BEENSTOCK, Michael

file Beenstock

Copies to Barnum
Chernichovsky

Any comment?
Please comment (or
decline to comment)
by mid-Nov.
Nancy

THE CITY UNIVERSITY
BUSINESS SCHOOL

Frobisher Crescent,
Barbican Centre, London EC2Y 8HB
telephone: 01-920 0111 telex: 263896

MB/SP

14th October 1985

Nancy Birdsall
World Bank
1818 H Street NW
Washington DC 20433

Dear Ms Birdsall

As you will see from the enclosed paper I am trying to estimate an econometric model of mortality rates in India by pooling time series and cross section (by state) data. The model may then be used for simulating alternative policies as described in the paper. These proposals develop some work that I did while I worked at the Bank in 1978 and which subsequently was published in my Health, Migration and Development (Gower Press, 1980).

It seems to me that some of my ideas complement quantitative research initiatives that the Bank has been undertaking. I was wondering whether the Bank might be able to take an interest in my project. Fred Galladay, Deepak Lal and Norman Hicks at the Bank know me well and may be able to advise you. In the meanwhile I would welcome the opportunity of telling you more about my work in this area.

Yours sincerely

Michael Beenstock

A MODEL FOR PUBLIC HEALTH SECTOR PLANNING IN DEVELOPING COUNTRIES

By

Michael Beenstock

City University

Business School

I would like to thank participants at seminars at the London School of Hygiene and Tropical Medicine and the Institute for Development Studies (Sussex) for their critical comments. I particularly wish to thank Anne Mills for her help.

" There is no other country in the world in which the tribe of pundits called economists are held in greater honour. Perhaps they are the only pundits who are at all honoured by us now. So India has become an El Dorado for every kind of economist from every part of the world".

Nirad C. Chaudhuri
The Continent of Circe, 1965.

I. INTRODUCTION

Micro & Macro

As reviewed in Appendix 1, a number of quantitative models have been developed during recent years with the objective of improving resource allocation in the health sector in Third World contexts. The models constructed by Grosse et al (1979) and Barnum et al (1980) are essentially microeconomic in nature. For instance, Grosse's model was developed for a specific region in rural Java rather than Indonesia as a whole. Similarly, Barnum et al are concerned with a specific region in Colombia rather than the country as a whole. These models cannot be regarded therefore as a basis for public health planning at the national level; they were never intended for this purpose. It is in this sense that they are microeconomic rather than macroeconomic.

In contrast, a macroeconomic resource allocation model is concerned with health sector planning at the national level rather than with the deployment of resources in specific regions and contexts. A comprehensive analysis requires both micro and macroeconomic frameworks for policy evaluation. The former is concerned with the general thrust of policy in terms of the overall allocation of resources to say preventive and curative measures. The latter is concerned with the deployment of resources in specific locations within the overall budgeting constraints implied by the former. Just as the overall stance of public expenditure might be determined at the macro level and specific deployment of this expenditure determined at the micro level, a similar juxtaposition applies to macro and micro decisions in the health sector. In this sense, macro and micro models are complementary.

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As yet there has been no attempt to develop a quantitative macroeconomic model for national health sector planning in Third World countries. Yet it is surely at the national level where the policy making process begins. Our objective is therefore to develop such a model using India as a test bed.

Indian Micro Analysis and Policy

In India a considerable volume of research has taken place at the micro level in the health sector, e.g. the Khanna and Narangwal projects. These and similar projects have been reviewed by Faruqee Johnson (1981) and Hardiman (1982) among others.Our purpose here is not to review this extensive literature but to note that there is a dearth of macro analysis of the health sector. On the other hand there is no shortage of criticism aimed at the government's handling of health sector planning at the national level, e.g. Rudra(1978), Ramasubban (1982) despite the enlightened approach to public health policy which the government has traditionally espoused in the form e.g. of the Minimum Needs Program. While much has been learnt from the micro level research, it seems worthwhile investigating whether it is possible to contribute to the health sector planning debate by research at the macro level. If the Indian test bed proves fruitful the methodology might be replicable in other countries.

It is stressed that the objectives of the research are methodological and are certainly not directly concerned with Indian public health policy. If the research is successful it will obviously have policy implications but not just for India. Rather we are using Indian data merely to see whether the methodology can be applied successfully in the first place. In Section II the theoretical framework is described. Section III discusses problems in implementation. Section IV considers the relationship between the health sector and the economy as a whole.

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I. MACRO THEORY

Basic Model

The number of deaths in age cohort j, in region r during time t from disease i is defined as

$$X_{jrti} = P_{jrt} A_{jrit} D_{jrit} \qquad (1)$$

where

P = number of people in the cohort

A = morbidity rate applicable to cohort j due to disease i

D = fatality rate applicable to cohort j with disease i

Thus in each time period a proportion A_{jrit} of the people in the cohort catch disease i. Of these a proportion D_{jrit} die. The A's must, of course, obey the following constraint

$$\sum_i A_{jrit} \leq 1$$

unless it is possible to have more than one affliction at a time. In terms of equation (1), measures that affect the A's might be thought of as preventive, while measures that affect the D's might be termed curative.

Morbidity rates are hypothesized as follows:-

$$A_{jrit} = F_{jri}(\bar{N}_{jrt}, \bar{W}_{jrt}, \bar{L}_{jrt}; A^{+}_{krit}, A^{+}_{jrkt}; U_{jrit}) \qquad (2)$$

In equation (2) partial derivatives are indicated over the variables to which they refer. We shall discuss the arguments in the function in turn.

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N_{jrt} is an index of nutritional status of cohort j in region r at time t. In the Indian context this may be denoted by average calorie consumption since as Sukhatme (1970) has noted Indian dietary habits imply that protein deficiency is not normally an independent problem. However, the work of Chen et al (1980), among others suggests that the relationship between A and N is nonlinear and that as N declines a point is eventually reached at which A rises rapidly. The specification of $\partial F/\partial N$ should also reflect the fact that after some point this partial derivative tends to zero. This point will be determined by the data rather than by preconceived notions of calorific adequacy suggested e.g. by WHO (1973).

W_{jrt} denotes the proportion of the cohort in the region who have access to potable water during time t. A similar variable could be proposed for sanitary disposal. The justification for variables of this type reflects the fact, see e.g. Bradley and Feachum (1978), that many diseases are transmitted via contaminated water and are in turn passed on by the lack of sewerage. Indeed, the preliminary efforts of Ruzicka (1982) suggest that W is likely to constitute an important part of the model.

L denotes the literacy rate. The case for specifying this type of variable is less transparent than the cases for N and W. However, students of development generally agree that health status generally improves with the spread of education although nobody is sure about the precise transmission mechanism. Cassen (1978) accepts that both improved health and lower fertility rates are likely to follow from the spread of education in the Indian context and Krishnan (1975) finds (see appendix 1) that statistically L is an important variable for explaining inter state differences in

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mortality rates in India. Therefore, L is a candidate variable in the model.

In the case of very young cohorts L_j is likely to be zero and the relevant variable is L_k where k refers to the parent cohort, i.e. the morbidity rates of children vary inversely with the educational attainments of their parents, as evidenced e.g. by Zachariah and Patel (1982).

If there are synergies between the transmission of diseases then the incidence of disease i will not be independent of the incidence of disease k. Hence the specification of A_{jrkt} in equation (2). Similarly, the incidence of disease i in cohort j may depend on its incidence in other cohorts. Hence the specification of A_{krit}. Thus A_{jrit} for all j and i are jointly determined.

Finally, u denotes a stochastic term since morbidity is inherently stochastic. However, the u's are unlikely to be independent.

First

$$E(U_{jrit}, U_{krit}) > 0$$

because random factors that affect one cohort are likely to affect other cohorts too. Secondly,

$$E(U_{jrit}, U_{jrkt}) > 0$$

because random factors that induce one disease may be responsible for inducing others too. Thirdly,

$$E(U_{jrit}, U_{jkit}) > 0$$

because random factors that affect morbidity in one part of the country are likely to be present in other parts of the country. Hence equations (2) which are jri in number are likely to have a

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complex stochastic structure, suggesting a GLS rather than an OLS framework for econometric estimation.

Equation (2) would in certain cases include medical expenditure. For instance, in the cases of malaria, TB and smallpox morbidity rates vary inversely with the degree of immunization and other preventive measures. Most probably in these cases the most important explanatory variable is expenditure on preventive programmes.

Fatality rates are hypothesized as follows:-

$$D_{jrit} = G_{jri}(H_{jrt}^{-}; N_{jrt}^{-}, W_{jrt}^{-}, L_{jrt}^{-}; V_{jrit}) \qquad (3)$$

Note that here D denotes the conditional rather than the overall fatality rate, i.e. D is conditional on morbidity. The overall mortality rate is of course equal to AD.

In equation (3) H denotes a vector of curative health measures including doctors per capita, hospital beds per capita, etc. Or H could be consolidated into per capita health expenditure. In the Indian context it would most probably be necessary to include Primary Health Care centres and related institutions in the H vector. Empirical estimates of $\partial G/\partial H$ would then measure the influence (if any) of PHCs on mortality rates.

N, W and L have also been specified in equation (3) on the grounds that they most probably have an independent influence upon fatality rates. Thus, for example, nutritional status not only affects the individual's susceptibility to disease as in equation (2), it also

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affects his ability to survive the disease once he has caught it. Thus,

$$\frac{\partial X}{\partial N} = P\left(A\frac{\partial D}{\partial N} + D\frac{\partial A}{\partial N}\right)$$

i.e. the relationship between death and nutritional status reflects the preventive and curative aspects of N.

The V's are stochastic disturbances which as in the case of the U's are likely to be related. In addition, there may be interdependencies between the U's and the V's. For example

$$E(U_{jrit}, V_{jrit}) > 0$$

i.e. random factors that increase morbidity may also raise fatality rates.

In principle equations (2) and (3) can be econometrically estimated by pooling cross section and time series data (i.e. over r and t space). The model would then consist of $2ji$ equations which solve for the ji morbidity rates and ji conditional mortality rates. GLS estimation is necessary for two reasons. First, because OLS estimate of equation (2) would be inconsistent reflecting the interdependence between the A's. Secondly, the stochastic structure of the model implies that OLS estimation would be inefficient.

Further Constraints

We have said very little thus far about the appropriate functional forms for equations (2) and (3). Any estimation in this area must recognise the fact that fatality and morbidity rates have lower

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limits irrespective of the values of the exogenous variables. However, it remains to be seen whether these constraints are of any practical relevance in the Indian context. In addition, there may be synergisms between the explanatory variables. For example, as W falls enteric disease is likely to be more prominent in which event anthropometric measures of nutritional status are likely to decline, i.e. a given calorie intake is less nutritionally effective because of the so called 'leaky buckets' effect. These arguments suggest for example that $\partial F/\partial N$ is not independent of W. A functional form that achieves this is e.g.

$$A = \alpha N^{-\beta} W^{\gamma} \ldots.$$

which implies that

$$\frac{\partial^2 A}{\partial N \partial W} = -\alpha \beta \gamma N^{-(\beta+1)} W^{\gamma-1} < 0$$

i.e. as W rises the effect on morbidity of the marginal calorie is increased. This is because a smaller proportion of the marginal calorie is leaking from the bucket.

Policy Analysis

The model may be used to simulate the effects of policy and resource allocation more generally. In India (and indeed in most countries) the government at both state and national levels plays a major role in determining H, W and L. At the same time the private sector sometimes makes important contributions in these areas. This is not the occasion to discuss the optimal balance of provision between the public and private sectors. Rather we concern ourselves with the best allocation of resources irrespective of their source as between the public and private sectors.

Most probably the most important determinant of N is income per capita (Y_{jrt}) which in India (and indeed most countries) is outside the direct control of the government. However, insofar as the government is able to influence the rate of economic growth its

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policies have indirect implications for N. Therefore, one way or another, the model implies that the government has a major influence on mortality and morbidity.

If the goals of economic development include reduction in death rates the model may be used at the macro level to determine the optimal resource allocation that minimizes the number of deaths. Thus, the government might seek to minimize

$$X_t = \Sigma\Sigma\Sigma X_{jrit} \qquad (4)$$
$$jri$$

More likely still it would seek to minimize death rates over time

$$X^* = \sum_{t=1}^{T} X_t (1+\partial)^{-t} \qquad (5)$$

where ∂ is a policy discount rate. As we shall see, X^* is the appropriate minimand because the problem of resource allocation in health sector planning is inherently intertemporal in nature. Therefore the model consisting of equations (1), (2) and (3) may be used to minimize X^* subject to the budget constraints, represented by equation (6).

In specifying the budget constraint it becomes clear that the policy instruments have different current and capital characteristics and that they have different fixed and variable costs. For example, the supply of doctors entails current expenditure while the supply of, say, literacy, entails capital expenditure. For purposes of simplification once a person has become literate he remains so in the future. Similarly, once investment in water supply has been undertaken these facilities remain in the future.

While L and W are similar in this regard they differ in other respects.

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W essentially consists of fixed costs; once the facilities are established many people may use them at negligible marginal cost. In contrast, L costs are entirely incurred at the margin.

The individual cost items may be summarized as follows. The cost of nutrition to the nation is

$$C_{1t} = K_1 \sum_{jr} \sum N_{jrt} P_{jrt}$$

where K_1 denotes the shadow price of a calorie.

The cost (per period) of water supply provision is

$$C_{2t} = K_2 \sum_{r} P_{rt} \Delta W_{rt}$$

where K_2 is the marginal capital cost of provision of water supply (and sanitation). Costs during time t are only incurred by increased coverage; hence the specification of $\Delta W > 0$. Also, the supply of services are location specific rather than cohort specific; hence the absence of subscript j. This implies that

$$W_{rj} = \frac{P_r}{JP_{rj}} W_r$$

for a given regional coverage the coverage of a specific cohort varies inversely with its share of the total population in that region.

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The cost of literary provision is

$$C_{3t} = K_3 \sum_{jr} \sum P_{jrt} \Delta L_{jrt}$$

where K_3 is the marginal cost of literacy. Because of the capital dimension to literacy C_3 is defined in terms of $\Delta L > 0$ because the costs are incurred by increased coverage. However, because literacy is specific to the individual it is necessary to specify the subscript j.

Finally, curative health costs (H) are assumed for simplicity to consist of current expenditure on a regional but not cohort specific basis. Thus

$$C_{4t} = K_4 \sum_{r} H_{rt}$$

which implies that

$$H_{jrt} = \frac{H_{rt}}{P_{jrt}}$$

In each time period the budget constraint is

$$C_{16} + C_{2t} + C_{30} + C_{4t} = C_t \qquad (6)$$

Because the objective functional depends upon the levels of L and W via equations (2) and (3) while the constraints depend upon ΔL and ΔW static optimization is neither appropriate nor possible. Instead dynamic optimization is necessary.

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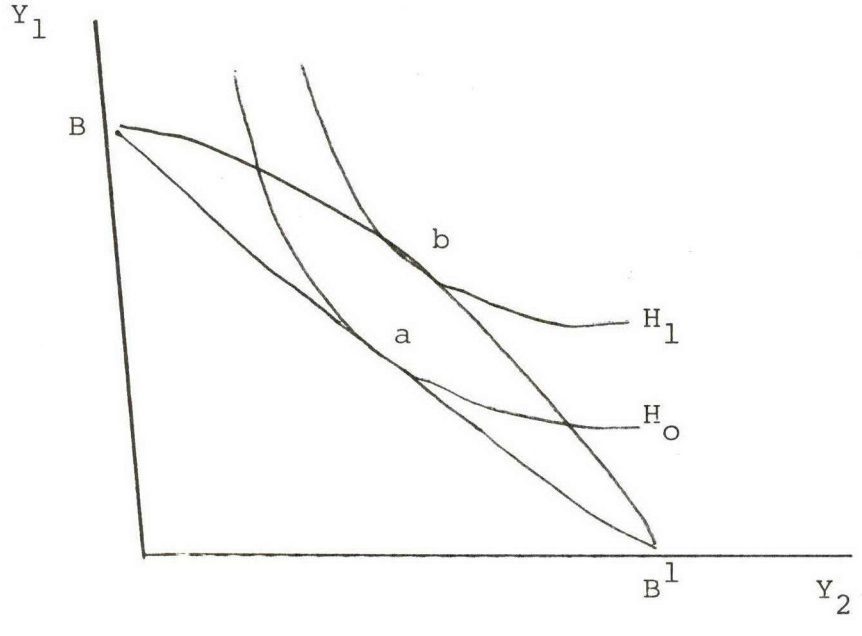
Overview

There is little purpose in working out the optimization analysis here in the absence of an estimated model. In any case, policy makers will in the first stage find the dynamic multipliers of the model to be of greater interest perhaps than the optimal policies themselves.These multipliers will indicate what is likely to happen over time if, say, water supply provision is increased on a ceteris paribus basis. In this way, policy makers will learn a great deal about the sensitivity of public health to various policy initiatives and indeed to economic growth.

Static Optimization:-

Nevertheless, a number of simple illustrations of how the model might be used for allocating resources are made.



On the diagram Y_1 and Y_2 are inputs of different health resources and the budget line is represented by $B_a B^1$. H_o and H_1 are members of a family of iso-health schedules. Thus, H_o describes the locus of combinations of Y_1 and Y_2 that keep the public health index at a constant level. The iso-health schedules are implied by the model

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$$H = F(Y_1, Y_2)$$

The optimal resource allocation is determined at a. However, if there are economies of scale or synergies in the supply of Y_1 and Y_2 the budget line will be concave to the origin e.g. BbB^1, in which case the optimal resource allocation is determined at b and a higher level of public health can be obtained for a given budget.

This emphasizes the point that it is not only important to develop the model, it is also important to consider the possibility of economies in the joint supply of health related inputs, e.g. water supply and sanitation, doctors and hospitals, etc.

Dynamic Optimization:-

As has already been observed, because public health depends, for example, on the level of literacy while the budget is affected by changes in literacy, the optimization problem is inherently dynamic. To illustrate this, we hypothesize the simplest of models

$$X(t) = F(L(t))$$

where $F^1(L)<0$, i.e. the number of deaths varies inversely with the literacy rate. The objective functional is

$$J = \int_0^\infty X(t)e^{-\partial t}dt$$

where ∂ denotes a policy discount rate. In each period the budget costs of literacy promotion are

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$$C(t) = k\dot{L}(t)$$

where k denotes the unit cost of literacy promotion which for simplicity is assumed to be fixed. The planners have a budget constraint of the form

$$C^* = \int_0^\infty C(t)e^{-rt}dt$$

where r denotes the rate of interest. The planners' problem is to minimize J subject to the model and C^* which is an isoperimetric constraint. The functional to be minimized is therefore

$$J^1 = \int_0^\infty \left(F(L(t))e^{-\partial t} + \lambda k\dot{L}(t)e^{-rt}\right)dt$$

where λ denotes the Lagrange multiplier. The Euler equation is

$$F'(L(t))e^{-\partial t} - r\lambda ke^{-rt} = 0$$

To calculate the optimal time path for $\dot{L}(t)$ the last expression is totally differentiated with respect to time and the result solved as

$$\dot{L}(t) = \frac{\partial F'(L(t))}{F''(L(t))} - \frac{r^2\lambda ke^{(\partial - r)t}}{F''(L(t))}$$

λ and $\dot{L}(t)$ are simultaneously determined by the last equation and the constraint C^*.

Similar solution paths will exist for all stock-type health interventions e.g. investment in water supply and other capital expenditures.

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Equation (5) is no more than illustrative. The objectives of public health planning will no doubt relate as much to morbidity as to mortality. Not only is the incidence of morbidity important but also its duration. However, there is a particular dearth of data regarding duration. Planners might also ascribe differential weights towards the morbidity and mortality rates of various groups. For example, they might give more weight to the young at the expense of the old. Clearly, for any given model of the health sector the optimal resource allocation will be conditional on the specification of the objective functional. The central concern of this research is therefore with the estimation of the model.

III. IMPLEMENTATION

Data

Mortality and Morbidity:-

To estimate a macroeconometric model of the Indian health sector data are required for all the variables that appear in equations (1) to (3). The observation space will consist of pooled cross section and time series data. Most probably the cross section will be aggregated at the state level because it is often in this form that the Sample Registration System (SRS) and the National Sample Survey (NSS) prepare their tabulations of the data. However, it may be possible to disaggregate beyond this subject to SRS and NSS cooperation and of course the data themselves. The states plus union territories generate 29 observations. If at least three time based observations can be found there would be at least 87 observations from which to estimate the model. No doubt much of the information would be generated by the cross section. Nevertheless, it seems probable that sufficient degrees of freedom can be generated in order to fit a model.

This is especially true for mortality data generated by SRS. The quality of Indian morbidity data leaves much to be desired, as elsewhere. Certainly, the SRS causes of death data are crude.

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Morbidity surveys have been undertaken in rounds 17 and 28 of the NSS. In addition, there are some intra state surveys of morbidity as well as some more accurate causes of death data (in Maharashtra). Most probably these data constraints imply that it will not be possible to estimate equations (2) to any degree of satisfaction. However, equations (3) can be substituted into equations (2) to obtain reduced form equations in terms of overall mortality rates (AM). In this case, it would most probably be no great loss to drop the i subscripts by aggregating diseases. This would force us to use death rates as indices of public health.

One exercise that seems worth carrying out is to see how closely morbidity and mortality rates are correlated. If they were highly correlated either variable would be a good index of public health status since what happens to one happens to the other. There is of course no logical reason why a highly morbid society should be afflicted also by high mortality rates. However, it would be convenient in the present context if this were the case and it seems feasible to check this correlation from all-India and state specific sources.

Medical Data:-

Barnett (1977) and Ramasubban (1982) have described how public health expenditure on health at the state level may be identified. In addition, the Pocket Book of Health Statistics of India breaks down the allocation of personnel and materials by state. Thus data on H in equation (3) are available. Indeed, it is most probably possible to identify several elements in the H vector. Another useful data source in this context might be rounds 28 and 35 of the NSS.

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Nutrition & Income:-

The NSS is a rich data source on the geographical distribution of income and consumption. In addition, it is a source of data on the size distribution of these variables. Therefore the distribution of income at the state level may be a further explanatory variable in the model; ceteris paribus, those states with a larger proportion on low incomes may experience higher rates of morbidity and mortality.

Various rounds of the NSS have also elicited data on calorie intake by region. e.g. Panikar (1980). The National Institute of Nutrition in Hyderabad has also conducted all India nutrition surveys.

Literacy, Water and Sanitation:-

The censuses are a primary source of data on literacy, water supply, sewerage and caste. Indeed, in many respects the censuses constitute integrated data sets for other variables too. In addition, there have been a number of literacy surveys as well as surveys of other types of educational achievement, e.g. NSS rounds 31 and 34.

It therefore appears that a data base can be developed for the econometric estimation of a health sector model in India. The model would be primarily concerned with mortality but it may also be concerned with morbidity and its determinants. The cross section dimension of the data could be extended by including other subcontinental countries in the model such as Pakistan, Bangladesh, Nepal and Sri Lanka.

Aggregation

The epidemiology of public health is an inexact science. The linkages between infectious diseases are incompletely understood,

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There are, in terms of equation (2), two main aspects to this uncertainty, which are reflected in imperfect knowledge about the following partial derivatives

$$\partial F_{jri}/\partial A_{kri}$$

$$\partial F_{jri}/\partial A_{jrk}$$

The former refers to the marginal effect on morbidity for a given disease when other cohorts are diseased. This is the inter cohort transmission mechanism. The latter refers to the marginal effect on morbidity due to other diseases within the same cohort. This is the inter disease transmission mechanism. A third possible transmissio mechanism might be

$$\partial F_{jri}/\partial A_{krh}$$

where the transmission mechanism extends both across cohorts and diseases and a fourth might reflect inter-regional transmission

$$\partial F_{jri}/\partial A_{jki}$$

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This implies that excessive disaggregation of diseases and cohorts (i.e. higher orders of j and i) runs the risk of placing too much of a burden on the data. It is difficult to determine the level of aggregation ex ante which in practice will be determined through experimentation. However, the following relatively aggregated starting scheme is proposed:-

Disease (i)	Cohort (j)
Malnutrition	infants (first year)
Respiratory	children (1-4 years)
Infections	5-14 years
Diarrhoeas	15-44 years
Skin	45+ years
Cardiovascular	women of child-bearing age

This implies 36 morbidity and fatality rates. However, this maximum is unlikely to be attained because certain cells are unimportant, e.g. children do not generally suffer from cardiovascular disorders. It may also be possible to aggregate various cohorts for given disease groups and various disease groups for given cohorts. Therefore, the model might consist of approximately twelve behavioural equations for morbidity and fatality rates respectively.

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Estimation

It has already been observed that GLS rather than OLS estimation is appropriate. There is also a choice to be made between structural and reduced form estimation. This is best illustrated by considering the following very simple linear model for 2 cohorts and 2 diseases based on equation (2):-

$$A_{11} = \alpha_{11} + \alpha_{12}N_1 + \alpha_{13}A_{12} + \alpha_{14}A_{21} + U_{11}$$

$$A_{12} = \alpha_{21} + \alpha_{22}N_1 + \alpha_{23}A_{11} + \alpha_{24}A_{22} + U_{12}$$

$$A_{21} = \alpha_{31} + \alpha_{32}N_2 + \alpha_{33}A_{22} + \alpha_{34}A_{11} + U_{21}$$

$$A_{22} = \alpha_{41} + \alpha_{42}N_2 + \alpha_{43}A_{21} + \alpha_{44}A_{12} + U_{22}$$

In a 2 x 2 model there are four equations for A_{ji}. For simplicity we have assumed that apart from the interdependence between morbidity rates, morbidity only depends upon random factors (the U's) and nutrition (N). These equations may be estimated structurally using GLS. The system may be written as

$$\underset{\sim}{\alpha} A' = \underset{\sim}{\alpha}_2 N' + \alpha_2' + U'$$

where $\underset{\sim}{\alpha}$ and $\underset{\sim}{\alpha}_2$ are 4 x 4 matrices of coefficients and A', N' and U' are column vectors of A_{ji}, N_j and U_{ji} respectively. The reduced form for A is

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$$A' = \underset{\sim}{\alpha}^{-1}(\underset{\sim}{\alpha}_2 N' + \alpha_1' + U')$$

which may also be estimated by GLS since the matrix E(u'u)is non diagonal.

The advantage of reduced form estimation is that it removes the burden of estimating the interdependence between the As unless of course the model is exactly identified. The disadvantage is that it does not disclose this otherwise highly desirable information. In practice, it is sensible to estimate the model both structurally and in terms of its reduced form.

Equivalently, equation (3) may be written as

$$\underset{\sim}{\beta} D' = \underset{\sim}{\beta}_2 N' + \beta_1' + v'$$

where D' is a (4 x 1) vector of fatality rates, etc. The solution for D' is

$$D' = \underset{\sim}{\beta}^{-1}(\underset{\sim}{\beta}_2 N' + \beta_1' + v')$$

in which case overall observed mortality rates are

$$X' = \underset{\sim}{D} A' = \underset{\sim}{D}\underset{\sim}{\alpha}^{-1}(\underset{\sim}{\alpha}_2 N' + \alpha_1' + u') \qquad (4)$$

where $\underset{\sim}{D}$ is a (4 x 4) diagonal matrix where the leading diagonal consists of the elements of D'. The last equation is the ultimate reduced form of the entire system since it is based on observed death rates.

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IV. THE WIDER ECONOMY

Health and Productivity

Thus far we have ignored any beneficial effects that improved health might have upon productivity and output. If such benefits exist they should in principle be included in the cost benefit calculus discussed in Section II. Although our central concern is with the macroeconometric modelling of the health sector it is appropriate to consider how the relationship between the health sector and the wider economy might be analysed.

The literature concerning the influence of health upon productivity and output has been reviewed by Barlow (1979), Correa (1975, cap.4) and Beenstock (1980, cap.2). Broadly speaking, this literature has reflected two distinct methodologies. First, there have been micro studies e.g. Weisbrod and Helminiak (1977) where the data refer to individuals and specific diseases. Secondly, there have been macro studies, e.g. Malenbaum (1970) where the data are highly aggregated. For the most part, however, those studies have been in the minority.

By and large the evidence in favour of beneficial effects of health upon productivity is weak and patchy. The strongest evidence concerns the effects of iron deficiency anaemia on productivity; but in India iron deficiency is unlikely to be a major problem. There have been several micro studies in India. For example, Belavady (1966)was unable to find any relationship between calorie consumption and productivity in a sample of agricultural workers. Similarly, Satyanarayana et al (1972) were unable to find this relationship in a sample of coal miners. A criticism of these studies is that the observations on calorie consumption were most probably too high

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to have constrained productivity. In this context it is interesting to note that although Tandon et al (1975) could also not find any relationship between calorie consumption and productivity in a sample of North Indian road construction workers, they were able to find significant relationships between productivity and anthropometric measures of nutritional status.

For India in particular (and other countries more generally) it seems that there is little if any evidence to go on and that all we are left with is our intuition that at some point productivity must be constrained by health.

The Identification Problem

As Malenbaum has suggested the interaction between health and productivity may be analysed using macroeconometric methods. However, the causality is two-way; not only might health benefit productivity, but productivity might in turn benefit health. Indeed, the latter has been the concern of some of the studies reviewed in Appendix 1. By way of illustration, the following simple aggregate production function is postulated:-

$$Q = F(K, L(X), X) \qquad (5)$$

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where K denotes the capital stock and L denotes employment. The death rate (X) proxies public health status. X appears twice in equation (5). As X rises absenteeism is likely to increase so that the number of days worked declines; hence $L_x<0$. But even if absenteeism is not affected it may still be the case that labour productivity is adversely affected by poor health; hence $F_x<0$. Therefore

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$$\frac{\partial Q}{\partial X} = F_L L_x + F_x$$

is the overall effect of public health on output.

Equation (4) implies, however, that X is not independent of output

$$X = G(N(Q/P^{-}),H^{+}) \qquad (6)$$

i.e. X varies inversely with nutrition which in turn varies directly with income per capita. A loglinear representation of equations (5) and (6) may be written as follows

$$L = \alpha_o X^{-\alpha_1} P$$

$$Q = \beta_o K^{\beta_1} L^{\beta_2} X^{-\beta_3}$$

$$N = \gamma_o (Q/P)^{\gamma_1}$$

$$X = \partial_o N^{-\partial_1} H^{-\partial_2}$$

in which case

$$q = \beta_1 k + \beta_2 p - (\beta_2 \alpha_1 + \beta_3) x \qquad (7)$$

$$x = -\partial_1 \gamma_1 (q-p) - \partial_2 h \qquad (8)$$

where lower case letters denote natural logarithms. Equations (7) and (8) are over-identified; there are five independent parameters in the structural equations but there are six independent reduced form parameters (assuming k,p and h are exogenous). In practice the model is likely to be even more over-identified than this because equations (5) and (6) most probably should include further

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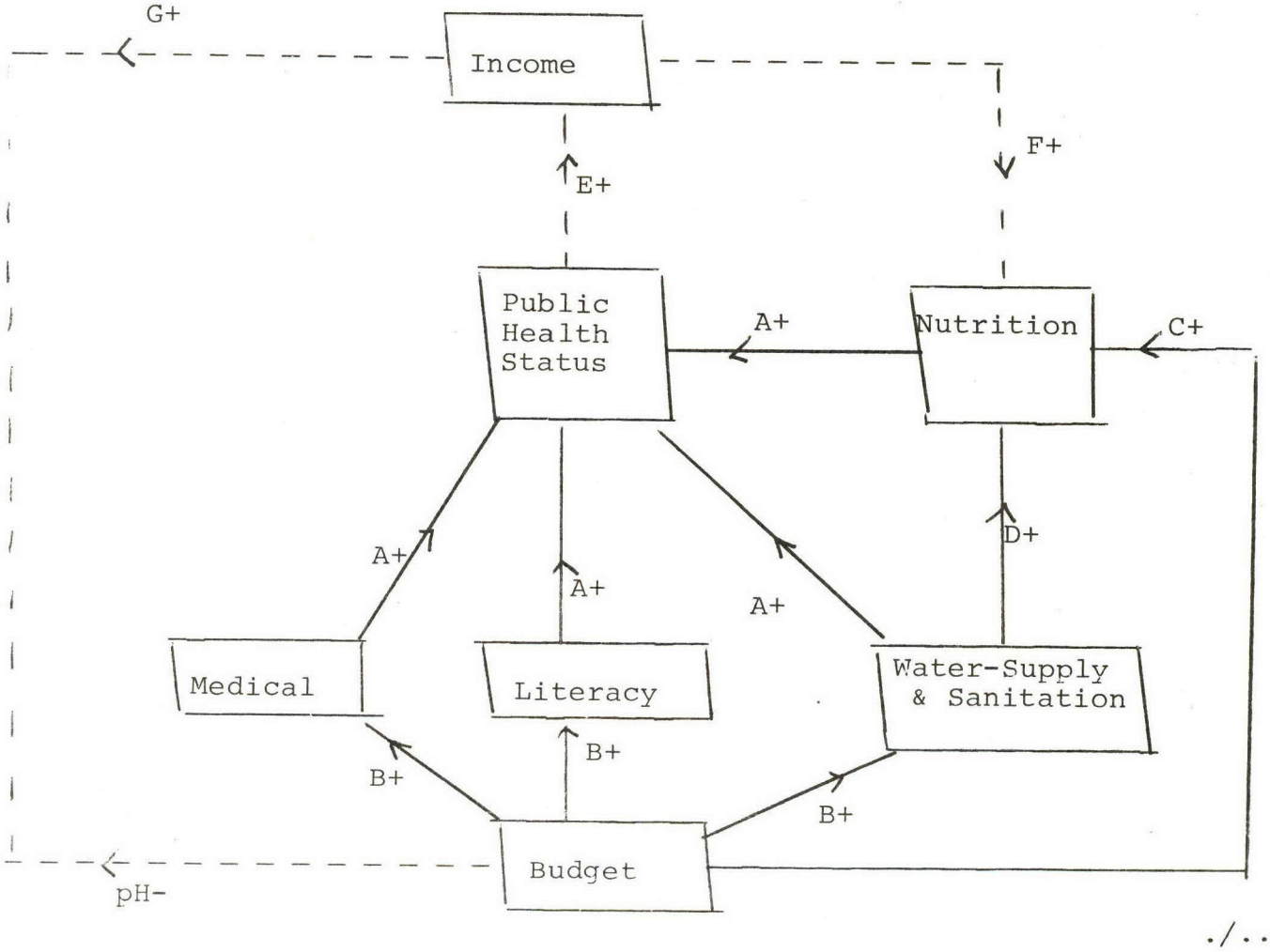
explanatory variables.

OLS estimation of equations similar to (7) and (8) will fail to unravel the two-way interaction between X and Q. Simultaneous estimation techniques are therefore appropriate. By pooling cross section (at the state level) and time series data for India it should be possible to estimate equations of this type. This should be feasible because the data requirements are similar to those discussed in Section III. Data for variables such as Q and K are available from the NSS.

Summary

The diagram summarizes the proposal. Broken lines refer to health-productivity interactions. Solid lines refer to the health sector model. Arrows indicate causal flows.



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The diagram is intended to simplify a complex set of relationships.

The relationships marked A reflect equations (2) and (3). Thus public health varies directly with literacy rates, nutritional status, medical resources and water supply and sanitation. The relationships marked B indicate that the resources are financed through the state or national budgets. Nutrition is provided out of household budgets, however, C reflects government financed nutrition interventions e.g. fair-price shops and other subsidized food distribution, Kumar (1979). D indicates that nutritional status might improve with water supply and sanitation since the number of 'leaky buckets' is reduced.

E reflects the hypothesis that output rises as public health improves. This in turn makes more resources available to households to increase their food consumption (F). It might also raise the tax base thereby enhancing the health sector budget (G). Finally, H denotes the opportunity cost of resources that are consumed by the health sector budget. It is for this reason that H carries a negative sign; as the budget is increased output is likely to be adversely affected. But if E is greater than H at the margin the net effect on income of the marginal rupee consumed by the health budget is positive.

The diagram implies that there are a number of multiplier effects. On the health side we have A → E → F → A. On the income side we have B → A → E → G → B. Econometric estimates of these relationships would enable a complete simulation analysis of these and other effects. Models of this type would most probably constitute an important advance in national health sector planning and economic planning more generally.

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Appendix 1.

QUANTITATIVE MODELS FOR PUBLIC HEALTH POLICY PLANNING IN LESS DEVELOPED COUNTRIES

By

Michael Beenstock

City University

Business School

December, 1983.

INTRODUCTION

Broadly speaking, the literature on the relationship between health and economic development has three main components.

i) There have been studies of public health improvements in the now advanced industrialized countries e.g. Eversley and Glass (eds.) (1965). These studies have largely been concerned with the factors responsible for the decline in mortality rates and morbidity rates over long periods of time within specific countries such as Great Britain and the United States. The factors that have typically been investigated consist of

a) medical discoveries and the removal of killer epidemics,
b) economic growth and its effects on health and nutrition,
c) socio-economic developments concerned with education, housing, water supply and related improvements.

The evidence from these longitudinal studies will not be reviewed here but an ecclectic conclusion is probably the most reasonable; all factors have been important. Nevertheless, it seems clear that dramatic reduction in mortality rates in nineteenth century Britain can only be marginally explained by the successes of preventive medicine.

ii) There have been studies based on international cross section data, e.g. Preston (1975). These will be reviewed briefly below. However, their concerns have been essentially the same as in the first set of studies.

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iii) Over the last few years studies have begun to emerge on quantitative health planning models for specific developing countries. The central concern of these studies is the optimal allocation of resources across the health sector as a whole, when the objective of policy is public health improvement in specific locations. The focus of these studies is different from the previous two types; however, there is a considerable degree of complementarity between them.

International Cross Section Analysis

These studies in turn fall into two groups. One group is essentially concerned with the relationship between mortality rates (M) and economic development when the latter is defined by per capita income by country (Y).

$$M = F(Y) \qquad (1)$$

where $F_y<0$. The second group is concerned with other socio economic factors (Z_i) such as nutrition and sanitation on public health as indexed by M or life expectancy:-

$$M = F(Y, Z_a) \qquad (2)$$

Examples of the former type are Preston (1975) and Cumper (1980). Preston examines equation (1) in three periods, 1900, 1930 and 1960. His main conclusions are

i) The evidence supports the hypothesis.

ii) However, its explanatory power is poor, suggesting that other factors apart from Y are important determinants of life expectancy.

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iii) The function has shifted over time, e.g. in 1960 a given level of Y was associated with higher life expectancy. This evidence further suggests the role of other factors in determining life expectancy.

Equation (1) scarcely provides a basis for health planning. In any event, it is a 'black box' that conceals the numerous interactions between M and Y that have been exhaustively discussed e.g. by Barlow (1979). As a possible basis for a quantitative analysis of public heal equation (2) seems more hopeful because it explicitly sets out the factors that might affect public health. Equation (2) is essentially a reduced form derived from a structural model of public health. Examples of studies based on equation (2) are Grosse (1980), Beenstock (1980) and Berrebi and Silber (1981). The latter authors inappropriately use factor analysis; their factors are loaded onto vectors of M and Z - type variables and it is difficult to imagine (since the authors do not explain) the justification for this. Their exercise amounts to little more than an unstructured analysis of correlations between the M and Z-type variables.

In contrast, Grosse and Beenstock use multiple regression techniques. Both authors find that literacy seems to be a dominant factor. However, other factors are also important. For example, Beenstock (1980, p. 44 equation 4) reports the following estimate of equation (2) based on a 1970 cross section of 36 developing countries

$$\begin{aligned} LIFEX = \text{constant} &+ \underset{(7.6)}{0.2126}\ LIT + \underset{(1.92)}{0.1136}\ CAL - \underset{(1.2)}{0.0004}\ DOC \\ &+ \underset{(2.14)}{0.06533}\ WAT + \underset{(2.22)}{0.05251}\ EXC \end{aligned} \qquad (3)$$

$\sigma = 2.67$ $\qquad \bar{R}^2 = 0.943$

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where

LIFEX = Life expectancy (years)

LIT = adult literacy rate (percent)

CAL = average calorie consumption per capita (percent of WHO-FAO requirements)

DOC = population per doctor

WAT = population served with potable water (percent)

EXC = population served with excreta disposal (percent)

't' values are shown in parentheses.

All the variables have the expected sign although it is clear that LIT is the most important regressor while DOC is the weakest. Apart from statistical significance the parameters suggest that a one percentage point change in LIT has a much greater effect on LIFEX than a one percentage point change in CAL, WAT and EXC. However, equation (3) is no more than an illustration of this kind of research. Nevertheless, insofar as estimates of equation (2) are reliable they provide an important basis for quantitative analysis of public health policies in developing countries. Apart from statistical problems the crucial question is whether international cross section results are applicable in country specific contexts. For example, are the Indian parameters the same as those in the international cross section? Without direct evidence there is, of course, no way of knowing.

Preston (1980) reports the following estimate of equation (2) based on a 1970 cross section for 120 countries:-

$$\underset{}{LIFEX} = \text{constant} + \underset{(6.51)}{4.249\ lnY} + \underset{(9.84)}{0.2086LIT} + \underset{(0.23)}{0.317\ lnCAL}$$

$$\bar{R}^2 = 0.858$$

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The sample therefore embraces both developed and developing countries. CAL is defined here as the excess over 1500 per day and is not statistically significant. However, LIT is highly significant and the implication of the equation is that this is the variable responsible for the upward shift in the relationship between life expectancy and GDP per capita previously observed in Preston (1975).

National Cross Section Analyses

However, there have been a number of attempts at estimating equation (2) using cross section data at the national rather than the international level. Patel (1980) uses regional (1971) data for infant mortality rates in Sri Lanka. In this case the model that is fitted reflects regional disparities in the dependent and the independent variables. The Z variables consist of water supply, sanitation and similar variables. Unfortunately, they do not include socioeconomic variables such as literacy rates or medical personnel and materials. However, Patel found that infant mortality rates could not be explained by regional health expenditure. Statistically, the most significant variable was access to well water; for every one percentage point increase in access the infant mortality rate fell by 0.81 per 1,000.

Curiously enough the second study is based on a cross section of eleven Indian states. Krishnan (1975) used the crude death rate as his dependent variable. Table 1 indicates the independent variables he used as well as their explanatory power from a step-wise regression analysis.

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Table 1. Krishnan's (1975) Model

Variable added	Variation explained (%)	Increase
Literacy rate	47.03	47.03
Bed-population ratio	58.65	11.63
Doctor-population ratio	65.35	6.70
Per capita income	70.85	5.50
Hospital-population ratio	74.49	3.64
Dispensary-population ratio	78.58	4.09
Urbanization	84.41	5.83
State per capita expenditure in medical and health area	86.34	1.93

It is clear from the table that literacy is the most significant factor. On this basis Krishnan fitted the following model:-

$$CDR = \text{constant} - 0.349Y - 0.243LIT - 0.00047DOC + 0.0004BED$$

where BED is the number of hospital beds per capita and Y is measured in rupees. Y, LIT and DOC have the 'right' signs but BED appears to take the 'wrong' sign. The R^2 is approximately equal to 0.7 but there are only five degrees of freedom.

The above table excludes variables such as sanitation and water supply, but it is noteworthy that like Patel, Krishnan found that health expenditures had no significant explanatory power.

Using specially collected micro (panel) data for 8921 births in three districts of Kerala in India over the period 1950 - 1978 Zachariah and Patel (1982) estimated various forms of equation (2). Table 2 reports their estimates of a probit model for infant mortality. The table suggests that the probability of survival to 12 months varies directly with the number of years the mother has

been educated. Water supply and sanitation are incorrectly signed but are not significant at the 95% level. The table emphasizes the importance of caste, birth status and order and total household expenditure.

Table 2 : PROBIT ANALYSIS ON SURVIVAL STATUS DURING FIRST YEAR OF EACH BIRTH

Dependent Variable: Probability of a Live Birth Surviving The First 12 months

Independent Variables	Coefficient	T-Statistics
Mothers education (years)	+0.04778	6.0
Caste 1/	+0.1937	3.4
Birth Status (single or twin)	-0.8947	8.8
Year and month of birth	+0.001326	5.3
Birth Order	-	+1.4
Linear	+0.1082	+3.6
Quadratic	-0.0108	-3.3
Per Capita expenditure	-	0.1
Land	-	0.2
Water Supply 2/	-	-1.1
Total Household Expenditure*	+0.0004096	3.7
Toilet Facility* 3/	-	-1.0

*From a different regression from which the variable per capita household expenditure was excluded.

1/ Scheduled castes and tribes = 0; all others = 1

2/ Pipe and well = 1; all others zero. A modification of this in which pipe = 1; all others zero did not affect the statistical significance.

3/ Flush and ESP Slab = 1; all others zero.

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While the model is statistically significant its explanatory power is very low suggesting that either infant mortality is extremely random or that other variables e.g. medical expenditures have been omitted from the model. It is interesting to note that there is a time trend in the model (year and month of birth) which suggests that survival rates have been rising over time. Whether this is autonomous or whether it reflects omitted variables that are time trended is important to consider.

Table 3 reports Zachariah and Patel's probit estimates for neo-natal and child mortality. These results largely mirror the estimates

Table 3: PROBIT ANALYSIS OF NEO-NATAL AND CHILD SURVIVAL STATUS OF EACH BIRTH

Independent Variable	Neo-Natal Survivourship (first month)		Child Survivourship (1-4 Years)	
	Coefficient	t-Statistic	Cofficient	t-Statistic
Mother's education	+.05219	6.2	+.03737	3.6
Caste	+.1316	2.1	-	1.7
Birth Order	+.02249	2.0	-	-1.7
Single or Twin Birth	-.9123	-8.8	-	-0.9
Year of Birth	+.007802	3.0	+.002406	6.8
Per Capita Expenditure	-	-1.6	+.001564	2.1
Land	-	-0.1	+.0003671	2.4
Water Supply	-	-1.1	+.1411	2.0
Total Household Expenditure*	+.0002669	2.4	+.0003343	2.3
Toilet Facility*	-	-1.0	+.1768	2.5

* From a different regression from which PCE was excluded.

reported in table 2. However, in the case of child mortality water supply and sanitation are significant and are correctly signed.

Simmons and Bernstein (1982) have carried out a quantitative analysis of neonatal and postneonatal mortality using panel data obtained in rural North India. Information on mortality is based on retrospective histories collected in 1972 from parents living in Uttar Pradesh. Retrospective data are particularly vulnerable to error. Using dichotomous multiple regression techniques Simmons and Bernstein tested for the following influences on mortality.

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- health environment, i.e. time to hospital and village survival rates.
- education.
- land holding (as a wealth measure).
- age of mother.
- parity.
- additional and gender of children desired.

Their main conclusions were that education of the parents does not significantly affect neonatal mortality. Nor does it affect post-neonatal mortality in the case of boys. However, education has a significant impact on female post-neonatal mortality. Another conclusion is that the health environment does not systematically affect mortality.

We now turn our attention to studies of the third type mentioned in the introduction of which unfortunately there are only two. We discuss these in turn.

The Michigan Model

Grosse et al (1977) working at the University of Michigan have developed a planning model with reference to Java but which is being applied to a range of other developing countries. Our concern here is with the theoretical structure of the model and its parametrization rather than with its detail.

Two measures of public health are proposed; death and days of disability. If there are P_j people in age cohort j and R_{ij} is the age specific attack rate of disease i it follows that the number of diseased people in the cohort is

$$\sum_i R_{ij} P_j$$

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Diseased people either die or they are disabled. A proportion F_{ij} are likely to die, hence the expected number of deaths in the cohort is

$$\sum_i R_{ij} F_{ij} P_j$$

If, however, a proportion N_{ijk} of the cohort with disease i obtain health care k the total number of deaths will be

$$M = \sum_{ijk}\sum\sum R_{ij} P_j (N_{ijk} F_{ijk} + (1-N_{ijk}) F_{ij}) \qquad (4)$$

On a similar basis it is possible to calculate the number of days of disability. However, equation (4) is sufficient for our present purposes.

Health sector planning is essentially concerned with reducing the attack rates (R) and determining the coverage of the services (N). Broadly speaking, R-type interventions are preventive while N-type interventions are curative.

Data for F_{ij} are either obtained by direct survey or they might be available from basic demographic sources. N_{ijk} is a policy parameter under the control of the planners. Assumptions are made for F_{ijk} by informed guesswork in the light of 'best medical opinion'. Since P_j is given it remains to determine the attack rates in terms of the following hypothesis

$$R_{ij} = H_{ij}(Y_j, Z_{aj}) \qquad (5)$$

where equation (5) correspond to cohort specific versions of equation (2). Assumptions for H_{ij} are arrived at through informed guesswork. Y_j is given.

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Total costs are

$$C = \sum_{ikj}\sum\sum C_{ijk}N_{ijk}P_j + \sum_{ja}\sum C_{aj}Z_{aj} \qquad (6)$$

where C_{ijk} is the unit cost of service k to those ill with disease i in cohort j and C_{aj} is the unit cost of preventive measure a to cohort j.

The optimal health strategy may be solved by minimising M with respect to Z_{aj} and N_{ijk} subject to equations (5) and the budget constraint, equation (6). In this we assume that C_{aj} and C_{ijk} are appropriately 'shadow' priced.

Apart from data difficulties regarding the F_{ij} and F_{ijk} the main proble covers the estimates of equation (5). These in practice are gleaned from international studies and from available pieces of local information. There can of course be no guarantee that the optimal solutions are genuinely specific to the locality. This would only be the case if the assumptions for H_{ij} and F_{ijk} are genuinely specific to the locality. However, these efforts which follow in the quantitative tradition first suggested by Feldstein (1970) and Paglin (1974) must be regarded as important aids in health planning as indeed conclude Russell and Black (1983) in their evaluation of the Michigan model prepared for USAID.

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Barnum et al (1980)

This model was developed in relation to child mortality in Colombia. While its scope and purpose is similar to the Michigan model, it differs in certain important respects. As before, our concern here is with the logical structure of the model and its parametrization rather than its details.

Barnum et al take account of the fact that attack rates are interdependent. For example, if the attack rate for measles is reduced, this is likely to affect the attack rate for respiratory problems. There are two aspects to this interdependence. First, as attack rates are reduced for disease i this automatically increases the chances of contracting disease n. For instance, if cancer were eliminated we all would become exposed to diseases that we would have contracted but for cancer. Secondly, there are synergies between diseases. These considerations imply that equation (5) should be modified to

$$R_{ij} = H_{ij}(Y_j, Z_{aj}, R_{nj}) \qquad (7)$$

The number of deaths is

$$M = \sum_{j}\sum_{i} R_{ij} F_{ij} P_j \qquad (8)$$

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as before. Whereas the Michigan model distinguishes those who are covered by service k from those who are not covered the present model directly hypothesizes the determinants of fatality rates as follows

$$F_{ij} = T_{ij}(U_{kij}, Z_{kj}) \qquad (9)$$

where U_{ij} are estimates of utilization rates of the k curative services. Total costs are

$$C = \sum_j (\sum_a C_{aj} Z_{aj} + \sum_k C_{kj} Z_{kj}) P_j \qquad (10)$$

To calculate the optimal public health plan, it is necessary to minimize M with respect to Z_a and Z_b subject to equations (7), (9) and (10).

Estimates for equations (7) and (9) were obtained by consulting a panel of experts and by using other available information. However, their analysis of the judgements provided by these experts was considerably more systematic than the analysis carried out by the Michigan team. This reflected the fact Barnum et al conducted independent interviews of their numerous experts. However, it is inevitable that this oracular methodology is always open to criticism irrespective of the care and sensitivity on the part of the investigators.

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Conclusions

A further study in the tradition of Grosse et al and Barnum et al is an Egyptian child health planning model described briefly by Correa (1975, pp. 133-146). In this model three diseases are considered, bronchitis, diarrhoea and measles; attack rates are assumed (on an oracular basis) for protected and non-protected groups and linear programming solutions are obtained for the optimal deployment of doctors, nurses and other financial resources in terms of preventive and curative interventions.

Perhaps the most important conclusion from this brief review is that the quantitative approach to public health planning in Third World countries has not only established itself within the academic community but increasing attention is being paid to it by policy makers. The main problem concerns the 'oracular' basis of the models and their parametrization. Optimization tends to expose the weak points in models and it is perhaps understandable if administrators and policy makers who cannot be guaranteed that the model is in fact valid are less than enthusiastic.

However, there is an alternative to the oracular approach. This is to follow in the tradition described above concerning the international and interregional cross section studies. In this tradition the model is fitted to data using econometric techniques - the data are the oracle. On this basis cross section and time series data for a given country may be pooled and econometric models of the health sector estimated. There have, as yet, been no attempts to estimate macroeconometric models of the health sector. The estimation of such a model would then provide a quantitative basis for the macro or national planning of public health in developing countries.

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