the beginning of that age group is modified by selection, from the original distribution.

Specifically,

$$p(B_j,1) = \frac{p(B_j,0) * \text{Pr}(\text{survive to 6 mo.} \mid \text{in frailty group } j)}{\sum_j p(B_j,0) * \text{Pr}(\text{survive to 6 mo.} \mid \text{in frailty group } j)}$$

where the probability (Pr) is given by  $\exp\{-[z(B_j,0)*W(0)]\}$ .

Finally, the proportion of infants who survive to age 12 months is given by:

$$\exp\{-[Z(0)*W(0) + Y(0) + Z(1)*W(1) + I(A,1)*C(A,1)]\}$$

where  $Z(1) = \sum_i \sum_j \sum_k \sum_l z(B_i,1) * z(D_j,1) * z(R_k,1) * P_l(1) * p(B_i,1) * p(D_j,1) * p(R_k,1) * P_l(1)$

and  $W(1) = I(D,1)*C(D,1) + I(R,1)*C(R,1) + I(M,1)*C(M,1)$ .

Results for this model will be compared with those from a second model having the same structure of incidence and case fatality but without frailty.

Table 4 shows input values used for frailty, incidence and case fatality. Also shown are changes in these parameters that will be modeled. With the given estimates, the cohort infant mortality rate is 90 per 1000.

### Simulating Interventions

The simulated interventions are as follows. In a first set of simulations, the relative importance of each cause of death was assessed by estimating mortality with only the one disease present in the population and then with only the one disease eliminated.

In a second set of simulations, more realistic changes were introduced. First, the proportion of low birth weight infants was reduced from 30 to 10 percent, with the distribution between preterm and IUGR infants following that derived from Villar and Belizan (1982). Second, malaria-a cause only of frailty in this model-is eliminated. Third, the incidences of diarrhea and respiratory illness are reduced separately, by one-half. For the frailty to reflect the halving of incidence, the mean proportion of days ill was determined in the original data, this was halved, and the adjusted proportions in each group were determined to yield the new mean. Fourth, the case fatalities of diarrhea and respiratory illness were halved. Finally, case-fatality and incidence of these diseases were halved.

### Results

Table 5a shows the estimate of infant mortality if single causes of death were operating.

The effect of competing risks can be seen by adding the percent of mortality due to individual causes and noting that they add to 105. From these calculations one might wrongly conclude that diarrhea represents 42% of deaths.

Table 5b shows the actual reductions from eliminating selected causes in the presence of competing causes. Here the elimination of diarrhea only reduces the IMR by 33 points ( $90 - 57 = 33$ ), instead of the 38 from Table 5a. Similar differences are seen for the other causes of death.

The elimination of malaria would lead to a 9% reduction in mortality. Reducing low birth weight from 30% to 10% would reduce the IMR by 27%. As modeled here, this occurs through its impact on frailty alone. These results are illustrative of the effects of reducing a number of conditions which do not directly contribute to death but do affect frailty.

The importance of frailty can be seen by comparing the reductions due to a 50% decline in the incidence versus a 50% decline in the case fatality rate (Table 5c). For tetanus both reductions yield a 12% reduction in IMR because tetanus does not affect frailty. For respiratory disease 50% reduction in incidence would lead to a 28% reduction in mortality while the same reduction in case fatality results in only a 14% decline in mortality. A similar pattern is seen in the case of diarrheal disease. Noteworthy, if frailty is not considered, a reduction in incidence or case fatality would have equal effects. The results of reducing both incidence and case-fatality by 50% are shown in Table 5d. As might be expected by comparing with Table 5c we see that the effects are

less than additive. Again the reason is competing risks and frailty.

### Strengths and Limitations of the Model

The purpose of the model is to illustrate the complexities in studying quantitative effects on mortality of interventions directed at specific diseases. The concepts of competing risks and frailty are crucial in this context. General models of frailty have been presented by Woodbury and Manton (1977), Manton and Stallard (1984) and Vaupel, Yashin and Manton (1988), and such models have been used to show the overestimation of potential gains in life expectancy from health interventions (Vaupel, Manton and Stallard, 1979) and the effects of selection (Vaupel and Yashin, 1985). To date, most of the applications of these models have been with respect to mortality throughout the lifetime or in the older ages. Thus, to our knowledge, this is the first attempt to consider frailty and competing causes of death in the study of child mortality.

However, neither precision of the numerical inputs nor complete adequacy of the mathematical model is presumed. With regard to the former, estimates from various populations were used as much as possible but some frailty estimates were invented. With respect to the mathematical model, since mortality risks in the absence of frailty are not observable, it can be argued that the usage of average risks in this model is problematic. Thus, though the individual frailty components were standardized to a mean relative risk of 1.0, the sums (across diseases) of the products of these separate relative risks with the associated probabilities do not yield 1.0 (e.g.  $Z(1) \neq 1$ ). The result is that the model with frailty included yields an infant mortality rate slightly different from that of a model with only the average mortality risks and no frailty.

However, the model does perform well with respect to the property of symmetry, defined as follows. When the input distribution of one frailty component is changed, the ratio of the new survival proportion to that of the original model is some value  $r$ . Now, if we had instead started with the changed frailty distribution and then introduced the original one, the ratio of these latter two survival rates should be  $1/r$ . This property was checked with the frailty distribution changes shown in Table 4. In each case the products of the two ratios were within 3% of 1.0.

### Further Developments/Needs for Further Research

In considering modification of the model, rather than have separate frailty components for each disease, it may be useful to have one frailty parameter which is determined by values of the disease incidences. In essence this would be a two-stage approach. Further use of the methods of hazard analysis by fruitful, e.g. following the suggestion of Rashad (1988). In addition, modeling of heterogeneous subpopulations will be necessary, since interventions will not reach all groups evenly. Estimation from observed data will remain problematic, however, since in real populations, frailty and mortality are virtually impossible to separate.

## CONCLUDING COMMENTS

One final point relating to this model needs to be considered as it relates to child survival program strategies. Essentially all of the discussion and the models have assumed a homogeneous population in terms of exposure to risk and hypothesized interventions. In fact, in real populations there is striking heterogeneity which is a function of social and economic factors. This can be seen not only between communities but also among families. The most obvious indicators are the differentials in child survival by mothers' education (Caldwell, 1979). There are also differences in the probability of survival among children within families which are socially determined. This is most strikingly revealed by the sex differentials favoring males in many societies (Das Gupta, 1987). An extension of this analytical model must take into account this heterogeneity.

An important consequence of this heterogeneity in exposure to risk, given that this translates into frailty, will be a widening of the differentials across subgroups in the population over time. Evidence that this, in fact, does occur comes from the prospective study in Bangladesh where one-to-two-year old children were classified into nutritional categories by anthropometry and then followed prospectively over a two-year period (Chen, 1980). The subgroup of children with the poorest nutritional status at the initial observation not only had higher mortality in the first twelve months but, in fact, had an increase in mortality in the second twelve months while the better nourished subgroups showed the expected decline in the death rates with age. Bairagi (1985) has shown that these anthropometric indices of chronic malnutrition not only reflect the relative risk of death (frailty) at the time of measurement but also, in fact, are indicative of the background social and economic circumstances of the child. Since these background characteristics persist into the future, it is not surprising that, given a cumulative increase in frailty, children in the highest risk group would die at even higher rates over time.

This interaction of social heterogeneity with frailty reflects what has been called "social synergism" (Mosley and Chen, 1984). It is this phenomenon which has profound implications for health program strategies not only in terms of the technical selection of interventions but also in the social targeting of program activities.

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**Table 1**  
**Potential Reduction in Infant and Child Deaths**

Disease	Estimated no. of deaths	Interventions	Effectiveness	Potential reduction
Immunizable diseases	3.3-5 million	Vaccines	80-95%	3-4.5 million
Pneumonia/lower respiratory infection	4 million	Penicillin	50%	2 million
Low birth weight, malnutrition	3 million	Maternal supplements Treat infections Contraception	30%	1 million
Diarrhoea	5 million	ORT	50-75%	2.5-3.5 million
Deaths	17 million		Reduction:	10 million

Source: Rohde 1983

**Table 2a**  
**Effect of TBA training and tetanus toxoid immunization on neonatal tetanus mortality in Bangladesh**

	Live births	Neonatal tetanus deaths	Neonatal tetanus deaths per 1,000 live births
Untrained TBAs	998	24	24.1
Trained TBAs	713	4	5.6
Immunization	771	1	1.3

Source: Rahman (1982: 164)

**Table 2b**  
**Effect of TBA training and tetanus toxoid immunization on neonatal mortality in Bangladesh**

	Live births	Neonatal deaths	Neonatal deaths per 1,000 live births
Untrained TBAs	998	85	85.2
Trained TBAs	713	17	23.8
Immunization	771	30	38.9

Source: Rahman (1982: 164)

Table 3

Causes of death and frailty determinants in infant mortality model.

Age group (in months)	Frailty determinants at start of interval	Causes of death dependent on frailty	Causes of death independent of frailty
0-5	birth weight	diarrhea respiratory	tetanus accidents
6-11	birth weight diarrhea respiratory malaria	diarrhea respiratory measles	accidents

Table 4

Initial values for frailty model of infant mortality and changes for simulation.

INITIAL VALUES				CHANGES	
<u>Frailty at age 0</u>					
Birth weight (B)	<u>Value</u>	<u>Prop.</u>	<u>Frailty</u>	<u>Prop.</u>	
	LBW preterm	.08	7.66	.05	
	LBW IUGR	.22	1.15	.05	
	Normal BW	.70	.19	.90	
<u>Frailty at age 6 months</u>					
Malaria (P)	<u>Value</u>	<u>Prop.</u>	<u>Frailty</u>	<u>Prop.</u>	
	Positive	.40	1.43	0	
	Negative	.60	.71	1.0	
Diarrhea (D)	<u>Value*</u>	<u>Prop.</u>	<u>Frailty</u>	<u>Prop.</u>	
	0 - 4%	.33	.5	.50	
	5 - 14%	.34	1.0	.48	
	15 - 70%	.33	1.5	.02	
Respiratory Illness (R)	<u>Value*</u>	<u>Prop.</u>	<u>Frailty</u>	<u>Prop.</u>	
	0 - 5%	.33	.4	.92	
	6 - 9%	.34	1.0	.07	
	10 - 100%	.33	1.6	.01	
<u>Causes of death at ages 0-5 months (per 1000)</u>					
<u>Cause</u>	<u>Incidence</u>	<u>Case Fatality</u>	<u>Mortality</u>	<u>Incidence</u>	<u>Case Fatality</u>
Diarrhea	2000	10	20	1000	5.0
Respir. Dis.	730	22	16	365	11.0
Tetanus	24	1000	24	12	---
Accidents	1	1000	1	---	---
<u>Causes of death at ages 6-11 months (per 1000)</u>					
<u>Cause</u>	<u>Incidence</u>	<u>Case Fatality</u>	<u>Mortality</u>	<u>Incidence</u>	<u>Case Fatality</u>
Diarrhea	2000	10	20	1000	5.0
Respir. Dis.	1400	10	14	700	5.0
Measles	100	20	2	50	---
Accidents	2	1000	2	---	---

\* Percent of days ill

Table 4 (Continued)

Sources of Inputs

**FRAILITY COMPONENTS**

**Birthweight**

- proportion 30% is near the high end of the data presented by Vella and Belizan (1982).
- frailty The relative risk of 40 for preterm LBW and 6 for IUGR LBW relative to normal birth weight were taken from Table 7.2 of Ferraz (1987).

**Diarrhea**

- proportion Terciles of the proportion of days ill with diarrhea were taken from tabulations of the Diarrhea and Growth study of Matlab, Bangladesh as described in Black, et al. 1982.
- frailty These values are a rough approximation.

**Respiratory Illness**

- proportion Terciles of the proportion of days ill were taken from tabulations of the Diarrhea and Growth study of Matlab, Bangladesh as described in Black, et al. 1982. Actually, days with fever was used since this has been shown to have more impact on frailty as measured by nutritional status (Becker, et al. 1988).
- frailty These values are a rough approximation.

**Malaria**

- proportion The proportion of infants who are positive for *P. falciparum* at regular intervals after birth is given in Molineaux and Gramiccia (1980).
- frailty The relative risk of 2.0 for those with malaria was also estimated from Molineaux and Gramicci.

**CAUSES OF DEATH**

- Diarrhea** A diarrheal mortality of 40 per 1000 was taken from Table 1 of Rohde (1983), representing diarrheal mortality in the Narangwal study.
- Respiratory Disease** The incidence of .73 is taken from Foster (1984), Table 15; it represents severe ALRI for infants. The case fatalities were adjusted to give a total mortality for infants of 30 per 1000 as suggested by Steinhoff (personal communication).
- Tetanus** The rate of 24 per 1000 deaths is from Foster (1984), Table 6 as the neonatal deaths due to tetanus when the traditional birth attendant was trained in Bangladesh.
- Accidents** The rate of 1 per 1000 is derived from the 1979 vital registration data of the Matlab Surveillance System reported in Chowdhury, et al. (1982).

Table 5

a. Model estimates of IMR by cause of death:

	<u>IMR</u>	<u>% of Total (all causes)</u>
All causes	90	100
Diarrhea only	38	42
Respiratory disease only	29	32
Tetanus only	23	26
Measles only	2	2
Accidents only	3	3
Malaria only	0	0
Low birth weight only	0	0

b. Model estimate of IMR with selective examination of specific causes of death or frailty:

	<u>IMR</u>	<u>% Reduction in IMR</u>
Reduce case-fatality to zero for:		
Diarrhea	57	37
Respiratory Disease	65	28
Tetanus	68	25
Measles	88	2
Accidents	87	3
Reduce low birth weight	66	27
Eliminate malaria	82	9

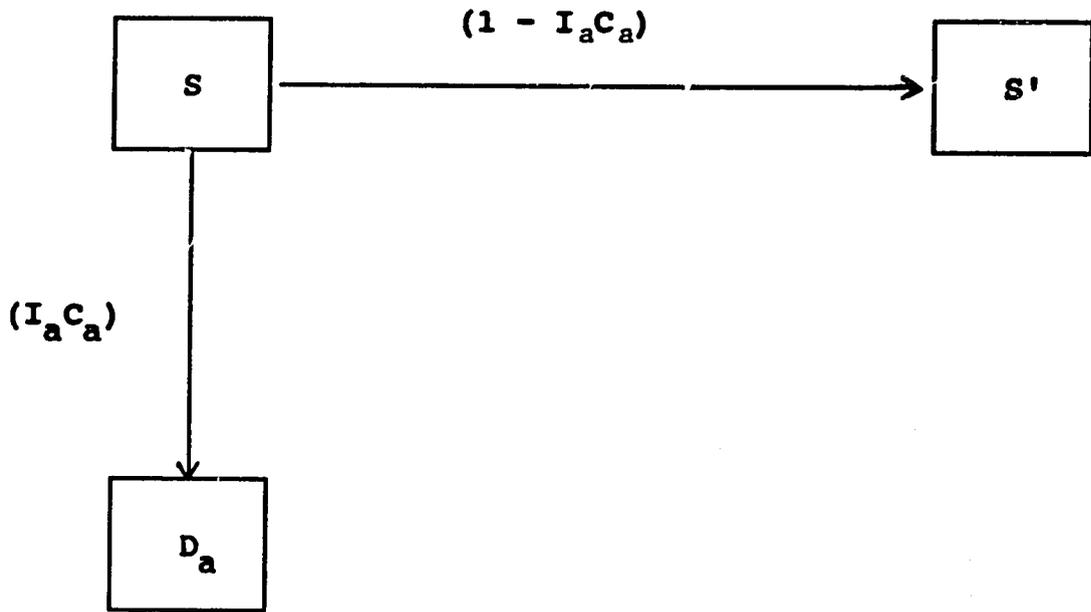
c. Model comparison of effects of reducing incidence vs. reducing case-fatality rates of selected diseases by 50%:

<u>Disease</u>	<u>Reduce Incidence by 50%</u>		<u>Reduce CFR by 50%</u>	
	<u>IMR</u>	<u>% Reduction</u>	<u>IMR</u>	<u>% Reduction</u>
Diarrhea	68	24	74	18
Respiratory Disease	65	28	78	14
Tetanus	79	12	79	12

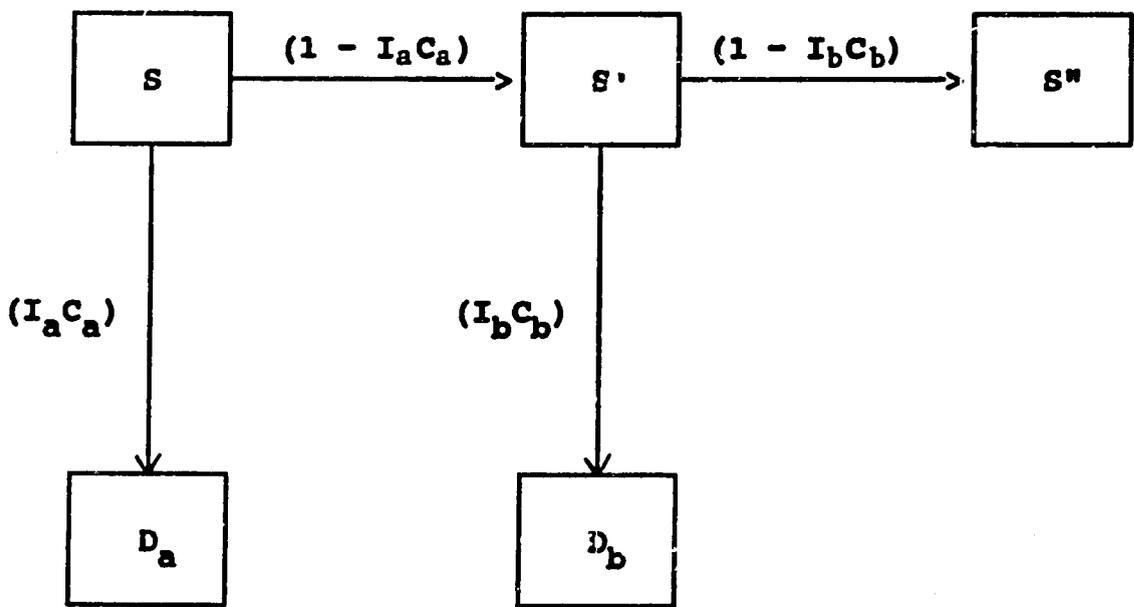
d. Model results from reducing incidence and case-fatality by 50%:

<u>Disease</u>	<u>IMR</u>	<u>% Reduction</u>
Diarrhea	61	32
Respiratory Disease	60	33
Diarrhea and Respiratory Disease	39	57

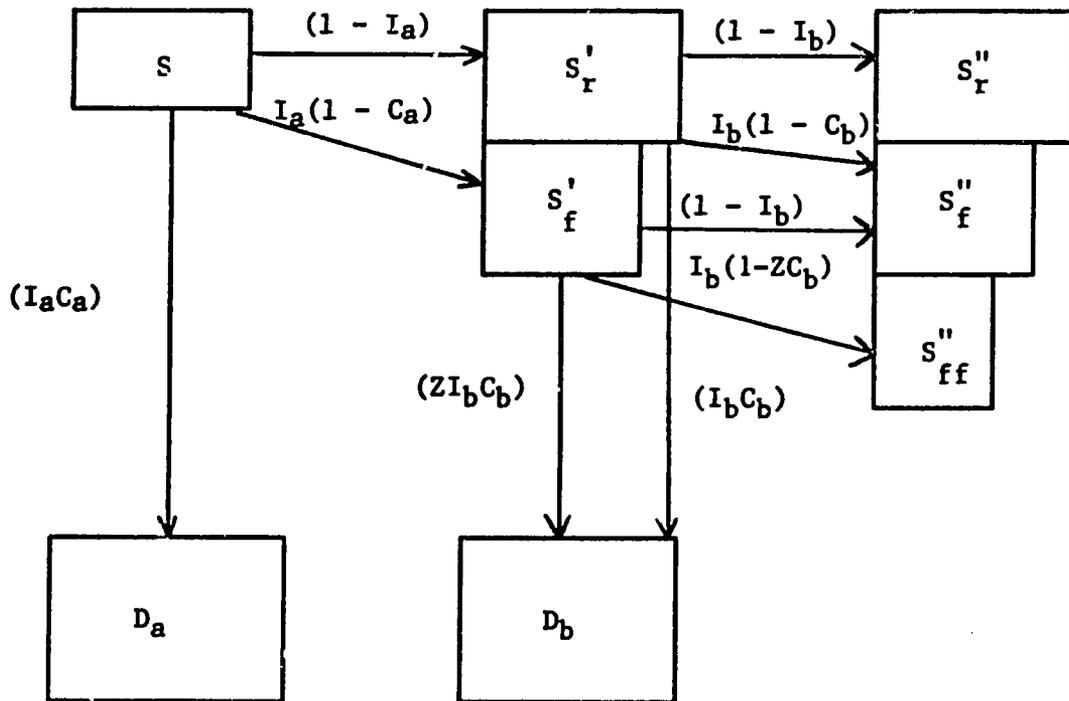
**FIGURE 1. Basic Model**



**FIGURE 2. Competing Risks Model**



**FIGURE 3. Competing Risks Model with Frailty**



# A Survey on Socioeconomic Development, Structural Adjustment and Child Health and Mortality in Developing Countries

Jere R. Behrman\*

The process of socioeconomic development involves numerous transformations of society associated with increased per capita income, increased human resources, at least initially increased market involvement, changes in relative prices, expanded governmental provision of social services, changed production structure, changes in time use and urbanization. Most, if not all of these changes may alter child health and mortality. But the process is complicated because it is embedded in the decision-making of numerous independent entities ranging from large numbers of households and extended private networks to centralized governments. In this paper I attempt to illustrate how economists think about some important issues related to this conundrum, to survey relevant recent empirical studies, and to reflect on issues that merit further exploration. Section 1 first presents a framework for organizing analysis based on the behavior of individual households and discusses some of the implications of this framework for empirical inference. Then three topics are considered within this framework: estimates of child health and mortality production functions as dependent on "inputs" such as nutrients, other health-related goods and services, time and education of the primary care giver, and endowments; estimates of reduced-form demand relations for child health and mortality as determined by such factors as prices, per capita incomes and women's schooling; estimates of the demand for nutrients and other health care inputs as determined by the same inputs. Section 2 then considers a framework for analysis and the evidence regarding the impact of economic recession and structural adjustment on child health and mortality. Section 3 summarizes the empirical evidence reviewed in the previous two sections and reflects on promising areas for future analysis.

## **Section 1. Empirical Evidence on Determinants of Child Health and Mortality in Developing Countries**

An increasing number of relevant studies exist. Before surveying them, I first consider a framework for analysis in order to organize what follows. Then I turn seriatim to survey recent empirical studies on child health and mortality production functions, child

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health and mortality reduced-form demand relations, and reduced-form demand relations for nutrients and other health-related inputs.

### Section 1.1 Framework for Analysis

The proximate loci for many of the decisions that affect child mortality are the households in which such children live.<sup>1</sup> I begin with a brief description of how economists model such households and what are the implications of such modeling for analyses. Then I even more briefly consider some considerations relating to efficiency and market imperfections. Finally, I touch upon some problems of inference on the merits of different strategies to improve child health and mortality given the nature of the available estimates even assuming that the estimates themselves are unbiased.

**1.1A. Household behavior:** A simple one-period model captures most of what needs to be emphasized even though the determination of child mortality inherently involves some dynamic decisions in a world of uncertainty. In this model parents are assumed to make the basic allocation decisions by acting as if they either maximize some agreed-upon preference function or bargain over the allocation of resources, although children -- particularly as they grow older -- presumably have an increasing say in many household decisions. While a bargaining framework appears to many to be more plausible than the maximization of unified household preferences as a motivation for economic models of the households, for most (if not all) existing empirical applications the difference is moot since the estimated relations do not justify confident identification of what the allocation mechanism is within the household.<sup>2</sup> The basic problem is an empirical one: most variables that are used in bargaining interpretations to represent the bargaining power of various parties (e.g. schooling, wage rates, income) also presumably affect productivity and the opportunity cost of time. For convenience in what follows I will refer to the maximization of household preferences, though by that terminology I do not wish to preclude the possibility that the observed outcomes reflect the outcome of some intrahousehold bargaining process that results in compromises about the preferences of all of the individuals involved in the bargaining process.

The preferences that are assumed to be maximized depend on the consumption of goods and services, the time use, and the quality of the human resources of each of the

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<sup>1</sup> Households may vary substantially in structure. For the purpose of this survey the term should not be interpreted to mean a nuclear family, but instead the unit in which children are raised. Perhaps "child-care unit" would be a more descriptive, though less standard, term for this survey. To avoid confusion, however, I stick with standard terminology and use the term household. Also household or child care unit structure may change, with substantial impact on children, although such changes are difficult to model in the same way as are decisions of existing households.

<sup>2</sup> For further discussion see Behrman (1988c), Behrman and Deolalikar (1988b), Folbre (1983, 1984, 1986), Rosenzweig and Schultz (1984).

household members.<sup>3</sup> Economists generally presume that the preferences functions are given, though some posit a dependence of preferences on childhood experiences or on consumption norms for a broader community (e.g., Easterlin, Pollak and Wachter 1980). Parental preferences regarding children within this framework often are characterized as reflecting concern about the quality and the quantity of children following the seminal contributions of Becker (1981) and Willis (1973). Quantity obviously refers to the number of surviving children, which is defined directly by births minus infant and child mortality. Quality refers to whatever attributes in a child are valued by the parents, though economists often focus (perhaps excessively) on those attributes that are reflected in expected labor market returns to the children's time over their lifetime. Parents may value the "services" produced by child quantity and quality for different reasons -- altruism, their contribution to household income while the children are still children, their expected support during the parents' old age, or maintenance of the family line. Which of these motives predominates may be important since in the development process the availability of alternatives to having one's own children for such services may change (e.g. governmental and private pension plans may reduce the attraction of dependence on one's children for old-age support).<sup>4</sup>

Preferences are assumed to be maximized subject to two types of constraints: First, there is a household full-income constraint. This states that total expenditures of the household on all goods and services for consumption, health care, other investments in household members (e.g., schooling), leisure, etc. must be less or equal to the value of the total resources available to the household. These expenditures include both the money cost and the time cost of all good and services; even if a health (or other) service is provided by the government at no monetary charge, the time cost may be considerable. The total value of the resources available to the household includes the total value of the time of all household members (which in turn depends on their human resources and on their options for time use) and on the value of all other assets, including any entitlements to transfers from the government or from relatives or friends. Second, there are a set of

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<sup>3</sup> And perhaps of other individuals outside of the household, as well, if altruism is important in interhousehold transfers, that often are substantial in developing societies. For recent analysis of the importance of such altruism and of insurance motives as an alternative explanation for such transfers, see Behrman and Deolalikar (1987b), Ravallion and Deardon (1988) and Rosenzweig (1986).

<sup>4</sup> If child mortality has costs beyond the time and monetary resource costs, preferences depend on child mortality experience in addition to the number of surviving children. This would imply an aversion to child mortality in the sense that parents would be better off with a given number of surviving children and no child mortality, than with the same number of surviving children and all else equal except for there having been one more birth and one child died. Since children who die absorb some resources, however, including child mortality in the preferences does not change the direction of predicted effects, just the magnitude. Empirically it would be hard to distinguish to what extent parents behave as if they are averse to infant and child mortality because of resource costs versus preference costs in addition to those caused by the resource losses.

household production functions, in each of which "outcomes" of interest to the household are determined by "inputs," some of which are under the control of household members. The most relevant of these for the present paper are health production functions (with mortality resulting if health falls below some critical level). The health production function for children, for example, determines their health as dependent on a number of current activities and goods and services of the household and on a number of predetermined or exogenous variables. Examples of the inputs that are determined by the household are pre-and post-natal health care, the nature and duration of breastfeeding, the nature and timing of supplementation, subsequent nutrients that each child consumes, other health inputs for each child, inoculations, the quality of water, and the time spent by the mother or others in child care. Examples of predetermined variables are the genetic endowments of the children, the schooling and capabilities of the mother and of other caregivers, the availability and quality of community health services, and the general community environment.<sup>5</sup>

The household is posited to act as if it maximized such preferences subject to such constraints to determine (perhaps with stochastic factors also playing important roles) all of the outcomes under its control, which include the health, consumption, health care, and time use of each individual and the number of births and deaths. These outcomes can be written as reduced-form demand relations in which the right-side variables are all of the determinants of household behavior that are predetermined or exogenous to the household: market prices, wages, availability of health and other services, the local environment, schooling or other form of training for all individuals for whom the gap since such time use is sufficient that they do not currently consider attending school or training programs, predetermined assets of the household or of individuals therein, entitlements from governmental programs or from friends and relatives, and the abilities and other endowments of all household members.

Conceptually the cleanest relations to estimate for the determination of child health and mortality are two. The first is the health or mortality production function along the lines described in two paragraphs above, with the right-side variables being the "inputs" that produce the health or mortality "outcome." Among these inputs are ones that reflect health policy interventions, either directly through the availability of health services or the nature of the environment or water supply/sewage system or indirectly through the prices paid for the health inputs that are used. Major problems in estimating such production function relations are four: (1) Many of the inputs are under the control of the household and are determined simultaneously with child health/mortality status (e.g., purchased health-related inputs, time devoted to health care of the mother and of other health-care providers) and must be treated as simultaneously determined in the estimation in order to avoid simultaneity bias. (2) Some important inputs always are not observed, most notably abilities and endowments and aspects of the environment. Failure to control for such

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<sup>5</sup> Whether some of these variables are predetermined or not depends in part on what questions are being asked and on what is the time period of relevance. Migration, for example, can change the local environment and community services of relevance to a child's health, but can be treated as predetermined unless it is undertaken in order to affect the child's health.

unobserved variables causes biases in the estimated effects of observed variables with which they are correlated.<sup>6</sup> Measurement errors may be substantial in both the indicators of the outcomes and the observed inputs. As is well-known, random measurement errors in the outcomes does not cause biases, but random measurement errors in the inputs tends to cause biases towards zero. Systematic measurement errors in outcomes or in inputs may cause errors either towards or away from zero. (4) Child health and mortality outcomes may respond with considerable lags to changes in the inputs, but theory provides no guidance on these lags and data often are not available to represent them well.

The second is the reduced-form demand relation for health and child mortality or for related health inputs. Among the right-side variables in these reduced-form relations are those that refer to public health interventions that affect the availability, monetary and time costs to the household of various health-related inputs, as well as the general environment. The first of the estimation problems for the health production functions is not a problem in this case since the right-side variables are not simultaneously determined if the relation indeed is a reduced form. But the problems with unobserved variables, measurement error and lags still are pervasive. In fact these problems may be more extensive for the reduced forms than for the production functions because the reduced forms include all of the predetermined variables in all of the relevant household production functions (not just those from the production function for child health or mortality) and all of the predetermined or exogenous variables in the household full-income constraint. This means, for example, that the unobserved endowments of all of the household members enters into each reduced form, including of course the ones for child health and mortality and for use of health-related inputs. It also means that all consumption prices enter into each reduced form, not only those directly related to child health determination. Moreover, if the household income generation can not be separated from household consumption because, for instance, of nutrient impacts on labor productivity, all of the relevant income production prices, assets, and endowments should be in the reduced forms.<sup>7</sup> Note, finally, that these reduced-forms allow for the possibility

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<sup>6</sup> Two important examples are individual endowments and the environment. Two recent studies provide some illumination regarding such possibilities. Behrman (1988a,d) finds that in the relatively surplus season households in rural south India parents allocate nutrients within the household to children so as to compensate for endowment differentials by giving poorer endowed children more nutrients. If the impact of nutrients on health of these children is estimated without controlling for endowments, therefore, the impact is underestimated. Similarly Rosenzweig and Wolpin (1984) find that governmental allocations of health services across space in the Bicol Region of the Philippines are allocated more intensively to the areas in which the unobserved (to the social scientist) health environment is poorer; once again, if the unobserved environment is not controlled in the estimation of the impact of governmental health services on health outcomes the estimated impact is biased downward.

<sup>7</sup> There long have been suggestions that in poor societies nutrient intakes and health may affect labor productivities. But, until recently, most empirical studies that claimed to find this result were contaminated by the failure to control for simultaneity.

of considerable substitution in the use of goods and services and time of household members in response to changes in the right-side variables. This substitution includes the technical substitution among inputs in the health production function, but goes beyond that since it may involve reallocations that change the inputs in that function. If such substitution is great, the impact of any changes in the right-side variables may be offset considerably by other changes in the household. Whether or not the substitution is great, of course, is an empirical question.

Though the child mortality production and reduced-form demand functions conceptually are the cleanest to interpret, not infrequently hybrid relations with both reduced-form (e.g. predetermined income, prices) and endogenous production function (e.g. health-related inputs that are a matter of choice) variables are estimated. Such relations are hard to interpret because the endogenous production function variable captures part of the effect of the exogenous reduced-form variable and the simultaneity of the right-side endogenous variable usually is not controlled.<sup>8</sup>

1.1B. Efficiency and distribution: Economists (at least those of a neoclassical stripe) often are concerned with efficiency. A set of outcomes is efficient (or Pareto optimal) if the situation of no one individual (or entity) can be improved without worsening the situation of someone else. The attraction of efficiency perhaps can be seen by considering a situation that is not efficient; in such a situation everyone could be made better off without making any person worse off merely by reallocation, with the same basic resources. If the costs were zero of removing all inefficiencies, it would seem attractive to do so. If all entities make their decisions regarding the use of resources on the bases of the social marginal costs of those resources, then outcomes tend to be efficient. This means that from the point of view of a concern with efficiency, it is desirable that all health services and inputs be priced at their social marginal costs so that the prices of one individual using them just equals the opportunity costs in terms of another individual not being able to use them. If health-related goods or services have

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Therefore it was not possible to be sure whether in fact they supported the impact of nutrients on labor productivity, or instead reflected that more productive individuals earned more income and purchased more nutrients. Recently, however, there have been at least four studies that report such results for rural areas in India, Sierra Leone, and Sri Lanka even with control for simultaneity (Strauss 1986, Deolalikar 1988, Sahn and Alderman 1988, and Behrman and Deolalikar 1989). Even if there is not a nutrient-labor productivity link, moreover, income generation may not be treated as separable from consumption if markets are incomplete, as is emphasized in the farm-household literature (e.g. Singh, Squire and Strauss 1986 and the references therein).

<sup>8</sup> There is not a problem if the endogenous production function variable has been introduced by solving the reduced-form relation of that variable for some exogenous variable and then substituting the resulting relation into the reduced form for child mortality and using the exogenous variable that has been eliminated in the process as an instrument for a simultaneous estimation. However this is not a common procedure (and, in fact, has no obvious advantage since it requires the same information as does the estimation of the reduced form for child mortality).

positive externalities (i.e. effects that are not transferred through the price system, such as immunization against infectious diseases), are "merit" goods for which public authorities think that they know better than do private individuals what is best for them,<sup>9</sup> or have "public goods" characteristics so that access cannot be limited to those willing to pay (e.g. spraying for insects affects everyone in a given area, not just the payers), pricing them below the marginal cost of production may be justified on efficiency grounds. Therefore there is some attraction for making prices of health-related goods and services approximate as closely as possible their social marginal costs.

But there are at least three problems with attempting to pursue such a strategy in practice. First, knowing what the social marginal costs of goods are, particularly of goods that have externalities, merit or public goods characteristics, is difficult indeed. In the absence of such characteristics (and of other factors -- such as increasing returns to scale, information imperfections, or regulations -- that limit the number of entrants in the production of the goods and services of interest), perfectly competitive markets would lead to the correct prices. But such a situation is merely a convenient analytical abstraction, not a close approximation to reality, particularly for many health goods and services. Second, pricing all health goods and services at their social marginal costs leads to efficiency if and only if all other goods and services are priced at their social marginal costs. But such a situation is very unlikely to prevail because of market failures in the rest of the economy.<sup>10</sup> Nevertheless a common article of faith among economists is that moving any particular price towards its social marginal cost is likely to be a movement towards efficiency. Third, even if efficiency were attained, that would not mean that the distribution of income and of consumption among individuals is desirable from the point of view of a social welfare function. In fact in general only if the distribution of assets is right for a given social welfare function will the movement towards efficiency result in maximization of the social welfare function. Otherwise, even though any move towards greater efficiency could result in an improvement in social welfare (since every one could be made better off), it does not necessarily do so and some movements towards efficiency (e.g. an increase in the price of a formerly subsidized basic staple) may lead to a deterioration in the well being of certain groups in society and a reduction in social welfare.

#### 1.1C. Further problems in interpreting the estimates surveyed in the rest of this

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<sup>9</sup> Merit goods raise difficult questions about paternalism and about the roles of parents versus society regarding the welfare of children.

<sup>10</sup> For example, the lack of credit markets may preclude sufficient health investments and the lack of insurance markets may prevent efficient sharing of risks for paying for major diseases. In the economics literature there is the well-known argument of the "second best" to the effect that it does not necessarily improve efficiency to set the price of a specific good at its social marginal cost unless all other prices are equal to the social marginal costs of the other goods. In fact it is possible to devise examples in which it is efficient to introduce distortions in the specific price in order to offset distortions in prices elsewhere in the economy as a second-best solution if the first-best solution of removing the other distortions is not possible.

section: I have noted several estimation problems that pertain to measurement errors, simultaneity, unobserved variable biases, and lags. But even if the estimates of child health and mortality production functions or reduced-form demand relations have no biases, there are additional problems in interpreting the implications of such estimates regarding the relative merits of alternative policies or strategies related to child health and mortality. I here note three such problems.

First, even if the coefficient estimates of observed variables are unbiased, it is possible that the more important determinants are not observed. But attribution to residuals always is tricky and often is more a measure of our ignorance than of our knowledge. Therefore the attribution of unobserved effects to factors such as genetic endowments and household and community endowments such as in studies (reviewed below) by myself (with Wolfe and Deolalikar), Bouis and Haddad (1988), Rosenzweig and Wolpin (1984) and others or to "a social consensus as to the value of educational and health goals" by Caldwell (1986: 210) must be viewed as fairly speculative.

Second, variance decomposition is not equivalent to identifying effectiveness. That a factor, such as per capita income, accounts for a small proportion of the variance in observed child health and mortality does not mean that changing it could not have a large impact on child health and mortality. The variance decomposition itself depends on the characteristics of the sample. For example, assume that the effect of social consensus on child health and mortality is great, and that countries either have high or low degree of such consensus, with different reactions in child health and mortality to per capita income increases depending on the extent of such consensus as in Figure 1, with the average reaction given by the dashed line. For both levels of social consensus, the incremental effect of income increases are constant over the whole range pictured (as reflected in the constant slopes of the lines), with a greater reaction in the high than in the low consensus case. However, the measured relative contribution of per capita income to child health and mortality depends very much on what range of per capita income change is considered for the estimate. To see this, note that the variance of child health and mortality over an income range can be divided into the variance along the average curve (that due to per capita income) and the variance around the average curve (that due to social consensus). Therefore, for two equal increments of income (say  $Y_1$  to  $Y_2$  and  $Y_3$  to  $Y_4$ ), the measured related contribution of per capita income to the observed variance can differ substantially (being much larger for  $Y_1$  to  $Y_2$ ) even though by assumption the marginal impact of income on child health and mortality is constant. Thus, the variance decomposition between income and social consensus is not very informative about the impact of income changes.<sup>11</sup>

Third, the estimated parameters do not indicate what the costs and lags are of changing the right-side variables. If the impact of women's schooling is relatively large, as compared to those of changing community health facilities, that in itself is not compelling evidence that the rate of return is higher to increasing women's schooling rather than to improving community health facilities. The latter may be enough cheaper,

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<sup>11</sup> This argument is analogous to that of Feldman and Lewontin (1975) and Lazer (1974) on partitioning phenotypic variance into genetic and environmental components. Also see Behrman, Hrubec, Taubman, and Wales (1980).

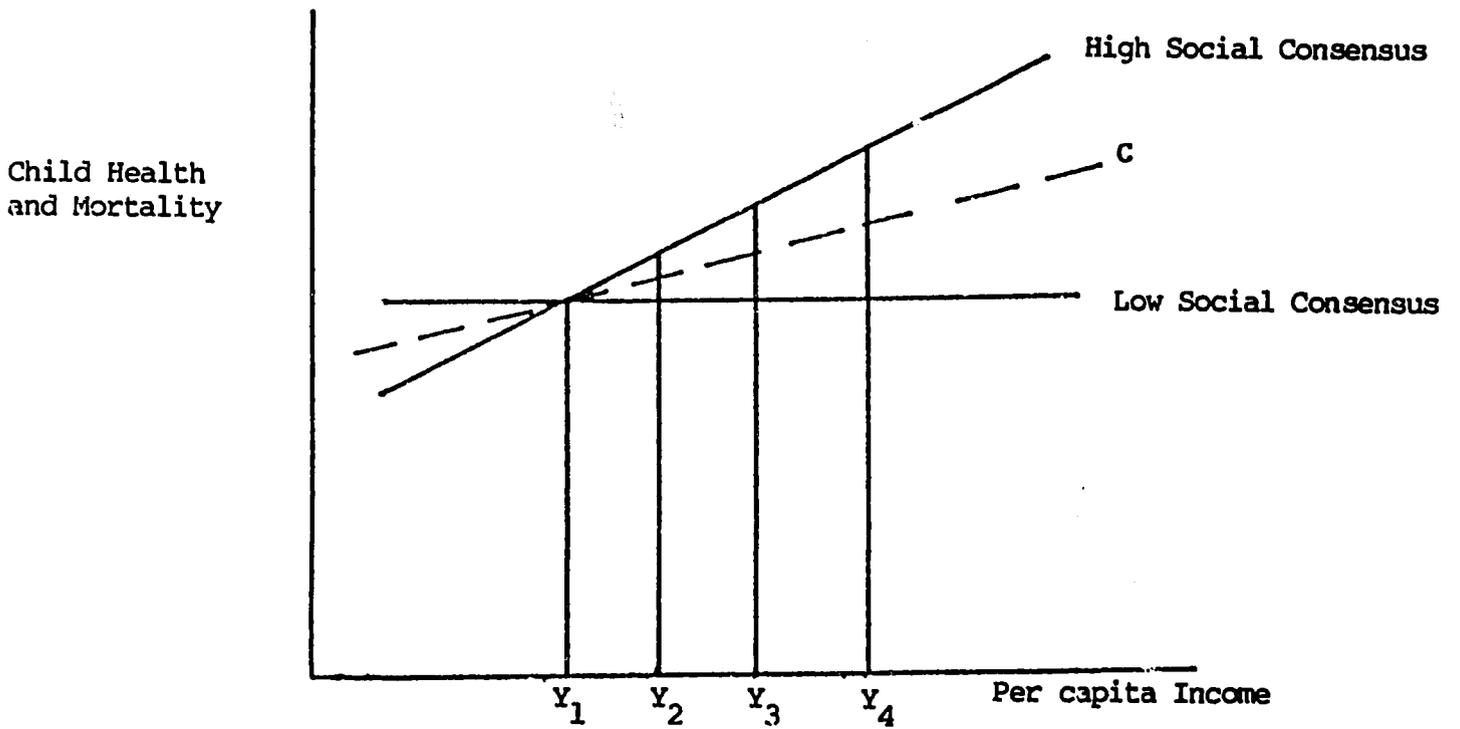


Figure 1. Hypothetical reaction functions of child health and mortality to income given high or low social consensus

and the gestation enough shorter, that the rate of return may be higher to improving community health facilities than to increasing women's schooling.

## **Section 1.2. Empirical Estimates of Child Health and Mortality Production Functions<sup>12</sup>**

Child health and mortality production functions are technical relations between a number of inputs, including nutrients and other directly health-related inputs, and outcomes related to health. The outcomes sometimes are positive measures of health such as anthropometric indicators and clinical measures of physical well-being, and sometimes are negative indicators of health such as morbidity and, in the extreme, mortality. Though the interest in this paper primarily is in child mortality, clinical studies have found fairly strong associations between anthropometric and morbidity indicators on one hand and child mortality on the other, so a broader perspective is adopted here.<sup>13</sup> For the purpose of this paper the primary questions of interest are what inputs that plausibly may be affected by the development process have significant impact on child health and mortality outcomes, and what is the extent of such effects?

The impact of nutrients and other health-related inputs on anthropometric, clinical, morbidity, and mortality indicators of health have been explored for both experimental and nonexperimental data sets.

Experimental investigations, such as those based on the INCAP project in Guatemala (conducted between 1969 and 1971) and the Narangwal project in Punjab, India (conducted between 1968 and 1973), have compared the morbidity of individuals in

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<sup>12</sup> In this and subsequent sections I survey a number of recent studies. However the number of related studies in various disciplines is so enormous that it would be far beyond the scope of this paper to include all of them. Therefore I have attempted to focus on studies that can be interpreted within the framework outlined in Section 1.1. This, plus my own disciplinary expertise, leads to a focus on studies by economists. I apologize for missing some possibly relevant interesting studies, probably particularly ones by noneconomists.

<sup>13</sup> A few of the studies that establish a link between anthropometric measures and mortality are Bairagi (1981), Chen et al. (1980), Kielmann and McCord (1978), Trowbridge and Sommer (1981), and Smedman, Sterky, Mellander and Wall (1987). Martorell and Ho (1984, p.61) provide a good survey of this literature and conclude that "the studies reviewed here all show that severely malnourished children (i.e., those with lower-than-average anthropometric readings) also showed increased mortality risks... Arm circumference was found to be an excellent predictor of mortality by all authors who included this measure." Black (1984), Bradley and Keymer (1984) and Foster (1984), among others, show that morbidity, particularly in the form of parasitic and immunizable diseases (such as diarrhea, tetanus, and measles), significantly increase the risk of mortality among children and infants. But there certainly are doubts about the importance of the associations among anthropometric measures, morbidity, and mortality such as in Aaby's (1988) recent work on measles mortality in Guinea-Bissau.

villages benefiting from nutritional supplementation programs with those in control villages not having such programs (Martorell and Ho). Clark (1981), for example, found that the physical growth of infants (weight gain up to 12 months) in the INCAP villages was associated significantly ceteris paribus with the number of calories of atole (a high protein-high calorie diet supplement introduced in one of the villages) consumed and length of breastfeeding. Taylor et al. (1978) found that (controlling for factors such as age, gender, caste, season, and number and composition of siblings) children in the Naragwal project villages who received nutritional care, whether alone or in combination with medical care, had the highest weights and heights, those in villages with medical-care alone the next highest, and those in control villages the lowest. Chernichovsky and Kielmann (1977) used the same data and found a significant positive effect of calories on weight of children aged 6-36 months with age and gender controls in two-stage least squares estimates.

Nonexperimental socioeconomic household survey data have been used to explore production function determinants for a number of different indicators of child health outcomes in a fairly large number of studies.

Sirilaksana (1982, 1985), for instance, estimates child health production functions<sup>14</sup> for 1006 preschool children and 410 children aged 7-13 in school, 582 households in 12 villages in Northeast Thailand. She is explicitly aware of possible simultaneity bias and treats many of the variables that would seem to be determined by the household (but surprisingly, not food intake) as simultaneously determined. She finds mixed signs for (simultaneously-determined) births and deaths (with the latter negative for younger and positive for older children), maternal age, and (not simultaneously-determined) food intakes and with nothing significant in her child mortality estimate. Months breastfeed, when significant, is negative, which she interprets to reflect inadequate supplementation with long breastfeeding. Predicted incidence of fever (but not of diarrhea nor other diseases), has significantly negative impact, suggesting important disease-anthropometric indicator interaction.<sup>15</sup> Community health programs and prenatal care are insignificant in the disease relations. Her most interesting finding is that the predicted hours mothers worked in the formal sector worsened significantly and substantially and predicted participation in the informal sector improved significantly and substantially child health as represented by three anthropometric indicators (in the sample as a whole and generally even for disaggregated samples by age of children). She interprets this pattern to reflect that informal sector work (generally in the home) can be combined easily with child care, but not formal sector work. She does not include mother's education explicitly in the estimates for anthropometric indicators (it is insignificant in probit estimates for disease incidence) so one might wonder whether these time-use variables in part are not proxying for mother's education since that is one of the instruments used for their predictions. But

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<sup>14</sup> Though in relations for disease incidence she includes household income which makes the relation a hybrid of the type mentioned in Subsection 1.1A above and obscures the interpretation.

<sup>15</sup> Predicted anthropometric indicators, however, are not significant in probit estimates for disease incidence.

since in the first-stage estimates mother's education enters negatively into both her formal sector hours and her informal sector participation, omitted variable bias due to the exclusion of mother's education from the production functions does not seem to explain the importance of time use in her estimates. Her time-use results are further reinforced by her finding that predicted mother's wage has a significantly negative impact on household food consumption, which she interprets to reflect the negative impact on nutrient intakes of higher opportunity cost of women's time. This is perhaps the most impressive evidence of which I am aware of the importance of mother's time use in child health production, though not for child mortality.

Magnant et al. (1985) analyze the determinants of child weight (treated as a dichotomous variable, with a value of one if the child weighs 75 per cent or more of the age-gender standardized weight and zero otherwise) for a sample of 1,500 children aged 1-59 months in the Philippines. Using ordinary-least-squares procedures (and not logit or probit, which would be more appropriate given the dichotomous nature of the dependent variable), they find that breastfeeding *ceteris paribus* reduces significantly the probability of the child weighing less than 75 per cent of the standardized weight. However, since breastfeeding is not treated as a choice variable in their analysis, their results may be biased. Indeed, their results simply may reflect that the sickest children or mothers may be least able to breastfeed.

DaVanzo and Habicht (1984) exploit the retroactive nature of their Malaysian data by estimating a fixed-effects logit model for infant mortality. They find that decreases in the durations of both supplemented and un-supplemented breastfeeding led to an increase in infant mortality between 1956-60 and 1971-75. However, increases in maternal education and in piped water availability (particularly for women who did not breastfeed) resulted in large (and more than offsetting) declines in infant mortality over the same period. By estimating the model in first-differences, DaVanzo and Habicht purge their estimates of unobserved additive household and community unobserved endowments, health management and taste effects. However, they do not treat duration of breastfeeding as a choice variable, thus making their estimates possibly susceptible to simultaneous-equations bias of the sort illustrated in the example in the previous paragraph.<sup>16</sup>

Wolfe and Behrman (1987b) estimate child anthropometric indicators (standardized weight, height, and arm circumference) and infant mortality production functions for the children of a 1977-8 Nicaraguan adult sister sample. Their standard estimates suggest a strong positive impact of women's schooling on child health, though not a significant effect of calories and breastfeeding.<sup>17</sup> When they control for mothers' unobserved

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<sup>16</sup> The control for unobserved fixed effects, however, may reduce the dangers of simultaneity bias.

<sup>17</sup> Their standard estimates use ordinary-least-squares procedures. However they report that when they use simultaneous estimators for calories and length of breastfeeding, women's schooling has even less estimated impact on child health than in the ordinary-least-squares estimates, apparently because it is highly correlated with the instrumented estimates for calories and length of breastfeeding.

childhood-background-related characteristics through adult sister deviation estimates, however, the coefficient estimate of mother's schooling is not significant. This suggests that in the standard estimates, mother's schooling is a proxy for her unobserved characteristics. Only the negative effect of duration of breastfeeding on child weight is significant at the 15 percent level in the deviation estimates. Behrman and Wolfe (1987) estimate a latent variable simultaneous equations system for the same data, including a child health production function together with some reduced-form demand relations. Standardized height, weight, and biceps circumference are used as the observed indicators for child health. Medical-care usage (i.e., the age-standardized number of injections received by the child, the term of the mother's first pregnancy-related medical examination, and social security coverage), household nutrition (i.e., standardized household intakes of calories and protein and whether the household owned a refrigerator), and water and sanitation facilities (i.e., the absence of indoor toilets and baths) are included as endogenous inputs. Household income, the mother's initial endowments (i.e., her own mother's schooling, whether her upbringing was rural, mother present in adolescence, father present in adolescence, and number of siblings), and community endowments (i.e., population, population density, number of hospital beds per 1,000 inhabitants, and the literacy rate) are some of the instruments used to identify the parameters of the production function. Behrman and Wolfe's results are somewhat discouraging for standard analyses: medical-care usage, nutrition and mother's schooling appear to have significant positive effects on child health if mother's childhood-family related endowments are excluded a priori, but all of these coefficients become insignificant if mother's childhood-family-related endowments are included. They interpret such endowments to include health-related abilities, knowledge and habits and prior health status, all of which relate to usually unobserved (and therefore uncontrolled) dimensions of mothers' childhood family background. Thus they conclude that the standard results about the positive health impact of nutrition, water and sanitation, and maternal schooling may be misleading due to the failure to control for maternal endowments.

Khan (1983) uses simultaneous techniques to estimate the impact of household per-capita calories on individual standardized height and of height on the number of sick days for boys and girls aged two to five years (as well as other groups) for a Bangladeshi sample. However his identification restrictions are arbitrary, so it is not clear whether his simultaneous estimates are better than ordinary-least-squares estimates. He finds that calorie intake is not a statistically significant determinant of height nor height a significant inverse determinant of the number of sickdays.

Blau (1984) develops his estimation model, including his choice of instruments and identification restrictions, from a well-specified theoretical framework that he uses for children under five years of age in the same 1977-8 Nicaraguan sample that Behrman and Wolfe use. He uses food prices and the woman's characteristics as instruments (with arbitrary exclusions) to identify the effects of breastfeeding duration (which he finds to be significantly negative) and average food expenditure per adult equivalent in the household (which he finds to be insignificant) on the standardized height of children.

Cohen (1988) estimates health production functions for age-sex-standardization height and weight for about 600 children under age five in urban Juba, Sudan in 1983. The right-side variables include three that are treated as endogenous (food expenditure per

adult equivalent, number of vaccinations, and the probability that a child in the household was ill the previous week) and three that are treated as exogenous (community variables reflecting probability of child contracting diarrhea, access to piped water, and child age).<sup>18</sup> The only significant effects on weight are positive ones of vaccinations. On height there are positive effects of vaccinations and piped water, and negative ones of the probability of contracting diarrhea (the last of which seems surprising since height is a longer-run measure and the diarrhea variable is for the last week). The results point to the lack of importance of food intakes (though measurement error may be masking a true effect), the positive impact of vaccinations (and perhaps correlated choices of the household) and piped water.

Barrera (1987a,b) uses simultaneous techniques and controls for truncation (due to ongoing breastfeeding) to investigate the impact on height-for-age of breastfeeding and supplementation for a sample of 498 children below 25 months of age in the 1978 and 1981 Bicol Philippines Multipurpose Survey. He finds a definite nonlinear effect of breastfeeding on child health, with a maximum benefit obtained with four-five months of unsupplemented breastfeeding and complete weaning at seven-eight months. These results contrast with some of those summarized above, suggesting that some previous studies may be misleading due to a failure to allow for nonlinearities, though his estimated developmental paths for older infants are so low as to be questionable. He also finds that more-educated mothers are better able to provide wholesome substitutes to breastmilk without producing ill effects and thus breastfeed less long.

Rosenzweig and Wolpin (1988) develop a simple dynamic model of child health that incorporates unobserved heterogeneity across households and uncertainty regarding unobserved heterogeneity in each child's health endowments prior to birth. They compare estimates using OLS versus fixed-effects procedures to control for heterogeneity for child health relations based on data from 109 households with two or more children under six in Colombia for 1968 to 1974. The dependent variables are the age-standardized weights of the children at birth and within six months of birth. The right-side variables include birth order, birth spacing and timing, per capita family food consumption, inoculations (DPT), breastfeeding, maternal age, and child sex, all except the last of which are treated as endogenous in a lagged instrumental variable fixed effects (LIFE) estimation alternative. They interpret their results to show that control for unobserved heterogeneity alters the statistical inferences substantially; in particular, they point to the -8% effect of birth order on birth at weight without controls, as compared with a -24% effect in their preferred estimates and to a 49% greater effect of prior birth interval on birthweight in their preferred estimate than in the estimates without control for heterogeneity. They also note that breastfeeding has positive coefficients on subsequent age-standardized weight without control for heterogeneity, but negative coefficients with control for heterogeneity. They then use their estimated relations to calculate unobserved family- and child-specific endowments (by averaging over the appropriate residuals). They find that family health

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<sup>18</sup> The use of simultaneous estimates makes a lot of difference. In contrast to the results summarized below, piped water appears much more important and various less in ordering least squares estimates.

environments are significantly correlated with parental education and family income, which implies that estimates of child health outcomes that do not control for such endowments have upward-biased coefficients since in part such variables are proxying for the uncontrolled health endowments. These results are suggestive of possible downward biases in the birth order and birth interval impacts on birth weight and upward biases in the breastfeeding, parents' schooling, and family income estimate effects on child weight if there is not control for unobserved household and child heterogeneity. The results are hardly conclusive, however, in that the coefficients estimates have such large standard errors that they do not appear to differ significantly depending on the controls for heterogeneity. However an overall test indicates that the unobserved heterogeneity is significant. Moreover, the original health production function appears to be misspecified due to the exclusion of maternal schooling and time; for the fixed-effects estimates, however, the impact of at least maternal schooling is controlled so that the association between the estimated household endowments and mother's schooling is not an artifact of the exclusion of schooling from the health production functions.

Rao (1989) uses cross-country data from the International Comparison Project (with its consistent treatment of prices, quantities and purchasing power parity incomes) and the World Bank to estimate health production functions for life expectancy at birth, life expectancy at age five, and child and infant mortality for 57 countries (43 developing countries) in 1980. Each of the production functions includes: per capita consumption of vegetables, dairy products and eggs; per capita consumption of meat and poultry; per capita consumption of staples; per capita consumption of public and private medical services; and adult literacy rates. The first four of these variables are treated as simultaneously determined, with prices and income used as instruments.<sup>19</sup> The estimates suggest a strong significant positive effect of adult literacy on life expectancy at birth and (negatively) on infant and child mortality, but not on life expectancy at age five. Thus adult literacy seems important for infant and child survival but not for older individuals. Per capita medical expenditures are significant with the a priori anticipated signs for all four health indicators, but again are much more important for younger individuals (i.e., the elasticities of life expectancies with respect to medical expenditures are about .06 at birth and about .01 at age five). Per capita food expenditure and food composition have some significant effects: meat consumption has negative effects on infant and child mortality, with the former somewhat surprising unless the effect is through the health of lactating mothers; vegetable consumption has positive effects on life expectancies, again larger for those at birth than at age five, but only if the literacy variable is excluded; vegetable consumption also has a priori surprising positive effect on infant mortality when adult literacy is included. In summary, subject to the standard caveats about crosscountry studies and possible omitted variable biases, this is a careful exploration that suggests that medical expenditures, adult literacy, and some elements of food consumption have important effects on major health indicators, particularly for infants and small children. But the sum of the elasticities of the included inputs is only about 0.15, so large changes in them are required to have much impact on infant and child mortality and life

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<sup>19</sup> A number of the estimates differ in apparent importance when ordinary-least-square procedures are used.

expectancies.

Thus, such studies lead to some limited insights about the determinants of child health and mortality and their relation to economic development. Improved nutrition has estimated positive effects on health in some studies. If development leads to improved nutrition through increased food expenditure, therefore, health for children would seem probably to be improved.<sup>20</sup> Breastfeeding duration has positive effects in some cases such as da Vanzo and Habicht's (1984) work on Malaysia and Magnant et al.'s (1985) Philippine study, but negative effects in Sirilaksana's (1982, 1986) work on Thailand and Blau's (1984) and Wolfe and Behrman's (1987b) studies on Nicaragua, and nonlinear (first positive and then negative effects in Barrera's (1987a,b) estimates for the Philippines. The Rosenzweig and Wolpin (1988) estimates for Colombia, moreover, suggest that the positive impact of breastfeeding in some studies may be overstated due to the failure to control for unobserved health endowments since inherently more healthy children are more likely to be able to breastfeed. The probable decline of breastfeeding with development due to the greater accessibility and reduced cost of alternatives and the higher opportunity cost of women's time, therefore, may affect child health in either direction, with the effect more likely to be negative the longer the breastfeeding duration. There is more limited evidence that the increased use of modern medical inputs, improved water and sanitation, and increased women's schooling<sup>21</sup> -- all associated with the development process -- improve children's health.

But even some of these results do not seem all that robust to estimation procedures. In the Behrman and Wolfe adult sibling and latent variable estimates, for example, the exploration of more sophisticated models leads to considerable questions about to what extent the reported significant health impact of women's schooling, nutrients, and water and sanitation in other studies may be misleading because of the failure to control for unobserved women's background variables.

Of course our limited knowledge of the child health and mortality production functions in developing countries in part probably reflects data inadequacies in the analysis. Some of the health and nutrient indicators, for example, are for very short periods and thus likely to be very noisy. In many cases data on the environment and on community endowments are quite limited or nonexistent. As Behrman and Deolalikar (1988c) emphasize, moreover, specifications for the estimated health production functions do not include the individual's own time use (through Sirilaksana 1982, 1986 includes the mother's time use) in contrast to the specification suggested in Section 1.1 and thus may miss that better nutrition largely may lead to greater productivity rather than affecting

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<sup>20</sup> But recent studies suggest that the estimated response of nutrients to income is quite small if substantial food substitution is not precluded a priori by aggregation and if measurement errors and leakages between food purchases and nutrients consumed by household members are controlled. See Section 1.4.

<sup>21</sup> Cochrane, O'Hara and Leslie (1980, 1982), in an extensive earlier review, came to a more definite positive conclusion about the positive impact of women's schooling on children's health. But their review does not include any studies that control for unobserved maternal endowments.

longer-run health indicators.<sup>22</sup> The only available evidence for children along this line of which I am aware are the studies by Jamison. But none of these studies control for the simultaneous determination of nutrient intakes, so they may reflect only that children from better backgrounds both have more nutrient intakes and do better in school. Likewise, the well-known impact of diseases such as diarrhea on the capacity of the body for utilizing nutrient intakes is not incorporated into most analysis, though Sirilaksana (1982, 1986) does include predicted values for disease and finds them insignificant except for fever. As suggested in the previous paragraph, finally estimation problems regarding simultaneity, unobserved variables, and lagged effects may be considerable for many of these studies.

### **Section 1.3. Empirical Estimates of Child Health and Mortality Reduced-Form Demand Functions**

Section 1.1 discusses the alternative of considering the impact of development on child health and mortality through estimating child health and mortality reduced-form demand functions. In certain respects, in fact, a priori such estimates might seem more promising than the production function estimates. Some of the data problems may be less since, for example, data on individual consumption of nutrients are not required. Simultaneity bias is not a problem for true reduced forms, though the a priori specification of variables such as income as exogenous may not be warranted (see Section 1.1). And the right-side variables are ones affected more directly by development than are many of those in the child health and mortality production functions. Therefore it may be surprising that there are relatively few studies on child health and mortality demand in the developing countries. Moreover, since most of the demand studies are based on cross-sectional data with little market price variation, few of them explicitly include market prices as explanatory variables. For the most part, they include household characteristics, income and availability of health care, the latter, of course, being interpretable as related to the price of health services. I review first micro studies based on household data and then some aggregate cross-country estimates.

Harbert and Scandizzo (1982) estimate linear health demand relations for weight and height from data on 1551 children 19 years old and younger in 400 rural and urban households in central Chile in 1974-5, controlling for age and sex. They find a significant impact of income on height, but not on weight. They are able to include parents' height or weight for a subsample of 827 children. Both father's and mother's weight have significant positive coefficients of about .03 in the children's weight relation, and father's weight has a positive significant one of about .05 in the children's height relation. The equal or greater significance of the father's characteristics than of the mother's suggests at least in part a genetic or general family environment explanation, and not a household productivity story since that would seem to imply greater significance for the women's characteristics given time use patterns. The income effects appear larger and significant in both relations (rather than just for height) once parental anthropometric

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<sup>22</sup> Though it also may lead to higher basal metabolism through the adjustment processes discussed by revisionist nutritionists such as Payne (1988) and Sukhatme (1982).

characteristics are included. This is somewhat surprising since in the full sample it would seem that income might be proxying in part for genetics and household environments, so the coefficient estimates might be upward biased. Of course the result also might reflect the nature of the selectivity for having anthropometric information on the parents; unfortunately Harbert and Scandizzo do not provide any information by which to make a judgement about such a possibility. In both of their sets of estimates, they include family size (which has a significant negative coefficient in three of the four regressions), but they do not control for its simultaneous determination, so it is not clear how to interpret its effects. Finally, they do not control for prices, except for residence, in which case they find that urban residence is associated significantly with greater height, but not weight.

Sahn (1988) estimates reduced-form demand equations for Z scores for child height and weight for 3,323 children under age six in rural and urban Ivory Coast in 1986-1987 with an instrumental variable control for simultaneity of income. He finds strong income effects for height with an elasticity of 0.87 for the poorest quintile of households in urban areas and of 0.28 for the poorest quintile of households in rural areas, but no significance for weight. Mother's schooling, in contrast, has significant effects on weight, but not on height. Father's schooling is not significant in either case. Mother's height has a positive impact on height, but not on weight, in both urban and rural areas. Father's height has a significant positive impact on height in urban areas that is about half as large as that of mother's height, but a puzzling significantly negative association with weight in rural areas. Thus the results suggest some importance of household income and parental, particularly maternal, human capital on child anthropometric measures. But there is some ambiguity, and no control for possibly important unobserved household and community fixed effects.

Horton (1986) estimates reduced-form relations for age-standardized height of children under 15 years old in 901 households in Bicol, Philippines in 1978. She finds no significant effect of assets on the household average child height, but somewhat peculiarly because of the contrast, a significant (though small) impact on individual children. Mother's and father's height are significant, with no significant difference in the coefficients, presumably representing primarily genetic and environmental endowments that usually are not controlled in such studies. Father's education has significant positive effects, but mother's does not, in contrast to many conjectures. Several community variables are insignificant, but the nature of water supply (which arguably might be endogenous) has positive effects. In the individual relations birth order has a negative impact, but -- again contrary to frequent conjectures -- girls fare better than boys.

Barrera (1987a, 1988) also presents estimates of height standardized by age for 3821 children under 15 years of age in the 1978 Bicol, Philippines Multipurpose Survey and 1981 Supplementary Survey. His right-side variables include five market prices (drugs, rice, cooking oil, kerosene, milk); income (other than the mother's earnings); the mother's schooling, age, and height (the last being an indicator of genetic traits and health endowments); the child's age and sex; and six community characteristics (community wage rate for women, village vs. town, travel time to least-cost child outpatient facility, predominant type of toilet used, predominant water source, and absence of excreta). He finds almost no evidence of income or market price effects, nor of sex discrimination (except that older girls are favored over boys). Maternal height and age have large and

significant positive coefficient estimates, the former of which he interprets as endowments and the latter as experience. Mother's schooling has significant positive effects that are larger for younger children. Mother's schooling also has greater substitutability with both a cleaner environment and health-care access for younger children. But the effect of mother's schooling is substantially less when mother's height is included (i.e., 26 to 81% less, depending on the age group), which suggests that mother's education in standard estimates proxies substantially for unobserved endowments for which mother's height is a partial control.

Several questions are raised by considering both of these two studies with the Bicol data. Why do they have such different results in regard to the significance of mother's schooling? Why is only height examined as a health indicator, particularly since the price variables a priori would seem to be more related to shorter-term indicators of health that are available in the data set, such as weight? Is there a selectivity problem in that anthropometric measures are not available for all children (particularly older sons) in the households? Do the included variables represent sufficiently well household and community endowments?

Thomas, Strauss, and Henriques (1987) estimate relations for height-for-age and weight-for-age for children under nine and for child survival for 41,233 households in Brazil in 1974-1975. They are quite sensitive to specification issues, estimation problems, and whether subdividing the sample differently (e.g. by child age, region, urbanization, father's presence) makes a difference. They find strong positive effects of parents' schooling on both child height and child survival, though with that for the father weaker than for the mother in the survival relation. Maternal height also has a large effect on child height and survival. Income has a significant nonlinear, though small effect. There are some important regional and urbanization differences, with mothers' education and household income having a greater impact on child survival (and the latter on their height) in the poorer Northeast than in the richer Southeast and with mother's height a more important determinant of child survival outside of the highest income area of the urban Southeast rather than within it. They also find less effect of mothers' characteristics on child survival for younger cohorts of mothers, which they suggest may reflect important substitution between community and household factors. Their study has some limitations: the absence of prices or specific representation of community factors, the failure to control for selectivity due to missing data on children, the limited control for unobserved endowments, the failure to control for the fact that less-educated women tend to have children when they are younger so such children are exposed to more mortality risk at any given age for their mothers. But all-in-all it is an interesting effort that suggests the importance of women's education and women's endowments as proxied by their height and, to a lesser extent, those of men and income in determining child height and weight and survival, probably with important interactions with community factors.

Thomas, Strauss and Henriques (1988) explore how the apparent importance of mother's education on the height of 1378 children under age six in Northeastern Brazil in 1986 varies depending on other controls and attempt to identify thereby what mother's education really is representing in such relations. In their simplest ("naive") estimates, for rural areas, a child's log height increases by about 0.5% with each additional year of maternal education and 0.14% with each additional year of her partner's education.

Inclusion of a number of other household variables (the household's nonparental income; the women's literacy, whether she regularly listens to radio, watches TV, and reads the paper) and control for observed and unobserved community fixed characteristics reduces the coefficient on mother's education to 0.056%, though jointly the education, literacy and information variables are significant. They conclude that much of the effect of maternal education on child health in rural areas is "transmitted through...better information gathering or processing [and]...through the presence of health services and infrastructure in the community" (p. 17). In parallel estimates for urban areas, the "naive" results indicate less impact of maternal education than in rural areas (0.28%) and more of the partner's education (0.21%). In their fullest specification including fixed effects, these two coefficients fall, respectively, to 0.16% and 0.15%. They conclude "that failure to include indicators of community service availability and, to a less extent, income results in biased estimates of the impact of parental education on child height -- at least in rural Northeast Brazil" (p. 20). Unfortunately, with the estimates that they present, it is not possible to tell how much of the reduction in the estimated coefficients of maternal education in their fuller specification is due to education working through the mothers literacy and information variables and how much due to education proxying for community characteristics. Most of the effects of the community variables on child height, moreover, are due to the unobserved variables controlled in the fixed-effects estimates. Among 12 observed community characteristics (about half pertaining to health services), only a strong negative association of the proportion of households with earth floors in dwellings in rural areas is significant at the standard 5% level. In the rural (but not the urban) areas, finally, the impact of income seems to be positive and robust, but not large. A 10% income increase apparently implies an increase of about 0.1% in child height.

Blau (1984) estimates a demand function for age-standardized height using 1977-78 Nicaraguan data on children under five years of age. He includes the mother's age, education, urban origin, other income, and predicted formal and informal sector women's wage rates (corrected for selection bias) as independent variables. Blau's rationale for separating the two types of wage rates is that female informal-sector jobs in developing countries may be combined with own childcare in a way that formal-sector jobs can not (see the discussion of the Sirilaksana study on Thailand in Section 1.2). If so, the substitution effect of the mother's formal-sector wage rate on child health should be negative and that of the informal-sector wage rate should be zero. After allowing for a positive income effect, the gross impact on child health of the informal-sector wage rate should be greater in magnitude than that of the formal-sector wage rate. Blau's results, however, indicate the opposite: the mother's (predicted) formal-sector wage rate has a significant positive effect on child health and the informal-sector wage rate has an insignificant though positive effect. The formal-sector wage result by itself suggests that a strong income effect is overriding the price (wage) effect through childcare, but the insignificance of the informal-sector wage is puzzling and calls for caution in interpreting the formal-sector wage estimate as implying a strong income effect. Neither women's education nor other income has a significant impact.

Simmons, et al. (1982) estimate logit conditional reduced-form relations for infant and child mortality, separate by age groups (e.g., first year and second and third year mortality) and by sex for 1980 children born in 1965-69 to 2064 couples living in rural

areas of Uttar Pradesh in Northern India. Their specification includes two variables related to health environment (i.e., time to hospital, village 3-year survival rate), parental education, income other than from the parents (but surprisingly, not income from the parents), parity and sexual composition variables, and reported additional children desired in 1972. The estimates do not vary much between the age groups (though they are a little weaker for the second and third year), so I summarize here the post-neonatal (first three years combined) results. There are some differences between experiences for males versus females. Mortality for boys (but not girls) is more for parity 4-6 and less if village post-neonatal survival rates are higher, if there is "much" support from other family members, and if the child is first parity. Mortality for girls (but not boys) is higher if both parents have no education and is less if there is "some" support from other family members. The additional children desired variables have plausible patterns, but their interpretation is difficult, particularly given their ex post nature. The results thus suggest some differential effects for boys versus girls, and for the former by parity. Taken at face value, community characteristics have limited effects (only on males), as do education (only on females if both parents have no education). But the limited representation of observed community characteristics, the lack of control for parental income and for unobserved community and household characteristics, and the inclusion of the probably endogenous ex post desired children variables makes it necessary to qualify any interpretation.

Merrick (1985) presents reduced-form estimates of Brazilian infant mortality that suggest that, while piped water supply has a negative impact on infant mortality, the effect of parental education on mortality is much greater. The effects of community endowments other than piped water and of most household endowments other than parental education are not considered.

Cohen (1988) estimates the reduced-form demand relation for recent child illness for 600 urban Sudanese children under five (see Section 1.2). He finds a significantly negative impact of the household head's wage and of some ethnic groups, but no significant effects of other variables including piped water, mother's schooling, and type of housing (representing wealth). Such results contrast with the health production functions for anthropometric outcomes that he presents for the same sample in which piped water, maternal schooling, and housing seem significant direct or indirect (through vaccinations) determinants.

Rosenzweig and Schultz (1982a) study the joint determinants of demands for fertility and child mortality with a four per cent sample of the 1973 population census for Colombia. They include a relatively extensive representation of community-level infrastructural variables, most of which can be interpreted as health-care prices broadly defined, to explain the household demand for health outcomes: the per capita number of hospital beds and clinics, family planning expenditures per capita, transportation time to the capital city, average daily temperature, food prices, and the average schooling of women aged 15 and above in the region of residence. The community-level variables, with the exception of the food prices and the regional schooling variable, are interacted with the woman's schooling. Separate equations are estimated for each five-year age-group of women residing in rural and urban areas. Rosenzweig and Schultz find that, in urban areas, child mortality in families with less-educated mothers is strongly affected

by public health and family planning programs. They thus conclude that "... urban public health institutions are substitutes for the health care knowledge and the management capacity that an educated mother brings to her family" (pp. 58-59). They also find that, in the urban areas clinics are a more cost-effective means of lowering child mortality than are hospitals. For rural areas, in contrast, they find little effect of health and family planning programs on child mortality. They attribute the lack of these effects to the greater dispersion of health and family planning programs in rural areas. In both urban and rural areas, finally, Rosenzweig and Schultz observe a strong negative effect of maternal education on child mortality. Though their study has a relatively extensive representation of community variables, it does not control for many household variables, such as these related to women's childhood family background.

Wolfe and Behrman (1987a) present reduced-form estimates for infant and child mortality in Nicaragua as part of a larger latent variable system (see Section 1.2). They find a significantly negative impact of income and of mother's education. The latter result is similar to that reported by Rosenzweig and Schultz, and in earlier studies by others (e.g., Heller and Drake 1979). But in contrast to Rosenzweig and Schultz, Wolfe and Behrman find no significance for six indicators of community endowments (including literacy rates, hospital beds per capita, and sanitation connections, among others). With the same sample, Wolfe and Behrman (1987b) estimate the demand determinants of anthropometric indicators of child health. Standard estimates for individuals are compared with fixed effects estimates in which their special adult sibling data are used to control for unobserved abilities and motivations of the mother that emanated from her childhood family background ("maternal endowments".) Women's schooling appears important in the standard estimates, but has insignificant (at the 5% level) coefficient estimates in the fixed effect estimates, thus suggesting that in the standard individual estimates for child health, women's schooling primarily is representing the impact of unobserved maternal endowments. Income is not significant.

Behrman and Deolalikar (1988a) use panel data on rural south Indian households and anthropometric indicators to estimate joint reduced-form health and nutrient demand relations which allow for differing price and income responses for different household members (e.g., men, women, boys, girls) and which control for individual, household and community fixed effects. They use a latent variable representation of unobserved health status, for which they have three observed (but imperfect) indicators: (age-gender) standardized arm circumference, triceps fatfold, and weight-for-height. They find two of the four food prices (i.e., of rice and milk) to have significant positive effects on health status. However, they find neither a significant income effect nor significant differences in price or income responses across household members. The positive (income constant) price effects on health status may be surprising *prima facie*. However they are consistent with strong cross-price substitution effects in the underlying food demand equations toward foods with high nutrient-to-food conversion factors (see Section 1.4). If the price of, say, milk increases, a sufficiently large increase in the demand for other foods (and thereby in nutrient intakes and health status) may be induced so that the direct deterioration in health status resulting from reduced milk consumption is more than offset by the induced increases in nutrient consumption of other high-nutrient foods.

Horton (1988) also analyzes the demand for individual health outcomes with data on approximately 2,000 predominantly rural children aged 15 or less from the Philippines. To correct for heterogeneous tastes (particularly with respect to child quality and quantity) across households, Horton explores the differences in weight-for-height and height-for-age among children within each family in terms of age, gender, and birth order. She also allows some household-specific variables to enter her health demand function indirectly by specifying that the coefficient on birth order depends on maternal education and total household expenditure per capita. Her results suggest that birth order has a significant adverse effect on both height-for-age and weight-for-height, but that maternal education significantly weakens these adverse birth-order effects.

Rosenzweig and Schultz (1982b) analyze the determinants of male-female differentials in child survival rates in rural India using both household and district level data. They argue that the male-female survival differential depends upon the expected relative returns to male and female labor through affecting the returns to parental investments in sons versus daughters. They use predicted employment rates of men and women as proxies for the economic returns to male and female labor, arguing (not entirely persuasively) that wage rates may not accurately reflect the value of time as well as employment rates because cultural factors such as religion and caste may prevent women from equalizing market and household marginal products. In both the household and the district-level samples, they find predicted female (but not male) employment rates to be a significant negative determinant of the male-female child survival differential. They interpret these results to imply that children who are likely to be more economically productive adults receive a greater share of family resources and therefore have a greater propensity to survive.

Probably the best known of the cross-country studies that might be given a health demand interpretation are those by Preston (1980, 1986a).<sup>23</sup> Preston (1980) estimates the determinants of life expectancy using cross-country data for 1940 and 1970. Per capita income and adult literacy are highly significantly determinants of life expectancy for both periods, with the coefficient estimates in the life expectancy equations very similar for the two periods. However, average daily calorie availability per capita is not significant for either period.<sup>24</sup> To assess the contribution of increases in per capita gross domestic product, literacy, and calories availability to the increase in life expectancy between 1940 and 1970, Preston calculates what life expectancy would have been for individual less-

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<sup>23</sup> There are a number of other cross-country studies. For example, Hill (1985) focuses on a smaller group of developing countries for which the mortality data are relatively reliable and finds more or less constant effects of per capita income and education over time, but less evidence of a slow down in mortality declines between the 1950s-60s and the 1960s-70s (and actually an acceleration in Asia) than suggested by some observers.

<sup>24</sup> This may not be inconvenient in one sense since, if a reduced-form demand interpretation is to be given to Preston's estimates, it would seem that calories should be excluded since they are determined in part endogenously through household food demands.

developed countries if no structural changes had occurred in the relationship. The difference between actual life expectancy in 1970 and that predicted if the 1940 relation had prevailed in 1970 indicates the amount of change in life expectancy attributable to structural shifts in the life expectancy equation. He finds that approximately half of the total gain in life expectancy during the 30 years was unrelated to changes in per capita income, literacy, and calorie availability.

One possible problem with Preston's study is that prices and endowments are not included in the relation; if they are associated with income or literacy (as a priori seems plausible), the coefficient estimates for the included variables are biased because they represent in part the excluded ones. Quite possibly, for example, the coefficient estimates for both income and literacy are biased upwards because they capture in part the effect of excluded endowments that also increase in the development process. However, when Preston estimates the life expectancy equation in first differences for a smaller sample of countries for which data are available for both 1940 and 1970, his results are largely unchanged. Since first-differencing purges his estimates of additive unobserved country-specific fixed effects,<sup>25</sup> it means that the omitted variables problem was not severe for such fixed effects (though no insight is provided regarding unobserved variables that changed over these three decades).

Since the measures of per capita GDP used in Preston (1980) are based on international exchange rates, which are subject to many distortions, Preston (1980a) reestimates the relationship between life expectancy and income using the International Comparisons Project (ICP) measures of per capita gross domestic product, which are based on purchasing power parities of various currencies. He estimates the life expectancy equation (in levels) for 1965-69 and 1975-79 with both measures of income (and with literacy rate and excess calories availability as control variables) and finds that the coefficient of income in the ICP-based regressions is 50 per cent larger than that in the other regression. Behrman and Deolalikar (1988e) also, like Preston, find that the use of ICP income results in greater consistency with the data and higher health elasticities with respect to income than does the use of standard income data. They find that even greater consistency with cross-country experience and higher elasticities are obtained, however, with ICP income predicted by physical and human capital stock, which can be interpreted as a more permanent income measure. They also report that the effects are largely on infants and small children, so that life expectancy at age five is relatively unaffected by income.

The United Nations (1985) is a monograph written by Barbara Mensch, Harold Lentzner and Samuel Preston that examines socio-economic differentials in child mortality in 15 developing countries based on time series data. The estimates suggest relatively strong effects of "sociocultural" variables such as mother's education, ethnic group, and father's education (the last only in urban areas) and much weaker effects of "socioeconomic" variables such as per capita income, occupational status, sanitary facilities, and urban-rural residence. The authors interpret the "very considerable impact of

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<sup>25</sup> These might include some cross-country differences in the measurement and definition of life expectancy and at least the additive component of income distribution measures, which is emphasized by Dyson, Bell and Cassen 1978 in their simulations.

mother's education and ethnicity... [to point] above all to the potential importance of child care practices in determining levels of child mortality" (p. 289) and suggest that such results "support some of the assumptions of the primary health care movement, which emphasizes broad outreach into every home with a range of simple preventive measures" (p. 290). The authors do not mention, however, that more women's schooling may induce more formal labor force participation (as in Sirilaksana's Thai study discussed above) and thereby reduce the mother's time (though perhaps increase the quality) in child care. The authors explicitly observe that their estimates do not yield the rates of returns for various alternatives on which one would like to make policy decisions because they do not control for costs nor gestation periods (see Subsection 1.1C above). They do not recognize as explicitly the possibility that some of their estimates, particularly for mother's schooling, may be upward biased because of the failure to control for unobserved endowments, as discussed above. They also ignore price changes, which the estimates below in Section 1.4 suggest may be more important than income changes and the exclusion of which may bias downward the income coefficient estimates (since food prices tend to increase relatively with development). These last considerations mean that their results must be interpreted with care, but nevertheless they are provocative in suggesting major roles for women's education and perhaps for child care.

Caldwell (1986) discusses the cross-country associations between child mortality and development. He observes that rankings of developing countries by per capita income differ substantially from rankings by infant mortality, which he interprets to mean that economic development is not all that dominant in determining child health and mortality. He considers in detail the experience of three "superachievers" regarding health, Sri Lanka, Kenya, and Costa Rica. He concludes that "low mortality...will not come as an unplanned spin-off from economic growth" (p. 210). Instead there must be "a broad social consensus as to the value of educational and health goals...." (p. 210). This leaves the question of how such a consensus is developed.

Horton, Kerr and Diakosavvas (1988) estimate a demand relation for infant mortality from pooled cross-country and time-series data for 1966-1981 for 34 developing countries, with the innovation of including the "price of cheap calories," i.e., the average of ICP-adjusted retail prices of rice, maize, wheat, millet, and sorghum from FAO series. Their cross-country estimates suggest significant price elasticities for mortality between 0.2 to 0.6. Separate time series demand equation estimates for each country yield that nine of the 34 countries have significantly negative income elasticities of infant mortality, while six have significantly positive price elasticities. But seven countries have the "wrong" signs (significantly positive) for income elasticities and eight have the "wrong" signs (significantly negative) for price elasticities (but see the discussion on price effects on nutrient intakes in Section 1.4). The country-by-country results thus are ambiguous and permit no broad generalizations.

Cochrane, O'Hara and Leslie (1980), in a widely-known paper, present a thoughtful review of micro and macro studies of child-health determinants as of the end of the 1970's. Their primary conclusion is that the most robust determinant is the positive impact of women's schooling. For aggregate estimates they also regress the absolute values of the estimated education coefficients from a number of aggregate estimates on life expectancy, income, illiteracy, governmental health expenditures, time and the

mortality of the uneducated (given by the intercept in the original regressions). They find  $R^2$ s of from .83 to .99 (the latter if the last variable is included), and interpret their results (with qualifications due to the small sample size) to reflect the extent to which the different estimated marginal impacts of education on mortality are sensitive to the right-side variables such as governmental health expenditures. It would seem to me that what should be emphasized from these estimates is that almost all of the variation in the estimated education impact across studies may be due to omitted variable bias. This interpretation still leaves the possibility that education is important, but apparently substantially less important than the aggregate estimates themselves suggest.

The estimated reduced-form child health and mortality demand relations, thus, like the estimated child health and mortality production functions, have some, but limited, implications for understanding of health and mortality and development. They suggest some positive associations of per capita income and health, but often do not control for the possible simultaneous determination of income,<sup>26</sup> nor for the possibility that income may be proxying for unobserved health infrastructure and individual and community endowments. They also often suggest that schooling -- particularly for women -- is important, though the studies of the one sample that permits adult sibling control for unobserved fixed effects associated with one's childhood family again (as for the health production functions) suggests that schooling largely is representing unobserved maternal endowments and not the effect of schooling per se. Observed community endowments often appear important, perhaps more so than a literal reading of the estimates suggests because per capita income might be proxying for unobserved endowments in a number of studies (particularly the more aggregate ones).

#### **Section 1.4. Reduced-Form Demand Relations for Health-Related Goods and Services including Nutrients**

Some of the goods and services that enter into the health production function are widely presumed to be fairly directly related to individual choices regarding health (given prices), such as the use of formal medical services by different types of practitioners, the use of medicine, and the type of household water supply and sewage disposal, and the consumption of nutrients.

1.4A. Nonnutrient health-related goods and services: In a relatively early study, Selowsky (1979) estimated demand relations for doctor visits in Colombia in 1974. He reports no significant results for rural areas, but significant estimates for urban areas with an income elasticity of 0.05 and elasticities with respect to education of .18 for the household head and .08 for the spouse. That the elasticity with respect to household's head's education (generally a male) is larger than that with respect to the spouse's

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<sup>26</sup> It might seem that the simultaneous determination of income would not be a problem for child health and mortality because most income is earned by adults. But children do contribute substantially to household income in many contexts. Moreover, even if the children do not contribute to household income, if their nutrient intakes are correlated with those of the adults in the household who do, the simultaneity possibility remains.

education (generally a female) is in contrast to the relative emphasis placed on women's education in determining health visits by many. The income elasticity is not very large even in urban areas and may be biased upward due to the failure to treat family size as endogenous. In a concurrent study of Malaysia, Meerman (1979) reports no significant impact of income in determining days hospitalized, outpatient visits, rural clinic visits, or rural mid-wife assisted births. He claims that this reflects the low or nonexistent fees charged for these services, though apparently neither he nor Selowsky controlled explicitly for prices in their demand regressions.

Behrman and Wolfe (1987) include demand relations for medical-care usage and household water and sanitation quality in a latent variable system of estimates for Nicaragua in 1977-8.<sup>27</sup> They find that income, mother's schooling, and community endowments all have significantly positive effects on both medical-care usage and household water and sanitation quality, but that the positive impact of women's schooling evaporates once again (as in the health relations discussed in Sections 1.2 and 1.3) if there is a latent variable control for unobserved maternal endowments.

Rosenzweig and Wolpin (1988) use the residual child-specific and family household health endowments from their health production function estimates for infants in Candelaria, Colombia in 1968 to 1974 (see Section 1.2 above) as right-side variables (together with family income, maternal schooling and enrollment in family planning programs) in relations that determine probabilities of short birth intervals (less than three years), breastfeeding, and inoculations. They find that there are significantly shorter birth intervals subsequent to having a child with a good health endowment, that such children are more likely (at a 25% significance level) to be breastfed, and that males are more likely to be inoculated. Neither family income nor maternal schooling has a significant impact on these outcomes at normal significance levels, though maternal schooling has a negative effect at the 25% level. They do not provide information with which to know whether family income or maternal schooling would appear to be significant were it not for the control for health endowments, though (as noted in Section 1.2) they do note that these variables are correlated with such endowments.

Cohen (1988), in his study of urban Sudanese children, estimates reduced-form relations for number of vaccinations for children under five, which in turn are strongly associated with anthropometric measures for these children (see Section 1.2 above). The most important determinant aside from the child's age seems to be piped water, the presence of which increases the number of vaccinations by 1.2; it is difficult to know whether this represents the availability of services (including vaccinations), household knowledge, or wealth. The second most important determinant is mother's schooling, each year of which increases the number of vaccinations by 0.15 (so it would take seven years of additional schooling to have the same impact as piped water). There also are significant effects of housing type (representing wealth?) and ethnic groups (representing

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<sup>27</sup> Both of these variables are treated as unobserved latent variables with imperfect indicators, which include formal medical attention during pregnancy, age-standardized number of injections for children and participation in the social security system for household medical-care usage and the nature of toilets, baths, water and sewage disposal for the household water and sanitation quality.

different child-care practices?).

Akin, Griffin, Guilkey and Popkin (1985) estimate multiple-choice logit models of the determinants of a household's decision to use medical services and its choice of a medical practitioner (i.e., whether public, private, or traditional) for 1,903 households from 100 barangays in the Philippines. The right-side variables include an extensive set of price variables for each of the four types of medical facilities: the cash price of using the facility, the transport time and cost to reach the facility, and the drug costs. Additional variables are included such as whether the sickness is covered by insurance; the value of household assets; gender, education, and urban location of the patient; and the severity of the illness. They obtain an almost total lack of statistical significance of any of the economic (price, time) variables. Since the pecuniary and nonpecuniary costs of consuming medical care are not trivial in the sample, results which indicate that these costs are not significant determinants of practitioner choice may be surprising.

Unfortunately, Akin et al. include the demand for health outcomes (e.g., illness severity) as an explanatory variable in their demand for health-care utilization, without treating it as endogenous. Indeed, they find that it is the only significant determinant of practitioner choice. While this result is logical (since there is likely to be segmentation in the facilities available for treating particular types of illnesses, with severe illness being treated in large public facilities and minor complaints by traditional medicine providers), that the severity of illnesses is endogenous introduces a bias into the estimated price effects. It could be the case, for example, that health-care prices do matter in determining the demand for health-care utilization and practitioner choice by influencing the degree to which individuals ignore their initial symptoms and allow their illnesses to become severe.

Birdsall and Chuhan (1986) also estimate a multiple logit system for the demand for type of curative health services in Mali. Unlike Akin et al., they find significant effects of a number of dimensions of prices -- i.e., distance and quality measures.

Gertler and van der Gaag (1988) estimate discrete health-care choice determinants for 1254 Peruvian adults and 969 children in 1984 who had had recent illness symptoms or accidents and for 1030 adults and 769 children in rural Ivory Coast who had experienced an accident or illness in the four weeks prior to their 1985 survey. They observe that if health is a normal good, as income rises, at a given health level the marginal rate of substitution for health must decline, so that richer individuals have less price elastic demands than do poorer ones -- but that most (if not all) previous studies precluded such a possibility a priori.<sup>28</sup> Their estimates indicate significantly negative effects of travel time and significantly positive associations of consumption with all three user choices, but with education causing a significant shift from clinics to hospitals and private doctors in Peru (though not in the Ivory Coast). The own-price elasticities are negative for all treatment choices, and are much larger for the bottom income quartile (up to -2.8) than for the top income quartile (up to -0.7). The estimated price responses, furthermore, tend to be larger for children (with own-price elasticities up to -2.82) than for adults (a maximum of -1.83). Therefore health-care for children, particularly those in

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<sup>28</sup> Gertler, Locay and Sanderson (1987) make a similar observation, but do not present estimates for children.

poorer households, is quite responsive to changes in prices (including time costs) of health care. Alderman and Gertler (1988) use a similar approach to study the substitution between private and public health care providers for the treatment of child illness in urban Pakistan. They also find larger price elasticities for lower income groups, though they do not compare estimates for children versus adults.

While the Behrman and Wolfe (1984) study does consider the impact on health outcomes of the same reduced-form variables (see Section 1.1), the other studies reviewed in this subsection give no estimates of the health impact of the health-related goods and services under investigation. And it may be naive to think that the health effects necessarily are positive. Wolfe and Behrman (1984) report, for example, that women's schooling increases health-care usage significantly in Nicaragua, but does not affect significantly a latent variable representation of health status. While their specification can be criticized as difficult to interpret fully because of its hybrid nature, this result regarding women's schooling is suggestive of the possibility that increased use of health-related inputs does not necessarily lead to improved health. Thus while the studies reviewed in this subsection suggest (with some ambiguity) that demands for health related goods and services well might increase with higher incomes, lower prices, more available services, and more women's schooling, the link to health per se is more speculative.

**1.4B. Nutrient Reduced - Form Demand Relations:** There have been a number of recent estimates of nutrient demand functions. These studies are summarized most clearly by considering in turn each of the major sets of right-side variables.

**Income/Expenditure:** There is considerable recent controversy about the magnitude of the nutrient responses to income. One widely-held view is that widespread malnutrition will disappear only with the improvements in income that accompany the development process. The World Bank (1981: 59) articulates this view forcefully: "There is now a wide measure of agreement on several broad propositions.... Malnutrition is largely a reflection of poverty: people do not have income for food. Given the slow income growth that is likely for the poorest people in the foreseeable future, large numbers will remain malnourished for decades to come.... The most efficient long-term policies are those that raise the income of the poor."<sup>29</sup> On the other hand, estimates of the income/expenditure elasticities (hereafter referred to as income elasticities) for calories vary widely from a low of below 0.1 for Nicaragua (Wolfe and Behrman 1983, Behrman and Wolfe 1988), Sri Lanka (Scandizzo and Knudsen 1980, Sahn and Alderman 1988), Chile (Harbert and Scandizzo 1982), the rural Philippines (Bouis and Haddad 1988), upper-income households in Morocco (Mateus 1985), rural south India (Behrman and Deolalikar 1987c, 1988f, Bhargava 1988) and rural Indonesia (Pitt and Rosenzweig 1985) to highs of 0.8 for low-income households in Sri Lanka (Sahn 1988), 0.9 for rural Sierra Leone (Strauss 1984a) and 1.2 for lower-income households in Morocco (Mateus). Reutlinger (1984) presents cross-country estimates based on 1970-1980 data and finds an average caloric elasticity with respect to income of 0.2, but with significantly lower values

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<sup>29</sup> The World Bank, however, hardly has been monolithic in this view. In a well-known World Bank study, for example Reutlinger and Selowsky (1976: 7) concluded: "Malnutrition is unlikely to disappear in the normal course of development."

if there was initial caloric adequacy. Gaiha and Young (1988) present estimates of caloric elasticities with respect to income based on time series for 1970-84 for 16 developing countries and find a range of estimates from -0.3 to 1.4, with a median of 0.2. Estimates of the income elasticity of proteins, which are far fewer in number than are calorie income elasticity estimates, also range from 0.0-0.1 for small children and infants in rural India (Levinson 1974), high income households in Morocco (Mateus), and rural households in Indonesia (Pitt and Rosenzweig) to 0.6-0.8 for rural Bangladesh (Pitt 1983) and rural Karnataka in India (Alderman 1986b) and 1.2 for low-income households in Morocco (Mateus).

One possible explanation for this range of estimates that may be consistent with nutrient responses to income being considerable for the poor is that nutrient elasticities with respect to income are inversely associated with income. A number of studies that have examined such a possibility have found empirical support for it (e.g., Timmer and Alderman 1979, Pinstrip-Andersen and Caicedo 1978, Murty and Radhakrishna 1981, Williamson-Gray 1982, Mateus 1985, Behrman and Wolfe 1984a, Behrman and Deolalikar 1988d, Sahn 1988, Ravallion 1988). But in some studies no inverse association of the nutrient elasticity with respect to income is found (e.g., Behrman and Deolalikar 1987a, Strauss 1984) and in many of the cases in which it is found, it is not sufficient to explain the wide range of estimates for calorie income elasticities cited in the previous paragraph.<sup>30</sup> And in at least one case (Reutlinger 1984), the calorie elasticity increases significantly with income, though inversely with initial caloric adequacy.

Another explanation for the wide range of these income elasticity estimates is related to the level of aggregation at which nutrient conversion factors are applied in obtaining the estimates. In some cases, most notably the more sophisticated food expenditure system estimates to which nutrient conversion factors are applied *ex post*<sup>31</sup> (e.g., Murty and Radhakrishna 1981, Pitt 1983, Strauss 1984, Alderman 1986b), foods are aggregated to relatively few categories (usually, less than ten) when nutrient-to-food conversion factors are applied. In other cases conversion factors are applied at much more disaggregate levels. Behrman and Deolalikar (1987c) demonstrate that, if there is substantial substitution among disaggregated foods and if prices paid per nutrient as a result increase substantially as income increases, the former procedure may overstate

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<sup>30</sup> Though in at least one case, Mateus' (1985) estimates for Morocco, the inverse association between nutrient elasticities with respect to income and income is sufficient to explain the wide range of estimates.

<sup>31</sup> In these studies food expenditure systems are estimated with imposition of cross-equation restrictions and with relatively broad food aggregates, and then nutrient conversion factors are applied to the results, which practically guarantees nutrient elasticities on the order of magnitude of overall food expenditure elasticities, which for poor populations typically are 0.6 to 0.8 or higher. Pitt suggests that the aggregate food expenditure system route is superior to direct estimates of nutrient reduced forms, apparently because of greater efficiency due to the cross-equation restrictions, but he does not discuss the aggregation issue.

considerably the true nutrient elasticity with respect to income. After citing evidence from several sources that nutrient prices paid increase substantially with income,<sup>32</sup> they compare nutrient elasticities for a rural south Indian sample obtained from application of nutrient conversion factors to food elasticity estimates for six food groups versus direct nutrient reduced-form estimates with the conversion factors applied for 120 more disaggregated foods (in both cases using simultaneous estimates for income). The former (more aggregate) procedure implies calorie elasticities with respect to income of 0.8 to 1.0 (depending on whether fixed effects are controlled); the latter (disaggregated) procedure yields calorie elasticities of 0.2 to 0.4 (and not significantly different from zero). A number of other recent studies report similar results with food expenditure elasticities about twice the magnitude of calorie elasticities: Greer and Thorbecke (1984) for Kenya, Garcia and Pinstrop-Andersen (1987) for the Philippines, Kumar (1987) for Kerala in India, Alderman (1986b) for Karnataka in India, and Reutlinger and Selowsky (1976) and Behrman and Deolalikar (1988d) for cross-country samples. Thus, even for very poor populations, calorie elasticities with respect to income appear to be positive, but much smaller than food expenditure elasticities and perhaps substantially smaller than suggested by the above quotation from the World Bank (1981) and others who suggest that income is critical in determining nutrient intakes.

A third explanation for the wide range of estimated nutrient elasticities with respect to income, proposed by Bouis and Haddad (1988), is a combination of not distinguishing between food purchases by the household and nutrients consumed by household members on one hand (which may differ due to food provided for guests and laborers and due to waste, all of which are likely to be correlated with income) and measurement errors on the other. Bouis and Haddad demonstrate that these possibilities might result in important biases in estimated nutrient elasticities. Then they present estimates for a rural Philippine sample that vary for these two reasons, as well as because of possible biases due to simultaneity and to the failure to control for fixed effects. The estimates vary by a factor of about ten, with their preferred estimate of the nutrient elasticity with respect to income equal to 0.06 (while others are over 0.50). They do not test the importance of their explanation versus the aggregation one offered by Behrman and Deolalikar (1987).

A fourth explanation might be that nutrient intakes respond little to current income fluctuations since such basic consumption is protected against transitory income fluctuations (by adjusting other consumption and savings), but nevertheless respond strongly to longer-run or permanent income changes. The range of available estimates,

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<sup>32</sup> Using 1970-71 data from the Indian National Sample Survey, Radhakrishna (1984) calculates that the average cost of calories from each of six broadly-defined food groups increases consistently with total expenditure for both rural and urban households. Pitt (1983) reports that the 25th percentile household spent 22 per cent more per gram of protein, 15 per cent more per calorie, and 44 per cent more per milligram of iron than did the 90th percentile household in his Bangladeshi sample. Williamson-Gray (1982) estimates the income elasticity of the average price paid per calorie as to be 0.3 even for relatively malnourished households in her Brazilian sample. Behrman and Deolalikar (1988d) estimate the income elasticity of the average price per calories to be 0.4 or 0.5 for the lower income levels in their cross-country sample.

thus, may reflect how well the income or expenditure measure used approximates permanent income. Behrman and Deolalikar (1988f) test this possibility by contrasting the estimates for nutrient responses to current versus permanent income (with the latter based on almost a decade of data). Bhargava (1988) tests it by allowing for dynamic adjustments in nutrient intakes in response to income changes. Neither study finds evidence that the response to longer-run income is much greater than that to current income for a rural south Indian sample.

Thus, apparently nutrient elasticities with respect to income often are fairly low in developing countries and the much higher estimates sometimes presented quite possibly are biased upwards substantially due to a combination of aggregation problems and prices per nutrient that are positively associated with income, differences between household purchases and consumption of household members that are positively associated with income, and measurement errors. In any case, there does not seem to be compelling evidence for the World Bank type position that basic nutrients -- particularly calories -- increase rapidly with income.<sup>33</sup>

Why might calorie income elasticities be substantially lower than food income elasticities? Reutlinger and Selowsky (1976), Shah (1983), Behrman and Wolfe (1984a), Bouis and Haddad (1988) and others have conjectured that the reason may be that at the margin people are concerned much more with food attributes other than calories and these food attributes may not be associated highly with caloric content. Examples include noncalorie nutrients, food texture, status value, appearance, taste, aroma, preparation, composition, and providing food for guests and laborers. Many of these characteristics are not measured in most socioeconomic data sets and therefore their relevance is difficult to assess systematically.

However several studies do present evidence on some of them. For example, Reutlinger and Selowsky (1976) present age-sex specific elasticities for calories, proteins, vitamin A, iron and calcium for a poor Calcutta sample. While those for calcium and vitamin A generally are higher than those for calories, they are not as high as food expenditure elasticities. Behrman and Deolalikar (1987c, 1988d) also present estimates for both micro data for rural south India and a cross-country sample for the income elasticities of nutrients other than calories (e.g., proteins, calcium, iron, carotene, thiamine, riboflavin, niacin, ascorbic acid, vitamin C, fat). They find that the income elasticities are higher for some of these other nutrients than for calories at low-incomes (e.g., for fat, calcium, riboflavin and vitamin C in the cross-country estimates), suggesting that part of the explanation for the low calorie elasticities is compositional shifts in food to obtain other nutrients at the margin that are not highly correlated with calories. But for none of these other nutrients is the income elasticity as high as that for food, so the preference at

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<sup>33</sup> Ravallion (1988) argues that even if the calorie elasticities with respect to income are low, if there are large numbers of individuals with calorie intakes close to requirements, the elasticity of some measure of undernourishment (e.g., a head count measure) with respect to income may be high. He demonstrates such a possibility for East Java in Indonesia. His results do not mean, however, that household calorie intakes are very responsive to income.

the margin for noncalorie nutrients is only a limited part of the explanation for the low-income elasticities for calories. Pitt and Rosenzweig (1985) report higher elasticities with respect to farm profits in their Indonesian study for most other nutrients than for calories, but all of their nutrient elasticities with respect to farm profits are so small (less than 0.03) that shifts towards noncalorie nutrients is a very small part of the explanation for the low calorie elasticities. Bouis and Haddad (1988), as noted above, present evidence that the discrepancy between household food purchases and food consumed by household members increases with income in the rural Philippines.

Behrman and Deolalikar (1988c) conjecture that part of the explanation for the relatively low calorie as compared with food expenditure elasticity lies in an increasing taste for variety as income increases, and thus a movement away from concentration on low-cost nutrient foods at low-incomes to greater food diversity as income increases. They argue that the taste for variety can be associated with two characteristics of preference curves defined over various foods: (1) the sharpness of the curvature since, if the curvature is greater *ceteris paribus* a more diverse food basket is consumed for a wide variety of food prices (rather than concentrating on the relatively cheap foods) and (2) the centrality of the preference curves under the argument that at low incomes preferences are likely to be located close to the axis for the traditionally cheapest nutrient source, but as income increases they may become more centrally located (i.e., closer to symmetric around 45° rays from the origin).<sup>34</sup> They test to see what happens to both of these preference characteristics as income increases in a cross-country sample for 1975 and 1980 for 66 countries using the International Price Comparison project carefully constructed prices and incomes. They find that starting from low incomes both preference dimensions of the taste for variety increase as income increases. Therefore they conclude that part of the explanation for the low income elasticities for calories and other nutrients is an increasing taste for food variety even at low income levels.

Prices: Demand analysis focuses on market prices in addition to income. A number of recent studies (as well as earlier ones) have estimated nutrient responses to market prices (in contrast to the relatively few studies that have included such prices explicitly in the health reduced-form demand relations discussed above). The focus is on food prices, though occasionally other prices, including those for time are included.<sup>35</sup>

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<sup>34</sup> Behrman, Deolalikar, and Wolfe (1988) note a third respect in which variety can be associated with preference curves. Assume that there is a minimum nutrient constraint at low food intake levels, on which preference curves effectively collapse at very low income so that individuals specialize on consumption of the food which provides calories cheapest. Then, as their income increases over a range, they will maximize their preferences by consuming the same number of calories by changing the composition of their food intakes (i.e., increasing variety) so that the nutrient elasticity with respect to income is zero as long as the nutrient constraint is effective. Once income increases enough so that the nutrient constraint no longer is effective, the two dimensions of taste for variety discussed in the text become relevant.

<sup>35</sup> Behrman and Deolalikar (1988f), for example, include wages in nutrient demand relations for rural south India to reflect the opportunity cost of time in household production and find significant effects. Senauer, Sahn, and Alderman (1986) report

Frequently, the estimated nutrient price elasticities are substantial. Perhaps of more surprise, often the nutrient elasticities for prices of a number of major foods are positive, indicating that increases in these food prices improve nutrient intakes.

Pitt (1983) finds that, even after controlling for income, calorie demand in Bangladesh has positive elasticities with respect to the prices of five of the nine foods he considers -- pulses, fish, mustard oil, onions, and spices. Alderman (1986b), also controlling for income, reports positive elasticities for calories and proteins with respect to prices for milk, meat and grain and negative ones for rice, ragi and other food in his fixed effect estimates for rural Karnataka in India. Behrman and Deolalikar (1988f) also find predominately positive food price effects, except for the price of sorghum, the basic staple, on the nutrient consumption of all household members, even with controls for income and individual, household, and community fixed effects. Sahn and Alderman (1988) report more significant positive than negative price elasticities (eight versus six) in calorie demand relations for rural Sri Lanka, though Sahn (1988) reports a positive elasticity only for the price of meat for low-income households in Sri Lanka. Pitt and Rosenzweig (1985) also find a large number of positive price effects on nutrient demand for a sample of Indonesian farm households even with farm profits held constant. The only food price which has a consistently negative (profits-constant) effect on all nutrients in this study is the price of milk.

Thus there is evidence of substantial positive income-compensated food price effects on nutrient consumption at least with respect to prices other than those for the basic staple of the poor. If two foods are substitutes and the price of the high-cost nutrient source increases there may be a sufficient shift to the low-cost nutrient source so that nutrient intakes increase.

Another interesting question regarding the price responses is whether they differ with income levels. Alderman (1986a) surveys 15 nutrient demand and studies and notes that the majority, including the studies by Pitt (1983) and Strauss (1984) reviewed above, find that own-price elasticities of food demand decline in absolute values with income or expenditure. Sometimes the trend is pronounced; Williamson-Gray (1982), for example, estimates compensated own-price elasticities of cereal demand of -0.74 for the poor, -0.16 for the middle, and not significantly different from zero for the rich in Brazil. A few studies, e.g., Williamson-Gray and Timmer and Alderman (1979), also observe systematic relationships between the absolute value of cross-price elasticities and the level of income or expenditure. Alderman's (1986b) finding that the cross-price elasticities differ substantially in his study on rural Karnataka in India with control for fixed effects, however, suggests that the cross-price effects should be interpreted with caution.

Such results refer to interhousehold differences in nutrient responses to prices, but there is much less evidence about intrahousehold differences in nutrient responses to prices, despite frequent conjectures and some evidence that intrahousehold nutrient distributions if anything, tend to favor males over females and older over younger children (e.g., Harriss 1987, Behrman 1988 a,b,c). One reason for the absence of such evidence is

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significant impact of the (selectivity-controlled) price of women's time on the composition of food consumption in Sri Lanka, though they do not explore the impact on nutrients consumed.

that most of the above studies are based on data on household, not individual, nutrient intakes. An exception is Behrman and Deolalikar's (1988f) study for rural south India which estimates separate nutrient demand relations for men, women, girls, and boys. These estimates suggest that nutrient price responses are significantly different for females than for males, with algebraically lower price elasticities for the former than for the latter. This may mean that females suffer because they have relatively large reductions in their nutrient intakes when food is scarce and prices are high (even though there are compensating movements in the opposite direction when food prices are low).

For infants, breastfeeding is a critical source of nutrients. Some evidence exists of important price responses for breastfeeding. Sirilaksana (1982, 1986), for example, reports that breastfeeding responds significantly negatively to the predicted women's wages in her rural Northeastern Thailand sample. That she controls for income and various wealth indicators and for contact with a modern medical program reinforces her interpretation of this variable representing the opportunity cost of women's time. But Anderson (1984) does not find a significant impact of women's schooling, which she interprets to represent time costs, in her study of 1262 households in the Malaysian Family Life Survey.

Other Determinants: The World Bank (1980, 1981), Colclough (1982), and a number of others claim that one benefit of greater education for women is improved nutrient intakes, given income and prices. Ward and Sanders (1980) and Pitt and Rosenzweig (1985) find no significant effect of women's schooling on household nutrient consumption in Brazil and Indonesia, respectively, and Horton (1985) reports a negative impact on calories obtained per unit expenditure in Gujarati, India (though she finds a positive impact of the household head's education). But Wolfe and Behrman (1983, 1987a,b) and Behrman and Wolfe (1984a, 1987, 1988) find that Nicaraguan households in which women have more schooling tend to be significantly and substantially better nourished, ceteris paribus, particularly in regard to Vitamin A, proteins, and calcium. The impact of women's schooling, moreover, is significantly greater than that of men's schooling for proteins (though not for other nutrients), providing some support for those who emphasize the special role of women in nutrient determination. Furthermore, this effect persists even if there is adult sibling deviation control for unobserved childhood-background related characteristics of the women, in sharp contrast to the estimated impact of women's schooling on health (Sections 1.2 and 1.3) or a number of other socioeconomic outcomes (Behrman and Wolfe 1984b). However, women's schooling is strongly negatively associated with the length of breastfeeding, perhaps reflecting in part the opportunity cost of women's time (Wolfe and Behrman 1987a).

Household size may affect nutrient demand per capita. Behrman and Wolfe (1984a) argue that the magnitude of the household size elasticity relative to the household income elasticity reflects the extent of returns to scale with respect to household size. They, as well as Ward and Sanders (1980) and Wolfe and Behrman (1983), obtain statistically significant negative effects of household size on nutrient demand which imply considerable increasing returns to scale. Iyenger, Jain, and Srinivasan (1968) also estimate economies of scale in the consumption of "necessities" such as cereals and fuel-lighting but not in the consumption of "luxuries" such as milk and clothing. Ravallion (1988), on the other hand, reports significantly negative effects of the household

size on per capita nutrient consumption. However, all the above estimates may be biased since fertility and hence household size may be endogenous variables that are jointly determined along with nutritional choices. Therefore the estimated household size variable coefficient may represent in part unobserved profertility determinants (e.g., tastes), which may be associated with less investment in nutrition; if so, the household size coefficient estimate understates the true returns to scale. Pitt and Rosenzweig (1985) do not include household size as a determinant of per capita nutrient intakes, but include household composition (treated as an endogenous variable) and find that per capita consumption of calories, proteins, carbohydrates, and phosphorous all increase significantly with mean household age.

Community endowments also may affect nutrient demands, though relatively few studies include proxies for such endowments in their estimates. Sirilaksana (1982, 1986) reports a significantly negative impact of one community endowment, whether the village has had contact with a health program, in her estimates for breastfeeding in Northeast Thailand. She interprets this to reflect better information about normal weaning times and the need for other food supplements among women who have had contact with such health programs. Anderson (1984) finds a significantly negative impact of modern sanitation (but not of distances to hospitals nor to family planning clinics) in her estimates for breastfeeding duration in Malaysia. Wolfe and Behrman (1987a) also find a negative effect of community endowments on the length of breastfeeding, which may reflect similar considerations. Pitt and Rosenzweig (1985) report significant coefficient estimates for 10 out of 45 coefficients of health programs and water source in their study of Indonesian nutrient demands, but they do not discern a clear pattern in these estimates. Behrman and Wolfe (1987) find a significant positive impact of community endowments related to population density, literacy and hospital beds per capita which, when included in the nutrient determinant relation, reduce significantly the estimated impact of women's schooling. Their community endowment variables might seem to represent the greater range of food options and greater knowledge of nutrition in more urban and more literate areas. However if a latent variable indicator of the woman's unobserved endowment is added, the sign of the community endowment variable becomes significantly negative; such a sign has a possible interpretation since the above-mentioned indicators of community endowments probably are inversely associated with food prices. In Behrman and Wolfe (1988), in contrast, the significant impact of community endowments (negative for population, positive for literacy rates) in standard and random affects estimates are, if anything, intensified in fixed effects estimates. But the lack of robustness with the latent variable representation of women's endowment in Behrman and Wolfe (1987) still leads to some doubts about the interpretation that community endowments are important.

Summary: The estimated nutrient reduced-form demand relations lead to a somewhat more positive appraisal of the state of our knowledge than do the relations discussed above for child health, mortality and other health-care determination. Income effects appear to be common and larger for poorer than for better-off people, though substantially smaller than seems to be suggested by the World Bank (1981) and many others. Apparently this reflects that even very poor people at the margin value a number of nonnutrient food attributes, including food variety per se. This means that on one hand the impact of development is substantially less -- maybe half as much or smaller -- than

often is assumed. On the other hand, that the poor seem to value nonnutrient attributes relatively highly at the margin suggests that they do not perceive their undernourishment to be as high priority a problem as many outside observers seem to claim it is, thus perhaps supporting the revisionist nutritionist interpretation of Sukhatme (1982), Payne (1988), Srinivasan (1981, 1988) and others.

Nutrient responses to food prices seem often to be substantial. But, perhaps surprising prima facie to some, often positive for food prices other than the basic staple even controlling for income, and even more likely to be positive for rural residents if their incomes are positively correlated with food prices. The poorer also tend to have larger (absolute values of) price responses than do better-off individuals. Thus effective price policies apparently can have substantial effects on nutrient intakes, but that these effects may be positive as well as negative means that the design of price policies may be tricky for demand reasons, in addition to the need to be concerned with supply responses. The impact of fixed effects on price elasticities in the two cases in which they have been explored for nutrient demand relations (i.e., Alderman 1986b, Behrman and Deolalikar 1988a), however, suggests some need for caution in interpreting the estimated price responses. Typically in the development process, food prices increase relative to nonfood prices due to policy shifts that move from discrimination against agriculture to favoring agriculture. The responses to such price movements may be much more important than the responses to income changes.

For the other determinants of nutrients that have been investigated, the evidence is less persuasive because of fewer studies, greater variance of estimates and more estimation and measurement problems. Women's schooling is important in some studies, and basically robust to control for unobserved maternal endowments in contrast to the health and mortality determinant studies, but is not significant in other cases so it is unclear that one should expect much of a direct nutrient impact of increased women's schooling in the development process. Economies of scale appear important, so ceteris paribus nutrient intakes may decline with the smaller household sizes usually experienced with development. The available estimates may be misleading due to the failure to control for the endogeneity of household size, but probably this causes a downward bias in the estimated returns to scale. Community endowments, finally, may have the impact of increasing nutrient intakes with development, but few studies have included them.

## **Section 2. The Impact of Economic Adjustment Programs on Child Health and Mortality<sup>36</sup>**

Most developing economies face periodic macroeconomic problems of imbalance between aggregate demand and supply, inflation, unemployment, and foreign exchange shortages. The affected countries often undertake macroeconomic adjustment programs to attempt to resolve the problems. These adjustment programs may be developed and

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<sup>36</sup> The section builds upon material in Behrman (1988b) and Behrman and Deolalikar (1988d). Section 2.1A draws on material in Addison and Demery (1985), which is presented in similar but reduced form in Demery and Addison (1986).

undertaken by the countries themselves or in collaboration with the IMF and (at least implicitly) with important international lenders. Such programs typically involve currency devaluations, governmental budget reductions, monetary restrictions, freeing of previously controlled prices, and wage restraints. This section focuses on the impact of the economic adjustment programs undertaken by the developing countries on health and nutrition in those countries.

Some observers, such as Jolly (1985), Jolly and Cornia (1984), UNICEF (1984), the Inter-American Development Bank (1985), the World Food Council (1985) and Cornia, Jolly and Stewart (1987), have concluded that recent economic recessions and associated macro adjustment programs in developing economies have had substantial deleterious effects on child health and nutrition in these societies. Jolly (1985) summarizes the results of these studies as follows:

As it mostly operates at the moment, adjustment policy...transmits and usually multiplies the impact on the poor and vulnerable. The result, as shown in many countries, is rising malnutrition in the short run -- and in the long run, reinforcement of a style of development which will primarily rely on accelerated growth and trickle down, if it works at all, to reduce malnutrition in the future.

Based on this kind of generalization, UNICEF (1984), Jolly and Cornia (1984), and Jolly (1985) call for what Jolly (1985) calls adjustment with a human face.<sup>37</sup> Despite the

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<sup>37</sup> In Jolly's words: *First, a clear acknowledgement in the goals of adjustment policy of concern for basic human welfare and a commitment to protect the minimum nutrition levels of children and other specially vulnerable groups of a country's population.*

*Second, the implementation of a broader approach to the adjustment process itself, comprising four components:*

- (a) actions to maintain a minimum floor for nutrition and other basic human needs, related to what the country can in the long term sustain;
- (b) restructuring within the productive sectors -- agriculture, services, industry -- to rely more upon the small-scale, informal sector producers and to ensure their greater access to credit, internal markets and other measures which will stimulate growth in their incomes;
- (c) restructuring within health, education and other social sectors, to restore momentum and ensure maximum coverage and benefits from constrained and usually reduced resources. Already, there are important examples of what can be done to reach all of a country's population, but still at relatively low cost;
- (d) more international support for these aspects of adjustment, including the provision of more finance, flexibly provided and with longer term commitments. The extremes of the present situation will often require a ceiling on outflows of interest and debt amortization if the protection of human needs is to be feasible in the short run.

*Thirdly, a system is needed for monitoring nutrition levels and the human situation*

confident assertions in such studies, however, considerable uncertainty exists about the impact of economic adjustment programs on health and nutrition in developing countries. The purpose of this section is to assess what we currently know about the impact of economic adjustment in the developing economies on child health and nutrition.

### **Section 2.1. Implications of Economic Theory for Analysis of Adjustment Policies**

Economic theory provides frameworks for analyzing many of the possible links between adjustment programs and the poor and child health and nutrition. Such structures are essential for gaining understanding of these complex phenomena given the very imperfect state of relevant information. But before sketching out the implications of economic theory for this topic, some limitations are noted of economic theory in this regard. First, for some links in the process there is considerable controversy about which of several alternative theories is most relevant. Second, economic theory leads to clear-cut predictions regarding the direction of changes in many contexts only by abstracting from some possibly relevant characteristics of the situation. Third, often the net effect depends on which of several counteracting responses is most important, which is an empirical -- not a theoretical -- question. Fourth, even if the direction of an effect is predicted clearly by economic theory, the magnitude still is an empirical matter. Fifth, economic theory is most useful regarding comparative statics between equilibrium outcomes, but has very little to say concerning the nature or the lag in adjustments between equilibria. For all these reasons, economic theory often leads more to raising useful questions about responses to the income and price changes induced by economic adjustment policies rather than providing precise answers.

With such caveats in mind, I begin with a discussion of the impact of the major components of standard stabilization programs on resources under the control of lower-income people and then turn to household behavior.

2.1A. Impact of Major Adjustment Policies on those in Lower End of Income Distribution: The macroeconomy determines the aggregate supply and demand of goods and services, the overall price and employment levels, and the aggregate balance of trade in goods and services and international financial flows with the rest of the world. In the simplest form, the short-run equilibrium aggregate output (income) level and price level are determined by the intersection of short-run aggregate demand and aggregate supply. Aggregate demand depends on private and governmental consumption and investment and net foreign investment (i.e., exports minus imports). These major demand components, in turn, depend primarily on real permanent income and wealth (and probably the distribution of each among members of society), governmental expenditure minus revenue,

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*during the process of adjustment.* We should be concerned not only with inflation, balance of payments and GNP growth -- but also with nutrition, food balances and human growth. The proportion of a nation's households falling below some basic poverty line should be monitored -- and treated as one of the relevant statistics for assessing adjustment.

prices of international goods and services relative to prices of domestic goods and services, and credit availability and/or interest rates. In the adjustment experience of a number of developing countries in the 1980's, for example, retrenchments in governmental activities, reduced prices and increased availabilities of imported substitutes, and restricted credit and higher interest rates have tended to shift the aggregate demand curve to the left, though increased exports might work in the opposite direction.

Expectations also may affect aggregate demand and may cause behavioral responses to offset anticipated policy changes, as in the so-called Phillips curve tradeoff between inflation and unemployment. For given inflationary expectations, the economy can be moved along a tradeoff between inflation and unemployment by contractionary policy. However such policy may change (reduce) inflationary expectations, causing the Phillips curve to shift downward so that the actual movement of the economy has little impact on output and employment. There are those who argue that in fact the impact of privately-held expectations offsets any anticipated policy change, thus rendering economic adjustment policies mostly ineffective. The available evidence does not seem to warrant such an extreme position, but it does seem to be the case that expectations can affect significantly policy outcomes.

Returning to the aggregate demand curve per se, if interest rates or inflationary expectations rise, aggregate demand is likely to shift to the left. This causes a decrease in equilibrium real output and the aggregate price level, with the balance between price and output changes depending on whether the initial equilibrium is on a more vertical or more horizontal segment of the aggregate supply curve. In many sectors in many developing economies in the 1980's there seemed to be excess capacity, so the changes might have been concentrated on quantities instead of prices.

Short-run aggregate supply reflects the conditions in short-run variable input markets, primarily for labor, intermediate inputs, and financing, given capacity production levels. The short-run supply curve is likely to shift to the left, resulting in a higher price level and lower output (income) level if wages, intermediate input prices, or interest rates rise, if rationed credit becomes less available (assuming that any parallel or "curb" financial market is not well developed), or if production becomes less efficient. Often adjustment programs attempt to restrain wages (though with some accommodation to labor pressures) and to increase productivity through greater exposure to world markets, both of which shift the aggregate supply curve to the right. However the greater cost of imported inputs due to devaluation and higher interest rates work in the opposite direction. In the longer run, aggregate supply tends to shift to the right with increased physical (e.g., machinery and equipment) and human capital, improved technology, and improved institutions. A critical question is how long does it take for the long run to occur.

To model all of these processes and the impact of policy changes, the relevant product, factor (i.e., labor, capital, land), and financial markets must be represented. Addison and Demery (1985) provide a diagram that illustrates these complexities. As noted above, conceptually an appropriately specified (e.g., to include the rigidities) economy-wide model in the computable general equilibrium (CGE) class may be useful for organization and analysis (e.g., Dervis, deMelo and Robinson 1982), though such an approach as usually applied does not incorporate well expectations (e.g., if policies are correctly anticipated, the private sector may adjust to them in ways that make them

ineffective) nor financial phenomena.

With this background, now consider the distributional effects of the major components of stereotypic economic adjustment programs.

Currency devaluation is frequently a key component of adjustment programs. The wisdom of devaluation, however, has been the subject of much heated debate because of controversy over the effectiveness of devaluation in eliminating supply-demand imbalances, because of inflationary effects of devaluation, and because of the distributional and related political consequences of devaluation.

Devaluation increases the costs of imports and the prices of exports in terms of the domestic currency. The impact of devaluation on the balance of payments and on distribution (of major concern here) depends on the extent of expenditure switching and the extent of expenditure changes.<sup>38</sup>

Expenditure switching results from the increase in the prices of internationally traded goods (whether imports or exports) relative to nontraded goods. Under strong simplifying assumptions (i.e., perfect competition, profit maximization, no externalities, well-behaved production functions), such shifts benefit the inputs used relatively intensively in traded-good production and the consumers (relatively) of nontraded goods. The implications within a particular context, thus, depend on the factor intensity of production and the nature of consumption patterns.

If importable production largely is in capital-intensive industries, for example, the factor-intensity effect tends to favor profits and increase income inequality. To the extent that importables are basic foods produced largely by poorer members of society as in some other economies (e.g., Thailand) but not in others (e.g., Jamaica), however, the factor intensity effect is beneficial to these poor. Likewise, the consumption effect depends on the nature of the traded goods and who consumes them. To the extent that importables are primarily staple foods that loom large in the consumption basket of poorer members of society as in Jamaica, the consumption effect in itself is likely to worsen the position of the poor (though poor farmers and landless rural laborers producing competing staples may be net gainers if for them the factor-intensity effect outweighs the consumption effect.) Such considerations lead to questions in any particular case about relative factor intensities and relative marginal consumption propensities.

The more one moves away from the simplifying assumptions noted above, the less strong are any predictions about the effects on distribution of expenditure switching due to devaluation. If the formal-informal sector distinction is important, for example, the above results hold if and only if factors are mobile. But if factors are not completely mobile between the formal and informal sectors there are no unambiguous predictions (Knight 1976). Returns to the factor used relatively intensively in the production of traded goods and services increase in response to devaluation, but both the formal and the informal sectors may produce such products with very different factor intensities (i.e., capital-

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<sup>38</sup> If the country is a large enough actor in any international market to affect international prices, there is a further question about the nature of that impact. There is not likely to be such an impact for most products of most developing countries, since their exports are small relative to world markets. I maintain this "small country" assumption throughout this subsection.

intensive in the formal sector and labor-intensive in the informal sector as, perhaps, in tourism or textile production.)

Devaluation also may induce aggregate real expenditure changes, with feedback on distribution. The conventional result is that eventually exports expand and imports decline in response to the relative prices changes induced by devaluation under the assumption that these relative price changes persist; this eventually leads to an improved balance of payments in international terms and probably an increase in aggregate demand and (assuming some unused capacity and/or efficiency inducements of devaluation) in output and income. However such a process may be quite slow, particularly if exports are goods for which gestation periods are long (e.g., tree crops such as coffee and cocoa, minerals such as bauxite) and for which initial capacity is more or less fully utilized. There also may be offsetting factors that lead to devaluation being contractionary. On the aggregate supply side these factors include the importance of noncompetitive, imported intermediate imports in production and wage indexation in the formal sector. Both of these shift the aggregate supply curve to the left with devaluation. On the demand side, the net trade component of aggregate demand in domestic currency may be reduced if there is an initial large deficit (particularly before exports respond), consumption and investment may decline if wealth falls with devaluation (due to an increase in net foreign debt in terms of domestic currency), or if income falls (due to more rapid induced inflation than changes in factor payments), and investment may be reduced because of higher prices for tradeable investment goods. These contractionary demand and supply factors are perceived by many to dominate in the short-run response to devaluation. If so, the reduction in real expenditure is likely to reduce the real purchasing power of many poor people. This would happen as a result of reduced demand for the services and products of the informal sector. In addition, workers who otherwise might have been employed in the formal sector may move into the informal sector -- along with people who enter the labor force because other household members have lowered earnings or have lost work. This labor supply increase, along with reduced demand, would increase unemployment and reduce labor returns in the informal sector.

Contractionary monetary and fiscal policies almost invariably are part of structural adjustment programs since excess demand is perceived to be at the heart of the problem.<sup>39</sup> Supply expansion presumably requires some time, so demand restraint is viewed as essential in the short run and contractionary fiscal and monetary policies are widely thought to be the major set of tools for restraining aggregate demand. Such policies are likely to shift aggregate demand and aggregate supply to the left initially, resulting in a fall in equilibrium output and income and an ambiguous change in the equilibrium price level. The extent of such an impact, however, may depend critically upon how firms' and individuals' expectations respond to such fiscal and monetary price changes. Under the assumption that these policies are likely to have some negative impact on output, the balance of payments is likely to be improved due to the decreased real purchasing power of the economy (thus reducing imports and increasing exports), reinforced -- if prices are

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<sup>39</sup> In most developing countries monetary authorities do not have much independence from the fiscal authorities (in part because of poorly developed financial capital markets), so the two are considered together here.)

sufficiency flexible -- by a decline in the relative price of nontraded goods and services. For reasons discussed above, such output reductions -- and the related labor demand -- probably lessen the real income of the poor. However, there are possible exceptions once again. For example, if prices fall but nominal wages in the formal sector do not fall, worker who receive such wages may experience an increase in real income.

The extent and duration of the negative impact on the income of such groups depends on several considerations beyond the extent of the initial leftward shifts in the aggregate demand and supply curves. One important consideration, of course, is the extent of and gestation required for a longer-run positive supply shift. The larger and the quicker such a response, the less the likely toll on the real income of the poor. A second consideration is how the government cuts expenses and increases revenue and how such changes affect the poor. Reductions in credit subsidies to capital-intensive manufacturers or increases in income taxes are not likely to have much negative impact on poorer households, but reductions in public health expenditures, food stamps, school lunches or subsidies for inferior foods may have significant negative income and price effects on many of the poor. To the extent that the main asset of the poor is their human capital, cuts in health, nutrition and education programs from which they benefit are more likely to have negative long-term effects on their future command over real resources.

Direct wage and/or price policies often are part of adjustment programs. Since wage increases could offset substantially the impact of economic adjustment policies such as currency devaluation by shifting the aggregate supply curve to the left, adjustment policy packages often include some limitation on wage increases for government and formal sector employees. If effective, such policies reduce the real income of individuals who would have been employed in the affected occupations without the wage controls. This may increase overall income inequality, but is not likely to have a strong negative effect on most of the poorest members of society. This is the case because the poorest people are not likely to have been in such occupations if there were no wage policy. In fact, to the extent that the limitation on wages is effective, these relatively high-wage occupations are likely to be more accessible to the otherwise relatively poor members of society than they would be if there were no effective wage controls.

Price policies as part of structural adjustment programs are likely to involve increases in or freeing of previously controlled prices -- such as for transport, fuel, food staples -- to induce supply expansion, reduce government subsidies, and discourage demand. If such price controls were effective prior to the adjustment program, such a policy shift means increased nominal prices for consumers of these items. This is likely to reduce their real incomes, particularly if there is an effective "asymmetrical" price policy, in Foxley's terms (1981, p. 206), of restraining their wages at the same time that prices are increasing.<sup>40</sup>

Policies beyond devaluation to limit foreign exchange uses and to encourage foreign exchange generation also often are part of economic adjustment packages.

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<sup>40</sup> To the extent that the price controls were not effective to begin with, removing the price controls of increasing price ceilings obviously has little impact on distribution except for reducing the legal and extra-legal incomes of the members of the price control and monitoring organizations (who are not likely to be among the poorer members of society.)

Commonly imports are liberalized with reductions in quantitative restrictions and tariffs.

Reduced tariffs on imports have at least three types of effects on income distribution. First, such tariffs are a major source of governmental revenues in developing countries. If reduced tariff rates do not encourage imports too much, such revenues fall and thereby increase the governmental deficit, thus partially offsetting the contractionary fiscal policy discussed above. Second, reduced import tariffs change the relative price structure to factor production of exportables. Within the simplest trade model, a country exports goods which make the most use of resources which are relatively abundant in the country (presumably labor for a developing country trading with a developed economy.) Third, once again there is an impact on the relative prices that consumers face that depends upon the marginal propensities to consume importables relative to alternatives. If the importables are luxury goods consumed relatively by the rich, then the consumption price effect of reduced tariffs in itself improves the relative position of the rich.

Reduced nontariff import barriers to imports (such as quantitative restrictions) have the second and third effects noted in the previous paragraph, but not the first. Instead, the losses from having access to such quotas are likely to come from the administrators and recipients of such quotas. Neither of these groups is likely to include many poor people.

2.1B. Impact on Health and Nutrition Given Changes in Income of and Prices Faced by Households: The structural adjustment programs described above ultimately affect individuals or households through altering their incomes or the prices that they face. Prices are broadly defined to include the total costs to an individual or household to obtain goods or services, whether from a private vendor or a governmental agency. If, for example, health clinic services are reduced as part of structural adjustment programs so that patients have to wait longer for free (in monetary terms) services, the total price for those services has increased.

Households (including single-individual households) make decisions about time-use and consumption, given their assets and prices that they face for the use of these assets, consumption items as is discussed in Section 1.1. For the poor households of interest here, the primary asset is labor, with varying degrees of skills. That means that wages and the nature of employment options are particularly important in determining household income. But many such households also have land (e.g., subsistence households and small farmers) and capital (e.g., many households in the manufacturing or maintenance parts of the informal sectors as well as farm households), so returns to other assets also may be important in determining their income. The prices of obvious importance, given the interest in child health and mortality, are for the items that determine these outcomes most directly -- food, potable water, preventive health measures, curative health care, housing, sewage, clothing. But since the actual use of such items depends on relative prices and on real income (which depends on all prices for goods and services important in the consumption basket of the poor), other prices may be important as well.

The impact of structural adjustment programs depends on (a) how the exogenous and predetermine variables in the reduced-form demand relations discussed in Sections 1.3 and 1.4 are altered (particularly the relevant returns on assets and prices broadly-defined, but possibly also the environment) and (b) how the impact of such changes are filtered through the household. With regard to the latter, there are a number of critical questions. How responsive are individuals or households to relative price differentials? If the price

of nutrients from important foods increases due to an economic adjustment program, for example, is there little or considerable shifting to cheaper nutrient sources? How much fungibility is there in the use of returns from assets among individuals? To what extent, for instance, is the loss of a formal-sector job due to an adjustment program by one member of the household offset by increase activities of other members of the household in the informal sector or elsewhere? Does it make a difference whose returns to assets are affected (e.g., women versus men)? If women are induced to participate more in the labor force by changes in the economic environment, what are the tradeoffs between their increased command over market resources and any concomitant reduced time in household activities pertaining to child health and nutrition? To what extent how for how long (particularly given the long adjustment experiences for a number of countries) can the household draw down existing assets in order to tide its members over a transitory bad period? At what point are changes in the economic environment likely to lead to substantially increased probabilities of disintegration of the household, probably to the disadvantage of some of the more vulnerable members?

## **2.2 Empirical Evidence on the Impact on the Poor and on Their Children's Health and Nutrition**

No empirical studies currently available examine all of the links between adjustment programs and the poor and their children's health and nutrition in developing countries with theoretical frameworks such as those outlined in the previous section and careful empirical control for all relevant factors. But some studies have considered systematically some of the critical links in the process, and a few others have considered relations in which intervening links have been collapsed to simplify the analysis. A number of these studies are reviewed now, roughly following the order in Subsection 2.1 by starting with studies on the effectiveness of macroeconomic policy in developing countries, then the nature of government cuts at the time of fiscal and monetary contraction, then the nature of household responses to changed income and prices, and finally country studies focusing on "collapsed" relations between economic recessions or adjustments and more direct measures of education, health and nutrition.

2.2A. The Effectiveness of Macro Adjustment Policies on Macro Outcomes in the Developing Countries: As is noted earlier, this is a controversial topic. Simple aggregate supply and demand curves suggest to some that macro adjustment policies that shift aggregate demand can be fairly effective in altering equilibrium output if the initial equilibrium is on the more horizontal part of the aggregate supply curve and that they can be fairly effective in altering the equilibrium price level if the initial equilibrium is on the more vertical part of the aggregate supply curve. But some analysts (e.g., Taylor 1974) have questioned whether well-intentioned policy makers in developing economies have enough policy tools to pursue their multiple macro objectives given such factors as inadequate capital markets and resulting ineffective monetary policy. Others (e.g., Rao 1952) doubt that the initial equilibrium could be on the more horizontal part of the aggregate supply curve because of the more severe supply shortages and the role of a traditional semi-subsistence sector. If so, macro adjustment policies primarily affect the price level, but not output nor employment. Still others (e.g., Lucas 1973; Fernandez

1979; Barro 1979) have queried whether expectations may make macro policies ineffective in developing economies as may claim is the case in developed market economies; an example is the Phillips curve tradeoff between inflation and unemployment conditional on expectations that is discussed in Subsection 2.1A.

If any of these arguments are correct, adjustment programs would not have much impact on output and employment, and therefore probably not much impact on the poor and their children's health and nutrition -- though conceivably there still could be some composition effects.

There are a great number of empirical studies that are related to this issue. Unfortunately they do not settle the issue definitively because of the institutional differences among developing countries; the difficulties in capturing the macro complexities of any economy in a tractable empirical framework (particularly such features as adjustment lags and formation of expectations); and that some of the results of macro models reflect the initial theoretical model specifications (the "closure" problem). Nevertheless, a selective review of some of these studies is suggestive.

The tests of the Phillips' curve trade-off between employment or output and inflation include Lucas's (1973) seminal paper in which he presents tests for 18 countries, including five in Latin America plus Puerto Rico (the rest are in Europe or North America). His tests suggest that the "apparent short-term tradeoff is favorable, as long as it remains unused" (p. 333), but in countries with a volatile price history in the sample and with relatively high variance in demand (namely Argentina and Paraguay), the inflation-output tradeoff is very unstable. Brodersohn (1979), Fernandez (1979) and Barro (1979) report results that they interpret as basically consistent with Lucas's conclusion for five Latin American countries. Both Barro and Fernandez report that unexpected money supply changes are relatively ineffective in changing output (rather than prices), though Barro does report a significant effect for Mexico. Hanson (1980) reports "a small though significant relation between output or growth and 'unexpected' inflation" for "moderately inflationary" Latin American countries that is substantially stronger than that obtained by Barro and between the Lucas values for the 16 relatively price-stable countries on one hand and the two price-volatile countries on the other hand. Nugent and Glezakos (1982) report estimates for sixteen Latin American countries. They find no significance of unanticipated inflation for the least agricultural countries and a negative (not positive as in the standard Phillips curve) output impact for the more agricultural countries. They interpret the negative association to reflect the lack of supply price rigidities (which the Lucas formulation emphasizes) in agricultural societies in the spirit, in a sense, of Rao (1952).<sup>41</sup>

A second group of studies attempt to evaluate the impact of macroeconomic policies on aggregate economic output in developing economies by the construction of models of these economies. For the purpose of the evaluation of short-run impacts of economic adjustment policies, a major long-standing tradition is composed of macroeconometric models in the Keynes-Tinbergen-Klein tradition. There exist literally hundreds of macro models in this tradition (Lau, 1975; Beltran del Rio and Schwartz,

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<sup>41</sup> This interpretation does not seem very persuasive. But, in any case, they do not find a positive tradeoff.

1986). Such models initially tended to be specified along Keynesian aggregate-demand oriented lines as if the initial equilibrium were along a horizontal aggregate supply curve, with the implications including fairly substantial impact of economic adjustment policies. However many subsequent efforts have tried to incorporate supply constraints, devaluation impacts on costs of intermediate inputs and of investment goods, adjustment lags and other more realistic features of developing economies. Such studies usually report some, but much more limited impact of economic adjustment policies, often with substantial lags that make successful detailed predictions and policy formulation extremely difficult (e.g., Behrman, 1977).

More recently emphasis has increased on the use of social-accounting matrix (SAM) based and computable-general equilibrium (CGE) models for empirical analysis of economic adjustment impacts in developing economies (e.g., Adelman and Robinson, 1978; de Melo and Robinson, 1982; Dervis, de Melo and Robinson, 1982; Pyatt and Round, 1979; Pyatt and Thorbecke, 1976; Taylor, 1979, 1983). An interesting example of the use of CGE models is provided by the simulation studies of the impact of devaluation on distribution within three archetype economies (i.e., a primary product exporter, a manufacturing exporter, and a relatively closed economy) with CGE models by de Melo and Robinson (1980), which also are summarized in Dervis, de Melo and Robinson (1982). Some of the results of these simulations are that much greater devaluation is required to eliminate an initial balance-of-payments deficit in the relatively closed economy than in the others because of the greater dependence on imported intermediate inputs in that economy, that devaluation leads to an improvement in the income share for small holders for all three archetype economies, but mixed income distribution effects for other low-income groups depending on the structure of the economy, and that price shifts play a major role in changes in real income. Such approaches tend to have the advantages over traditional macroeconomic models: specifications that are better based in economic theory and more complete, including more attention to income distribution, than those used in most models in the macroeconomic tradition. On the other hand at times the estimation of these models is quite cavalier, and the focus on market equilibria often is accompanied by inattention to adjustment lags and expectations that may vitiate the value of such models and other models, such models are limited by some of their a priori assumptions (e.g., unitary elasticities of substitution in production in de Melo and Robinson 1980).

A third group of studies are country studies that tend to use less systematic modeling approaches and empirical tests to evaluate the economic impact of stabilization. Probably the best known of these are the volumes edited by Cline and Weintraub (1981) and Killick (1984a,b). The lesser emphasis on formal frameworks in these studies leads to a richer, but also looser, range of interpretations. With regard to income distribution, for example, Cline and Weintraub (1981, p. 35) note: "The contrast between the 30 to 40 percent real wage reductions in the Southern Cone [of Latin America] and nominal wage increases to maintain real wages in Pakistan is striking, and success at reversing inflation was at least as great in Pakistan, although from a lower base." Such results suggest the possibility in practice, and not only in theory, of cushioning at least some of the poorer members of society from negative consequences of adjustment programs by appropriate selection of the components of those programs. But since some members of society have

their incomes reduced in the short run if the economic adjustment program is successful in its effort to reduce a demand-supply imbalance by contracting demand, the question remains what determines if there is political will and power to protect the poorer members of society.

The empirical studies on the economic impact of macroeconomic adjustment problems have their problems, in part because of the complexities of such economies, the limitations of macroeconomic theories, and data inadequacies. Often there are questions about simultaneity, structural changes, unobserved variables such as price expectations, the time period for observations -- almost always a year which may be long for some of the questions of concern, lag patterns, and a priori restrictions on the implicit or explicit models. Nevertheless, such studies do seem to support the conclusions that economic adjustment policies often have some significant impact on macro outcomes such as output and employment which feed back through the income distribution and relative prices potentially to affect the poor and to alter their children's health and nutrition and that there have been a range of varied experiences with regard to the income distribution effects of such policies, which suggests that choices may exist to buffer many of the poor if there is sufficient political will to do so.

2.2B. Changed Composition of Governmental Expenditures in Adjustment Programs: Changes in the composition of governmental expenditures as part of economic adjustment programs may affect the poor and their education, health and nutrition. The conventional wisdom regarding such changes is that they probably discriminate against social sectors including health and nutrition. Hicks and Kubish (1983, pp. 2-3) provide several quotations about the alleged vulnerability of these social sectors in various writings of the World Bank. Other examples can be found in other sources, e.g., UNICEF (1984, p. 161). Despite such strong claims about the vulnerability of social service sectors to government cut backs, very little empirical work was undertaken on this topic prior to the study of Hicks and Kubisch (1983, 1984).

Hicks and Kubisch examine the patterns in such cuts by considering all 37 cases for which there were reported cuts in real government annual expenditures in developing countries during 1972-80. For each case they asked whether cuts in government expenditures in each of five categories (social, defense and administration, production, infrastructure and miscellaneous -- the last largely transfers to local governments) were larger or smaller than the average overall expenditure cut. Their results suggest that, contrary to the conventional wisdom, on the average social expenditures are the most protected of the five categories (somewhat more so in low-income countries than in middle-income countries). Hicks and Kubisch caution about drawing too firm conclusions from their estimates because of the small number of cases and because the expenditure data are for the consolidated central governmental accounts -- they do not include expenditures of publicly-owned corporations and enterprises, nor the expenditures of state and local governments.<sup>42</sup> Nevertheless, such results suggest that the frequent assumption

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<sup>42</sup> Preliminary studies by others also suggest that the use of category-specific price indices and comparison with a longer-run trend rather than just the previous year may weaken the extent to which social sectors appear to be favored, but such adjustments do not seem to lead to the conclusion that the social sectors are cut significantly more than

that social sectors are particularly vulnerable to cuts in governmental expenditures are wrong -- and perhaps the social sectors are even favored. On the other hand, cuts in the apparently more vulnerable production and infrastructure categories may have indirect impact on income and health through associated reductions in unskilled demands for labor in the construction sector. Pinstrup-Andersen, Jarmitto and Stewart (1987) update this analysis for 1979-1984. They find somewhat more ambiguous results than Hicks and Kubish report for the earlier period, but still with the social sectors not cut more than overall governmental expenditures in two out of three cases.

Cheap food policies, including food subsidies, are one form of social expenditures that are particularly relevant for the present concern about the poor and their children's health and nutrition and that have been studied in some detail for several countries, perhaps in part because of the urban demonstrations (if not riots) that have accompanied efforts to reduce or remove such subsidies in countries such as Egypt, Peru, Algeria, the Dominican Republic, Haiti, Morocco, Sudan, Jamaica and Turkey. What are likely to be the health and nutrition impact of lessening or eliminating cheap food policies?

This question is less easy to answer than it might seem to be *prima facie*. It depends upon the incidence of such subsidies across income classes, whether there would be supply changes associated with the removal of such subsidies (e.g., due to increased prices for agricultural producers), how the affected poor respond to the increased prices for some foods and the reduction in their real income due to such a policy change and whether there are any compensatory policy measures for those affected.

Pinstrup-Andersen (1985a, 1986a,b) surveys many of the existing studies of the impact of the removal of cheap food policies on the poor. Since food expenditures are such a large proportion of expenditures of low-income consumers (typically 60-80 percent of their income), food price increases considerably decreased the real income of low-income groups and generally have much greater impact on the poor than on the well off. Pinstrup-Anderson (1985a, p. 71) reports estimates of the real income impact on the lowest and highest deciles of households by income: 8.5 and 4.1 for Sri Lanka; 6.0 and 2.0 for Thailand; 5.6 and 1.0 for Egypt; 7.3 and 2.9 for India; and 7.7 and 6.5 for Nigeria for a 10 percent increase in all food prices. Typically cheap food policies are in effect or are effective only for a subset of foods (though often including some basic staples), so these estimates probably overstate the negative real income impact of adjustment policies that increased currently subsidized food prices by 10 percent. Nevertheless they suggest substantially greater relative impact of removing food subsidies on the poor than on others.

On the other side of the market, elimination of cheap food policies are likely to result in increased production prices for food producers.<sup>43</sup> Pinstrup-Andersen argues that recent research indicates that the short-run impact of food prices on the rural poor is much less favorable than might have been anticipated because many of the rural poor do

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the average.

<sup>43</sup> Unless the cheap food policies involved consumer subsidies with arbitrage precluded between consumer and producer prices.

not derive a large share of their income from food production-related activities. He also cautions against assuming too high food supply elasticities even with a longer time horizon because of other changes required to alter significantly food production, with concomitant gains to the rural poor.

Based on such considerations and additional studies that have looked at other dimensions of the food subsidy programs, Pinstруп-Andersen (1985a) concludes that "food subsidies have increased incomes and improved nutritional status among the poor, particularly, but not exclusively among the urban poor." He also observes, as have many others, that the cost of general food subsidy programs may be quite high, particularly if there is a tendency (as often seems to be the case) to attempt to keep the nominal price of subsidized foods relatively constant in the presence of overall increasing prices. Behrman (1988a, p. 126), cites examples of food subsidies accounting for 2-3 percent of GDP and up to 12 percent of governmental expenditures. Reducing the costs of these programs without severely affecting the real income of most of the poor would seem possible in many cases given the broad incidence of many existing cheap food programs. For Morocco, for instance, Mateus et al. (1986) estimate that the upper 30 percent of the income distribution received 47 percent of the subsidies and the lower 30 percent received only 16 percent of the subsidies. Ahmed (1979) reports that in Bangladesh two-thirds of the subsidized grain was distributed to urban areas even though most of the poor people reside in rural areas. Alderman and von Braun (1985) estimate that the absolute value of the subsidy is almost constant among income groups, including those in the middle and upper parts of the distribution.

For such cases, if targeting to limit the beneficiaries to the poor and nutritionally vulnerable is administratively and politically feasible at a reasonable cost, it may be very attractive from the point of view of adjustment programs. Such costs eventually tend to rise substantially as efforts to limit leakages to other groups increase, but Mateus (1985), Mateus et al. (1986) and a number of others argue that substantial reductions in the governmental budget and distribution costs of existing general cheap food policies can be achieved by greater targeting. Examples of targeting include subsidization of inferior foods (i.e., those that are consumed less as income increases) and direct distribution of food to those thought to be most vulnerable (e.g., infants and pregnant women).<sup>44,45</sup> For Morocco, Mateus et al. (1986) estimate that expanded targeted programs could compensate most of the poor who benefit from current food subsidies at a budgetary cost of 11 percent of current food subsidies. Even if this is a substantial underestimate due to overoptimism about the marginal costs of expanding existing targeted programs and about

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<sup>44</sup> There generally are leakages to other household members in the latter type programs. Beaton and Ghassemi (1979) reviewed over 200 reports on food distribution programs targeted towards young children and found that the net increase in the food intake of the targeted children was 45-70 percent of the food distributed. The other 30-70 percent of the food distributed increased the real income of the rest of the household, perhaps with nutrition and health benefits.

<sup>45</sup> Pinstруп-Andersen and Alderman (1986) and Rodgers (1986) discuss in some detail targeting approaches.

the extent of leakages, it suggests considerable possible budgetary gains from targeting. Likewise, Gavan and Chandrasekera (1979) claim that a shift in the second half of the 1970's to a more targeted program (including a shift to food stamps and the exclusion of about half of the population from the program) cut by more than half the fiscal costs of Sri Lankan food subsidies to the poor as a share of governmental expenditure. Alderman and von Braun (1985) have conducted careful simulations of different options for the Egyptian food subsidy system; they conclude that "major fiscal savings may be obtained only by substantial modifications [i.e., reductions] of the bread and flour price subsidy and the subsidies paid to consumers of the cooperative shops (e.g., for meat and poultry, macaroni) or by targeting."<sup>46</sup> From such examples, it would seem that a number of governments could cut budgetary expenditures on cheap food programs without directly worsening the income of most of the poor.

Cheap food policies are not the only social policies related to children's health and nutrition that may be cut as part of economic adjustment programs. Direct cuts on health services may have both an immediate and a longer-run impact and those on education may have a longer-run impact given the evidence regarding the direct relations between better education and improved health and nutrition (e.g., Isenman 1980, Behrman and Wolfe 1984, Rosenzweig and Schultz 1982a, Wolfe and Behrman 1982) and the possible indirect relation through earnings. Jimenez (1987) provides a recent survey of pricing policy for health and education in developing countries. He concludes that the efficiency gains from user charges for selected types of health and education (i.e., those for which the benefits accrue primarily to the individuals concerned, such as hospital care and university education)<sup>47</sup> could be substantial. In these cases, the elimination or lessening of subsidies probably improves efficiency because of a lack of positive externalities and public good characteristics such as are discussed in Section 1.1B, though some might question whether merit goods are involved. He adds that the impact of increased user charges need not be inequitable, since

"the present distribution of subsidies tends to be highly skewed towards higher income groups, who obtain greater access to more costly social services ... even if they are uniformly free for all. Under these circumstances, the expansionary effect of fee increases for selected services (and if possible, for selected individuals) may actually improve equity in the distribution of public resources".

Such a characterization suggests that as part of structural adjustment programs, social programs in health could be altered through selected user charges so that the poor were

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<sup>46</sup> In their simulation of targeting, they assume that the basic and additional rations are discontinued for all but the poorest 25 percent of the population, that this part of the population also received an additional course flour ration, and that bread at the current fixed nominal price is available only in the poorest 20% of urban neighborhoods (p. 28).

<sup>47</sup> But not including those such as some types of preventive medical care for which the benefits are considerable to society as a whole (i.e., there are large externalities).

not diversely affected. Politically this may be very difficult, maybe even more difficult than food targeting -- and if it were done, there would seem to be no good reason to reverse the process at the end of the adjustment programs.

2.2C. Household and Individual Responses to Income and Price Changes: The most important effects of structural adjustment programs on households or individuals are to change their real income and the prices, broadly-defined, that they face. Subsection 2.2B summarizes some evidence that reducing or eliminating government programs such as those providing general cheap food may considerably reduce the short-run income of many of the poor -- not infrequently by 10-20 percent, and in some cases by much more. Relative prices may change substantially, at the same time, and, for cuts of the sort discussed in Subsection 2.2B, the relative price changes are likely to be ones that discourage the consumption of goods that are presumably positively associated with nutrition and health,

Perhaps the most information about the probable impact of these income and relative price changes pertains to food consumption. Numerous studies report large income responsiveness for food expenditures, with reductions in total food expenditures often of eight to nine percent for every ten percent drop in income among poorer populations.<sup>48</sup> Considerable evidence also exists that the poor are very responsive to relative prices in deciding what food items to consume (e.g., see Alderman 1986; Pinstrip-Andersen 1985a). The income and price responses of the poor also are large with respect to nonfood health inputs, though there are many fewer studies of these than of the food-related responses. Gertler, Locay, and Sanderson (1987) and Gertler and van der Gaag (1988), for example, provide persuasive recent evidence for Peru and the Ivory Coast on differential health care responses by income classes. (See Sections 1.3 and 1.4 above.)

However the large responses in expenditures on food (or in aggregate quantities of food) do not necessarily imply large changes in nutrients. As is discussed in some detail in Subsection 1.3B above, to the contrary, even quite poor households apparently substitute considerably among foods, and change their nutrient intakes relatively little in response to income changes. If so, the impact of economic adjustment policies on nutrients consumed by the poor probably is much less than often is assumed. Note that this means that many cheap food policies are as much or more means of transferring income as of affecting nutrition directly.<sup>49</sup> Therefore if one is concerned with the income or the welfare of the poor, one should be concerned that reductions in food subsidies decrease income. But one should not too easily assume that these reductions greatly impair nutrition. Whether such results carry over to other factors affecting health is not obvious. They may if there are a number of differentially priced close substitutes, but it is not clear that this is the case.

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<sup>48</sup> Based on cross-country estimates, Behrman and Deolalikar (1988b) report a drop on the average of 8 percent for every 10 percent in income, with a somewhat larger (smaller) drop for poorer (richer) populations.

<sup>49</sup> One might ask why direct income transfers would not be preferable. Pinstrip-Andersen (1985a) argues that food subsidies are more politically acceptable.

A further question pertains to the impact of nutrient changes, to the extent that they occur, on health status. This is a somewhat murky area. However a number of recent studies suggest that the impact of small changes in nutrients may be much more in terms of body heat and perhaps energy expenditure than in terms of somewhat longer-run anthropometric or clinical measures of health status (e.g., Behrman and Deolalikar 1986; Payne 1985; Srinivasan 1985; Sukhatme 1982). To the extent that this is the case, the commonly assumed negative impact on longer-run health of economic adjustment may be overstated. But again there are some counterexamples. For instance, Isenman (1980) presents time series evidence for Sri Lanka that mortality has responded positively and significantly to the price of paddy, presumably in part through a link between price, nutrient intake, and health. Further, even if there is not an effect on anthropometric and clinical health indicators, if there is an effect of short-run energy expenditures, that is a matter of concern. Recent studies for the impact of nutrients on labor productivity in poor populations, with control for simultaneity, suggest that indeed there may be such an effect (Strauss 1986; Deolalikar 1988; Behrman and Deolalikar 1988d; Sahn and Alderman 1988).

2.2D. Country Studies Directed to the Evaluation of the Impact of Structural Adjustment Programs on the Poor and their Education, Health and Nutrition: The most visible of these studies are those associated with UNICEF (Jolly and Cornia 1984; UNICEF 1984; Cornia, Jolly and Stewart 1987). These studies do not formalize explicitly the links between recession and/or economic adjustment and the health and nutrition of children. But they attempt to use secondary data to characterize some of the links relating to factors such as unemployment, the composition of governmental expenditures, and direct indicators of health and nutrition.

The individual chapters in Jolly and Cornia (1984) provide a useful catalogue of trends, but relatively little information on changes due to recessions and economic adjustment programs. Given the project focus on finding possible negative recessionary and economic adjustment impact on children, the authors appear to have pressed hard to find examples of deterioration in children's conditions, but provide relatively little direct evidence of such deterioration. To the contrary, in Preston's (1986) words:

What is remarkable is that the best data on children's status in most of the countries reviewed -- that on infant and child mortality -- shows continued declines nearly everywhere. Nutritional status indicators also typically show improvement and so do school enrollment figures, despite downturns in governmental expenditure on health and education in some countries.

Preston therefore suggests that the appropriate conclusion from these studies would seem to be -- subject to conceptual and data difficulties -- that the available evidence from these studies indicates "how much can be achieved even in the face of unusual economic adversity -- surely good news for social policy..." Instead of such emphasis, however, the editors have a "penchant for stressing the negative trends...(a distinct minority) [which] receive the lion's share of the editors' attention in the introduction and summary." Thus, a set of studies that seem to lead to the conclusion of little, or at least unproven, systematic impact of recession and economic adjustment on health and nutrition, is summarized as

finding that adjustment policy usually multiplies negative recessionary impact on the poor and vulnerable -- see Jolly (1985) and the statements by Cornia in the summary to Jolly and Cornia (1984) that "the present crisis...has severely aggregated the situation of several social groups," "...child welfare indicators...are unambiguous in pointing to a deterioration in child status...," and "In most countries one observes...a serious deterioration in indicators of nutrition, health status and school achievements..."<sup>50</sup>

In a spirit similar to that of Preston, after reviewing a number of studies related to the impact of economic adjustment on health and nutrition, in Behrman (1988a, p. 132) I conclude:

Certainly I share the judgements expressed in the UNICEF studies that the situation of millions of poor (including children) is appalling, that substantial deterioration may have occurred due to recession and economic adjustment that is not very visible because of the poor quality of data monitoring health and nutrition outcomes, that the effects of such deterioration may be lagged and may appear only much later -- particularly for children (e.g., Selowsky and Taylor 1973), and that better data are desirable to understand the impact on nutrition and health outcomes. But I do not see these studies as having demonstrated that economic adjustment policies have had deleterious effects on health and nutrition in developing countries or that health and nutrition would have been substantially better without the economic adjustment policies or with different economic adjustment policies.

More recently I have been involved in an extensive examination of the Jamaican experience regarding the impact of structural adjustment on the social sectors and the poor (Behrman and Deolalikar 1988e). Detailed examination of the data available in this case leads me to conclude that Cornia, Jolly and Stewart (1987: 113-115) overstate substantially the evidence for such deterioration in their summary of this case.

### Section 3. Conclusions

Taylor and Hall (1967: 651), in a survey on health and development, wrote:

No problem in international health is as important or as poorly understood as the complex interrelationship between health, population growth and

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<sup>50</sup> Preston (1986) also notes that the case of South Korea, nicely reviewed by Sang Mok Suh in the Jolly and Cornia volume, scarcely is mentioned in the editors' introduction and summary even though South Korea has had rapid gains in child growth and child survival as well as high economic growth and relatively great success in adjusting to world economic fluctuations. Preston goes on to state "There is a certain irony when a strategy of economic development is so successful that one's social successes get discounted. But survey countries like South Korea are instructive when setting the broad parameters of social economy policy."

general economic development. Most traditional dogmas dealing with these interactions have proved to be oversimplifications. Casual relationships are not straight forward. Instead, they are buried in a complex matrix of multiple causes and feedback interactions. Findings and interpretation which are valid under certain conditions and at certain times may be totally reversed in somewhat changed conditions and at other periods.

Two decades later definite progress has been made in understanding the "complex interrelationships" among child health and mortality and general economic development. Some further dogmas have been found to be oversimplifications. Understanding has been improved of the role of simultaneous feedbacks. The roles of initial conditions in both micro and macro analysis are better appreciated and controlled. A few dimensions of the complex interactions within a macro framework have been explored. But, while there has been progress, nevertheless there remain quite considerable lacunae in our knowledge. In this section I try first to summarize what we have learned and then to point to areas where further research seems most promising.

### **Section 3.1. Summary of Studies Reviewed**

Poor child health and high child mortality in the developing world are widespread. Consideration of human physiological adaptability to its environment and the extent of inter- and intraperson variations leads to a less pessimistic characterization of the current situation and somewhat different identification of who is at risk. Nevertheless, large numbers of children in the developing world have lower nutritional input and health status than most would think desirable. Such inadequacies are likely to be exacerbated at times of unfavorable relative price movements for the poorer members of societies. These inadequacies persist in the late 1980's despite considerable absolute and relative gains in indicators of average child nutrition, health, and mortality experiences in developing countries in recent decades (e.g., Preston 1980, 1986; Hill 1985; Behrman and Deolalikar 1988b). These gains have been larger in fact than the gains indicated by narrowly-defined economic indicators such as per capita income. Of course the national averages hide a wide range of variances and, since the situation of the poorer may be particularly critical in determining average health and mortality outcomes, countries that appear similar according to the per capita income averages have had widely different average nutrition, health status and mortality. The life expectancy at birth in 1985 of 70 years in Sri Lanka as compared to 40 years in Sierra Leone, though both had per capita income estimates of about \$370 for that year, provides a vivid example.

Investigations of the micro determinants of child health and mortality or of health-care utilization, whether by estimating health and mortality production functions or reduced-form demand equations, have met with limited success. Some micro health production function estimates suggest that direct nutritional supplements improve child health. There also is considerable evidence of the importance of endowments, either as proxied by parental height or controlled in fixed effects estimates. In fact some a priori plausible results, such as those regarding the positive health impact of health services in Rosenzweig and Wolpin (1986), are obtained only if there is control for unobserved fixed

effects. Reduced-form micro demand relations for child health find some, but limited evidence of responses to relative market prices, income or wealth. The studies by Gertler and collaborators (see Section 1.3), moreover, find much larger price responses in health-care for lower than for higher income groups, which suggests that general reductions in health-care subsidies would be regressive. The micro estimates, however, contrast with the aggregate estimates of fairly strong associations between measures such as life expectancy and per capita real product or income. This contrast raises the question of whether the micro results are misleading because of measurement errors for health and/or income and specification errors regarding lags and time use, or whether in the macro estimates per capita income is representing not the purchasing power of individuals so much as the general level of development and associated public health measures that are not well-represented in micro estimates. A number of both the micro and the macro studies, further, indicate a substantial role of women's schooling, in some cases substituting for other inputs. The United Nations (1985), for example, concludes that variation in women's schooling is associated with much more variation in child mortality than the composite impact of a number of other development indicators, including per capita income. In one sample in which adult sibling deviations permit extensive control for the women's unobserved childhood background related characteristics, however, the estimated impact of women's schooling on health almost vanishes with such a control. The last result raises the question of whether schooling really is representing schooling in other studies, or largely is a proxy for unobserved ability and motivation. Moreover, even if mother's schooling is relatively highly associated inversely with child mortality in a cross-section sample, it does not follow necessarily that increases in mother's schooling are the most effective measure for improving child health and reducing child mortality, or even that they are more effective than income increases. This does not follow both because of the difficulty of interpreting variance decompositions to imply relative effectiveness and because such estimates do not capture the relative costs and time lags in alternative interventions (see Subsection 1.1C).

Reduced-form demand estimates for nutrients suggest substantial responses to food prices. These large price responses mean that many policies and market developments affect nutrition whether or not that is their intent. Policy makers need to be sensitive to such possibilities in their policy design and implementation. The food price nutrient responses, moreover, are not always negative; for some foods other than basic staples the price elasticities with respect to food prices appear to be positive and considerable. For such cases, subsidies for foods other than the basic staples may worsen nutrition, though improve welfare (unless there are offsetting income reductions due to the financing of the subsidies). The same outcome is more likely to hold for the rural poor since their income may be associated positively with such food prices. Another interesting characteristic of the price elasticities is a tendency for them to be larger for poorer households. Differential price elasticities across the income distribution present some possibilities of price policies that favor the poor for distributional reasons without too great leakages to those who are better off, though the latter seem to have sufficiently large price elasticities for most foods so that the possibilities are limited.

Estimates of nutrient determinants indicate a wide range of income or expenditure elasticities. However most of the large expenditure elasticities seem to result from

aggregation problems that ignore unit price changes associated with income, measurement errors, and the distinction between household food purchases and food actually consumed by household members. Apparently other food characteristics -- taste, appearance, status value, degree of processing, composition -- are valued much more than nutrition at the margin even in relatively poor populations. Cross-country estimates also suggest that in part the low-income elasticities of nutrients (as compared with those for food expenditures) reflect an increasing taste for food variety as income increases. If nonnutritive food characteristics that are not highly correlated with nutrients are favored at the margin, then income increases and the general development process will not alleviate malnutrition nearly as much as the World Bank (1981), Srinivasan (1985) and others have claimed. On the other hand, the apparent limited importance to individuals in such populations on increasing nutrient consumption at the margin (if they are making informed choices) raises doubts about whether they perceive reducing their malnourishment to be such high priority as many outsiders seem to suggest, and thus provides a different form of evidence consistent with the Sukhatme-Srinivasan-Seckler-Payne hypothesis about individual adaptability to nutrient availabilities and "small but healthy" people. Of course such evidence does not speak to the question, why are many people in some populations so small, nor does it allay completely the suspicion that the malnutrition experienced by many children in such populations is associated not only with small adults, but also with high infant and child mortality.

Some, but not all, studies point to the possible importance of women's schooling, nutritional knowledge, and public health measures in improving child nutrient consumption. The impact of women's schooling and nutrient knowledge may reflect that better-educated consumers make more nutritious food choices, ceteris paribus. The impact of women's schooling on nutrient consumption, in contrast to that on health, is robust to control for unobserved childhood background characteristics in the one sample that permits such adult sibling control. Better public health services such as safer water or better sanitation may increase the value of nutrients when such factors are present because of their complementarity with nutrients in the health production function. Women's schooling also may be playing such a role in addition to or instead of working only by improving information about nutrient qualities of different foods.

Partial-equilibrium investigations of policies have focused on the impact of subsidies for food and, to a lesser extent, other health-related inputs and on the cost effectiveness of alternative policies. Such studies suggest that general food subsidies are not very effective in redistributing income to the poor, but that better targeted food programs may be used to shift income to some segments of the poor that depend on market purchases for food. On the other hand, targeting types of individuals (e.g., small children, nursing mothers) instead of households is likely to be difficult if there is much intrahousehold fungibility, as appears to be the case. However, the small nutrient elasticities with respect to income imply that nutrient intakes do not improve substantially because of the income effect of such subsidies, though welfare may be improved significantly. But the price effects may be considerable, though they may reduce nutrient intakes if subsidies are on foods other than the basic staples. However some (but not all, see Section 1.3) recent studies of food subsidies and other health related input pricing policies suggest that in fact they often redistribute income from the poor and are not

justified on the grounds of externalities, though subsidies for preventative measures for contagious diseases may be justifiable on the latter grounds and subsidies for basic staples of the poor may be justifiable on distributional grounds. Within a general equilibrium framework, the means of financing health and nutrient subsidies may change their distributional impact considerably.

UNICEF and others have made strong claims about negative multiplied effects of macro economic adjustment policies on health and nutrition. Careful examination of the relevant studies, however, suggests that the empirical basis for such a claim currently is quite weak. In fact the underlying studies seem to be characterized better as reflecting how well societies and people have adapted to minimize negative health and nutrition effects rather than the more negative interpretation given by UNICEF.

### 3.2. Research Issues

Despite the growing number of studies of child health and mortality in developing countries, the lacunae in our knowledge remain substantial.

Major questions remain regarding the measurement of child nutrient input and health status, and thus the extent, incidence and determinants of nutrient and health inadequacies. The adaptability hypothesis proposed by Sukhatme (1982), Payne (1988), Srinivasan (1988), and Seckler (1980), for instance, raises difficult questions about how policy makers or other analysts can identify at a reasonable cost who is malnourished in a population. The failure to find much in the way of positive results regarding the determinants of child health, for another example, may be due to substantial measurement errors in representing health status. Frequently respondent-reported (or respondent-parent-reported) disease data are used as indicators, though such reports are likely to be determined endogenously by characteristics such as education and wealth. The benefits of analyzing the interrelations among various indicators and the extent of systematic measurement error in data sets in which there are multiple health indicators, as in Butler et al. (1987), may be considerable. The gains of obtaining clinical-based health indicators also may be worth the added costs. Perhaps it would be cost effective to obtain such information not so much by expanding socioeconomic surveys to include clinical information on health, but by working with epidemiologists and other health professionals and expanding their data collection efforts to include broader, more representative samples and more socioeconomic data, with sample designs to assure sufficient variance in critical price and asset variables. In the mean time, conclusions based on the data available to date have to be qualified because of their conditionality on quite imperfect health and nutrition indicators. More studies might explore fruitfully how robust are their results to efforts to incorporate measurement error into the analysis, such as latent variable specifications of random measurement error in health and nutrition that have been undertaken in several recent studies or the less common efforts to incorporate systematic measurement error. Another, less costly measurement improvement may be to use available data better. Waaler (1984) and Fogel (1987), for example, claim that systematic associations are clearer if anthropometric measures are used to calculate BMIs (body mass indices,  $\text{weight/height}^2$ ), though I am unaware of much use of such indices to date for studying developing country experiences. Of course there also are measurement problems

relating to other variables relevant in the analysis: income, prices, non-nutrient health inputs, and individual and community endowments. Some of these can be improved by better use of existing data, for example, by linking socioeconomic data on health and nutrition to a broader representation of current and past prices, environmental conditions, and policies. But other improvements require more careful and extensive data collection, not only regarding observable variables (the definition of which is somewhat endogenous), but also panel and adult sibling data to permit control for unobserved fixed effects.

Major questions remain concerning the determination of child health and mortality. Does the limited success in estimating micro child health and mortality production functions and reduced-form demand relations reflect data inadequacies regarding health measures, inappropriate lag structures, energy expenditure adaptations, or the failure to represent well prices and individual, household and community endowments? Does the endogeneity of policy -- for instance, the distribution of public health facilities particularly in areas in which unobserved (to the social scientist) health environments are unusually poor as analyzed by Rosenzweig and Wolpin (1986) -- mean that estimates of the child health impact of health policies are downward biased? What are the nature of the biological processes involved, the extent of substitutabilities and complementarities in health production processes, the nature of lags, and the role of nutrition? How does the slope of survival functions with respect to food intake depend on needs? How important is women's education in determining health? Is its often significant role in standard estimates reflective of increased productivity in using given health-related inputs, changed tastes that favor child quality, or is it primarily proxying for unobserved individual and household endowments as suggested by the one available adult sibling deviation study and by studies that include mother's height or control for health endowments in child health determination relations? If individual and household endowments are relatively important, what are the implications for designing fruitful interventions? What are the relative rates of return in terms of child health and mortality to increasing women's schooling, general development, and direct health interventions once costs and time lags are incorporated? Are education and public health measures substitutes broadly, as Rosenzweig and Schultz (1982a) suggest for their Colombian study and Barrera (1987, 1988) finds for the rural Philippines or complements as Strauss (1987) finds in his work on the Ivory Coast? More generally, what is the nature of substitution and complementarities in health production functions? How important are seasonal variations, particularly in rural areas? How can the very limited success in estimating micro health relations be reconciled with the fairly strong association between health and development across societies? What role does uncertainty play in the determination of health? Can recent analytical innovations, such as Waaler (1984) curves, help in understanding the determinants of and the impact of health?

Progress has been greater with respect to the determination of nutrient intakes, but questions remain. Would more extensive specification of prices, assets and endowments change our current understanding? What is the nature of the intrahousehold allocation process? Is it better represented by a bargaining framework? If so, what difference does it make? How fungible are resources within the household? For instance, how much do school children benefit from food provided at school and to what extent is such food received at school offset by receiving less food at home? Likewise to what extent do food subsidies, food stamps or food rationing improve the nutrition of recipients, or only result

in the increased purchase of other food characteristics or of nonfood goods and services? How fungible are resources across seasons? How much is nutrition likely to improve with income increases associated with the development process?

Some have argued that the determinants of child health and mortality and health-care and nutrient intakes and their impact on economic growth only can be understood fully within an economy-wide framework. Such arguments seem persuasive for programs such as malaria control or large scale food subsidies which have multiple effects and are of sufficient magnitude so as to change some basic prices in the economy. But such economy-wide modeling to date has had a number of limitations. Can these be overcome by better utilization of micro results, better specification (particularly regarding prices, endowments, income distribution and lags), and better estimation (regarding simultaneity and unobserved variables) and the models still be at least somewhat understandable to individuals who are not immersed in them? If so, what insights do such models offer about the impact of health and nutrition policies and about the effects of other policies (e.g., exchange rates) on health and nutrition? Further investigations with computable general equilibrium (CGE) models, along the lines of de Janvry and Sadoulet (1987), would seem valuable.

A major area of recent excitement in theoretical economics has been the examination of the impact on economic growth of externalities in human resources. Romer's (1986) and Lucas' (1988) work in this area has been catalytic, but a number of studies are underway. For example, Azariadis and Drazen (1988) develop a growth model with multiple equilibria in which slight differences in nutrient, health and other human capital investments may tip the economy from one stable growth path to another with a substantially different growth rate. Such studies raise anew the question of how important empirically are externalities in health and nutrient investments and whether there are substantial downward biases in existing estimates of the social returns to such investments because of the failure to incorporate such factors.

One major reason for concern with the determinants of child health, nutrition and mortality is to provide a better basis for policy formation. What are the implications of the growing corpus of empirical work on child health, nutrition, and mortality for policy? Can the apparent greater price and income responsiveness of poorer members of society (at least for nutrients and health care) be utilized in more effective policies? Do the low nutrient responses with respect to income undermine the rationale for some nutrient policies? Is the relative optimism of Mateus (1983) and Pinstrup-Andersen (1985) or the relative pessimism of Beaton and Ghassemi (1982) and Kennedy and Alderman (1987) about targeting food subsidies warranted? If income increases have little impact on nutrient intakes, might direct income transfers for distribution reasons be preferable to food subsidies? Can famine policies be improved by better empirical estimates of survival functions and by better understanding of food markets? What is the nature and extent of externalities, returns to scale, imperfect information or distributional concerns that warrant nutrition and other health-related input price subsidies? Are the distinctions between nonreferred curative care versus preventative care useful in making operational policy decisions about health subsidies? What would be the distributional and health impact of increasing user charges or of new insurance schemes for different types of health inputs in specific contexts? How is dissemination of new inputs and knowledge affected by

governmental pricing and subsidy policies, information problems and industrial structure? Do pharmaceuticals present special problems because of their relatively large share in developing country health budgets, the information problems regarding long-term effects, quality control, and the role of multinational companies? Can more appropriate health technologies be developed for the chronic diseases that are of increasing importance for developing countries, but for which current technologies are more appropriate for use in developed than developing countries? To what extent are policies justified in particular circumstances by externalities, public-good characteristics, returns to scale and other types of market failures or by distributional objectives? To what extent do governmental policies cause inefficiencies in the provision of health-related inputs? Do macro adjustment policies have multiplied or mitigated health and nutrition consequences? Can better policy design and monitoring lessen the negative effects? Do vested interests preclude sufficient shifts towards primary instead of secondary health care and towards more appropriate (and less sophisticated) health care that the World Health Organization (1978) and others have advocated? What are the economy-wide consequences of major health and nutritional policies? How do their impact on child health, nutrition, and mortality compare with the impact of other macro policies? And, finally, what are the "new political economy" factors that determine the selection of health policies and how can understanding them improve policy formulation?

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# Child Survival Programs Issues for the 1990s<sup>1</sup>

## Agenda

Monday, November 21

10:00 - 10:15

Opening Remarks

Henry Mosley and Pamela Johnson

10:30 - 12:30

Session A: Child Health in LDCs: The Status Quo

Chairman: Henry Mosley

Paper 1: Levels and Recent Trends in Mortality

Authors: Kenneth Hill and Anne Pebley

Discussant: Birgitta Bucht

Paper 2: Use of Health Services from DHS Surveys

Authors: Shea Rutstein, Elisabeth Sommerfelt, and  
Juan Schoemaker

Discussant: Carl Kendall

2:00 - 5:00

Session B: Primary Health Care Interventions: Performance and  
Prospects

Chairman: Kenneth Hill

Paper 3: Impact of the Direct Interventions

Authors: Anne Gadomski and Robert Black

Discussant: Stan Foster

Paper 4: Indirect Health Interventions with Reference to Family  
Planning and Breastfeeding

Author: John Hobcraft

Discussant: Joe Potter

Tuesday, November 22

8:30 - 10:30

Session C: The Immediate Context of Child Health Programs

Chairman: Ismail Sirageldin

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<sup>1</sup> This meeting was held November 21-22, 1988 at The Johns Hopkins University School of Hygiene and Public Health. It was organized by Kenneth Hill with administrative support from the Institute for International Programs. Funding was provided by the U.S. Agency for International Development.

266

**Agenda, cont.**

**Paper 5: Child Health Programs at Very High and Relatively Low Levels of Child Mortality**  
**Authors: Doug Ewbank and Susan Zimicki**  
**Discussant: Ron Gray**

**Paper 6: Infant Mortality Rates and Cause-Attributable Profiles: Some Implications for Primary Health Care Design**  
**Authors: Norbert Hirschhorn, Mark Grabowsky, and Robin Houston**  
**Discussant: Henry Mosley**

**11:00 - 1:00**

**Session D: The Broad Context of Child Health Programs**  
**Chairman: Ron Gray**

**Paper 7: A Survey on Socioeconomic Development, Structural Adjustment and Child Health and Mortality in Developing Countries**  
**Author: Jere Behrman**  
**Discussants: Jack Caldwell and Rolph van der Hoeven**

**Paper 8: Competing Risks in Child Mortality**  
**Authors: Henry Mosley and Stan Becker**  
**Discussant: Tony Augustine**

**2:00 - 3:00**

**Session E: Discussion and Recommendations**  
**Moderator: Kenneth Hill**

**Adjourn**

# Child Survival Programs: Issues for the 1990s

November 21-22, 1988  
Baltimore, MD

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*260*

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