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HEALTH ECONOMICS FOR LOW-INCOME COUNTRIES

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ABSTRACT

Good health is a determinant of economic growth and a component of well-being. This paper discusses and synthesizes economic models of individual and household behavior, showing how they may be used to illuminate health policy making in low-income countries. The models could help address questions such as: How can the health of the poor be improved, and what are the economic consequences of better health? What policies would improve intra-household distribution of health outcomes?

An extensive literature on health human capital and household models, and on related field experiments is reviewed in an attempt to answer these questions. It is found that there are large returns to health improvements in low-income countries. Moreover, health improvements in poor nations can be achieved through implementation of simple interventions such as dietary supplements, control of parasitic diseases, and pro-poor social expenditures. The paper concludes with a discussion of these policy options.

1. INTRODUCTION

Good health is a determinant of economic growth and a component of the well-being of the population. This paper presents and synthesizes economic models that can be used to analyze dominant health policy issues in developing countries and thus provide information that policy makers can use to improve the health of the population. The models may be used to investigate the following sorts of questions.

Who produces health and how? An answer to this question is important in helping policy makers plan for health production, or provide incentives for its production. Knowledge of human biology, epidemiology and household technologies of producing market and non-market goods are important in answering this question (Becker, 1965, 1981; Ben-Porath, 1967; Grossman, 1972a,b). What are the channels through which health inputs affect health, and how can their effects be correctly measured? Models that endogenize health inputs are required in order to answer this question (Rosenzweig and Schultz, 1983). To what extent is demand for health inputs responsive to variables that can be changed by public policy, such as household income and time and money prices? Data from household surveys (Acton, 1975; Gertler et al., 1987) can be used to address these issues.

What are the causal effects of health on labor market outcomes such as wages and labor force participation? Randomized experiments such as provision of micronutrients to workers (e.g., Thomas et al. 2006) may be used to estimate these effects. IV methods can also be used to analyze effects of health on labor market outcomes (Strauss and Thomas, 1998). What determines the distribution of health outcomes and consumption of health care within a household? Collective models of the family are appropriate for addressing this question (see e.g., Alderman et al., 1995; Udry, 1996; Browning and Chiappori, 1998; and Strauss et al., 2000).

What health policies would increase growth and reduce poverty? Macroeconomic models that analyze effects of diseases on growth could be used to identify such policies (see Bloom and Sachs, 1998; Commission on Macroeconomics and Health, 2001). What pattern of industrial organization of health care exists in developing countries? Agency models of physician-patient relationship, monopolistic competition and managed care models (Culyer and Newhouse, 2000) can provide insights into this issue, but their application to date has been limited mainly to high-income countries. What are the demographic and economic implications of HIV/AIDS in low-income countries in light of the unfolding technologies for preventing, testing and treating this disease? (WHO, 2004; Lopez-Casasnovas et al., 2005; Thirumurthy, et al., 2005).

Each of the above issues is important in its own right. For example, a good understanding of the effect of health on labor market outcomes is not only a prerequisite for enhancing labor productivity through better health, but may also be the basis for designing better education and nutrition policies. Together the foregoing issues constitute the core questions to be answered before organizing health systems and programs for promoting adult health and for further reducing infant mortality in low-income countries. Health economics can undoubtedly be used to shed light on other health concerns apart from the issues highlighted above. Nonetheless, the above listing depicts examples of the

issues that economic analysis can help clarify, and thus empower policy makers to design and implement effective health interventions.

The remainder of the paper is structured as follows. An overview of health economics is presented in Section 2, with a special focus on its distinctiveness as a sub-discipline. Section 3 describes the main features of health economics for low-income countries. Section 4 reviews specific economic models and techniques for analyzing some of the health policy issues highlighted in this introduction. Section 5 concludes.

2.   HEALTH ECONOMICS

The issues in Section 1 fit well into standard categories of economic theory. For example, they fall under topics of demand, consumer choice, production technology, supply, markets, industrial organization, economics of information, incentive structure and social welfare. However, the standard economic analysis as conducted under these categories often fails to provide adequate understanding of health and health care phenomena (Culyer and Newhouse, 2000). The special characteristics of health and health care are the sources of this failure. For example, consumer theory cannot successfully be used to analyze health care demand under the usual assumption that income and money prices are the main factors affecting health care decisions because the effects of information and time prices are also quite important.

Health economics is concerned with the formal analysis of costs, benefits, management, and consequences of health and health care. Often, health economics is used synonymously with medical economics, the branch of economics concerned with the application of economic theory to phenomena and problems associated with health and health care (see Sweifel and Breyer, 1997; Mills, 1998; Feldstein, 1999; Jack, 1999). A notable feature of this application is the concern for equity in health outcomes and health care provision. The equity concern in health outcomes arises because health is universally accepted as a merit good, a minimum of which each individual is entitled regardless of ability to pay (WH0, 1978). In health care markets, the equity issue is manifested by widespread public subsidization or direct provision of health care. The case for health subsidies is particularly strong because evidently, an individual needs some minimum amount of health human capital to survive (see Fogel, 1997; Grossman, 2000).

Health economics has progressed rapidly from an infant state in the 1960s to a distinct sub-discipline of economics today. It draws its disciplinary inspiration from the fields of finance, insurance, industrial organization, econometrics, labor economics, public finance and development studies (Culyer and Newhouse, 2000). Arrow's (1963) article gave health economics its present form as a separate field of study, with its parallel development in human capital theory (T.W. Schultz, 1960, 1961; and Becker, 1962, 1964). The field has substantively contributed to mainstream economics in many areas, including human capital theory, the principal agent theory, econometric methods, the methodology of cost-effectiveness analysis, and the theory of supplier-induced demand (Newhouse, 1987; Culyer and Newhouse, 2000).

In the *Handbook of Health Economics*, Culyer and Newhouse (2000) provide a comprehensive discussion of the main components of health economics, originally proposed by Williams (1987). The components include the meaning and scope of health economics; determinants of health; demand for health and health care; supply of health care; health care markets; the relationship between economic growth and health; health sector budgeting and planning; national health systems; equity in health outcomes and in health care; and international health, under which topic, diseases such as HIV/AIDS, and bird flu may be analyzed. Jack (1999) looks at some of these components in the context of low-income countries.

The present paper covers only a few of the topics mentioned above to illustrate the type of models and methods that can be used to analyze the topics in the context of developing countries. The motivation of the paper is to show the type of knowledge that health economics research needs to generate to assist health policy-making in low-income countries. For example, although accumulation of health human capital is a key determinant of economic growth (Barro and Sala-i-Martin, 2004; Lopez-Casasnovas e t al., 2005), little is known about health production technologies and the institutional contexts in which health improvements occur (Fuchs, 2004). This knowledge gap is acute in low-income countries, where health policies are urgently needed to reverse declining health indicators due to disease epidemics of which HIV/AIDS is the leading example (see WHO, 2004).

3.   HEALTH ECONOMICS FOR LOW-INCOME COUNTRIES

A conspicuous fact about the body of knowledge encompassed by health economics is that this knowledge has so far been applied vigorously in developed economies. Indeed, the recent and the only handbook of health economics to date was designed to cover material relevant to service sectors of high-income countries (Culyer and Newhouse, 2000; Aaron, 2001; Grossman, 2004).

As indicated in Section 2, the basic principles of health economics for low-income countries are the same as the core principles of the parent discipline. Thus, health economics for low-income countries may be viewed as an adaptation of health economic principles and methods to institutional conditions of developing and transitional economies. Examples of institutions include (a) formal rules such as regulatory and legal structures, property rights, insurance laws, and constitutions; (b) informal rules such as customs, traditions and social values and beliefs; and (c) social networks and civil society organizations (North, 1990; Williamson, 2000). Despite their ubiquitous nature, institutions are country- and time-specific. Thus, the welfare outcomes of interventions based on the same theory can differ substantially across countries and over time (Oliver et al., 2005). Further, such interventions may not work at all if key institutions are absent or function imperfectly.

Indeed, even in the United States, managed care became a dominant feature of the health sector only after the 1980s, when institutional environment favorable to its development emerged (Newhouse, 1996; Glied, 2000). In particular, after the 1980s health care providers and health insurance companies began to form networks as cooperative mechanisms for cutting costs of health care provision and financing. Participation in these network by insurers was motivated by their desire to

deal with the moral hazard problem (excessive benefit claims by the insured), whereas participation of providers was prompted by a desire to cut costs of care to attract more patients.

Issues of industrial organization of health care, such as the preceding ones, are quite different in low-income countries, where formal health insurance schemes are limited to tiny fractions of the population in urban areas. The moral hazard problem, for example, is of little interest to policy-makers in low-income countries because the bulk of health care is financed through taxation or out of direct payments made by patients. The main health policy issues in such a setting, concern identification of the poor for "free" or subsidized care at government health facilities and design of incentives for motivating doctors to work in public rather than in private clinics. Moreover, because health care markets in low-income countries are much more imperfect than in industrialized countries or are missing altogether, the practice of free or subsidized service provision by the state is quite common. Policy concerns in such contexts often revolve around issues of service access and quality rather than around the moral hazard problem. Indeed, in many rural and slum settings, where the majority of the population in low-income countries reside, time and transport costs of using free health care at public clinics are substantial; so problems of moral hazard and frivolous demand should be rare.

Apart from unique features of the industrial organization of health care in low-income countries, disease burdens in poor countries are quite different from the burdens prevailing in industrialized countries. Therefore, policy-relevant research on issues such as cost-effective health care technologies, health care financing mechanisms, and training requirements for health professionals in low-income countries cannot be the same as the research conducted in industrialized countries on similar issues. It can thus be seen that a focus on health economics for low-income countries provides an opportunity to generate research information that policy-makers there can use directly to improve health or to enhance performance of their health care systems.

Because of country differences in institutions, behavioral parameters such as demand elasticities in high-income countries may not be applicable to low-income countries. For example, although price elasticities of demand from the Rand Health Insurance Experiment in the United States (that average around -.2) "have become the standard in the literature" and are "among the most definitive" (Cutler and Zeckhauser, 2000, p. 584; Contoyannis, et al., 2005, p. 2), they are nonetheless not very informative of how user fees affect health service utilization in developing countries because households there operate under very different institutional contexts. Obviously, price elasticities of demand for health care such as those from the Rand Health Insurance Experiment in the United States (Manning et al. 1987; Cutler and Zeckhauser, 2000) could be used to conduct an exploratory analysis of how high-income households in developing countries would respond to changes in user fees for hospital services. However, since such analysis would apply only to a small section of the population, its policy value would be limited.

Potential applications of Grossman's (1972a,b) theory of demand for health and health care in developing countries is in Schultz (2004). Schultz shows how concepts of health production and demand for behavioral and market inputs can be used to design policies for promoting preventive measures in the fight against HIV/AIDS in developing countries. Studies that demonstrate the economic burdens of malaria (see e.g., Bloom and Sachs, 1998; Commission on Macroeconomics

and Health, 2001) provide the basis for urgency in the design and implementation of policies to fight this deadly disease. The foregoing studies are good examples of health economics for low-income countries because interest in research on diseases such as HIV/AIDS and malaria is strongest in poor countries where these epidemics continue to have a large toll on economies and human lives.

4.   REVIEW OF THE LITERATURE

This section provides a detailed review of analytical frameworks that can be used to analyze the policy issues listed in Section 1.

4.1.   Special characteristics of health and health care

Health

Health is a component of human capital, which in some recent literature is referred to as *health human capital* to distinguish it from *education* human capital (see Schultz, 1999; Lopez-Casasnovas, 2005). This is in contrast to other literatures (see e.g., Mankiw, et al., 1992; Barro and Sala-i-Martin, 1995), where the term human capital is used to mean education.[1]

Human capital is part and parcel of human beings and is not easily measurable (T.W. Schultz, 1961; Mushkin, 1962; Becker, 1964; Lucas, 1988). The World Health Organization's definition of health clearly illustrates the conceptual nature of health, and the implied difficulty involved in measuring it: "health is a state of complete physical and mental well-being and not merely the absence of disease or infirmity" (WHO, 1948).

A further characteristic of health human capital is that it is positively correlated with other forms of human capital. Healthy individuals, for instance, are on average better nourished and better educated than individuals in poor health (Fuchs, 1996, 2004). However, although both health and education increase labor productivity, health has the additional feature that by reducing the time spent in sickness, it increases the total amount of time available to produce money earnings and commodities, as well as the time available for leisure (Grossman, 1972a,b).

As an asset, health is accumulated at the individual- or household level. To paraphrase Grossman (1972a,b; 2000), individuals must use their own time and transportation services to seek health maintenance care. The same idea has been echoed elsewhere: "Health is produced by households not

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[1] Since human capital encompasses more than just education, human capital models that are conditioned on education as the only form of human capital are mis-specified. The term "human capital" has been in use for a long time (Mincer, 1958; Weisbrod, 1961; Kiker, 1969). However, the theory of human capital was shaped into its modern form in the 1960s by T.W. Schultz (1960, 1961) and Becker (1962, 1964).

doctors or hospitals" (Dowie, p. 4). However, this does not deny the importance of hospitals and doctors as inputs into health production, as serious illnesses cannot be effectively treated without these inputs. The quotation emphasizes the role of individuals in choosing hospitals and doctors for treatment or in complying with treatment regimen. Moreover, households and doctors may, and often emphasize different dimensions of health. Many variables are used to summarize health status of households (Strauss and Thomas, 1998), and each captures only some facets of health and ignores others, and they generally measure even these emphasized facets with error and possible bias, adding to the econometrician's problems of estimating the effects of health capital on worker productivity or on consumer benefits.

Health care

The most important difference between health and health care is that health care is tradable in markets while health is not. However, health care markets are highly imperfect. The imperfection arises from the special characteristics of health care. These characteristics were introduced into the health economics literature by Mushkin (1962), Arrow (1963) and Klarman (1963). In the opening part of his paper, Arrow stressed that its subject matter was health care and not health. "It should be noted that the subject is the *medical-care industry*, not *health*" (Arrow, 1963, p. 94; emphasis in the original). The distinction is important because in the real world only markets for health care are observed. Although individuals trade health against other commodities over time (Claxton, et al., 2006), there are no markets in which sellers and buyers can exchange health.[2]

Although individually, the health care attributes discussed below are not unique to health care markets, when "taken together, they establish a special place for health care in economic analysis" (Arrow, 1963, p. 948). Following Arrow (1963), we illustratively discuss these characteristics with respect to a few categories of economic theory, as they relate to health care, namely: demand and supply, uncertainty, information asymmetry, and health care pricing practices.

A.   *The nature of demand for health care*

Health care demand is distinct from the demand for other commodities because illness incidence, the reason for medical care, is irregular and unpredictable. Consumption of health care, particularly preventive care, is often associated with positive externalities. For example, treatment of a patient

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[2]There exists a large variety of health care services that are not tradable. For example, physical exercise, which is an important input into health production is not tradable. Although facilities for exercise are tradable, the exercise itself is not. More generally, health inputs that are related to behavioral change are not tradable.

with a infectious illness does not only benefit the person treated, but also other persons because they are protected from exposure to infection. Similarly, immunization of an individual against a communicable disease protects other people from the disease. An individual therefore would typically understate the full value of such forms of health care, which is one reason for its subsidization in virtually all countries. In low-income countries, health care is typically provided by the state free of charge or at nominal cost, whereas in industrialized countries it is paid for through insurance.

B.   *Expected Behavior of Health Care Providers*

An intrinsic technological attribute of medical care is that it belongs to the category of commodities for which the product (treatment) and the activity of production are identical (Arrow, 1963). Thus, unlike other commodities, the quality of medical care cannot be assessed before consumption. Consequently, in providing care, a physician is expected to act in the best interest of the patient, rather than in his own financial interest. This agency relationship creates the opportunity for health care providers to induce health care demand (Fuchs, 2004). The relationship is conducive to observed inefficiencies in health care provision and consumption (e.g., unnecessary medical tests, injections, surgeries and drug prescriptions).

C.   *Product Uncertainty and Information Asymmetry*

The quality or outcome of medical care is uncertain to patients as well as to providers. Neither the patient nor the provider can predict the outcome of treatment with certainty partly due to unobservable aspects of treatment and partly because the treatment itself can have unpredictable adverse effects. This information uncertainty, as to treatment quality, is asymmetrically distributed. The asymmetry is double-sided in the sense that both patients and providers have advantages and disadvantages in observing some aspects of inputs into health production (Leonard, 2003; Leonard and Zivin, 2005). Specifically, providers are well informed about their own treatment effort, but about which patients are ignorant. Similarly, patients know their treatment compliance effort, of which providers are ill-informed. Because the patient is uninformed about treatment, the provider must decide for him the quantity of medical care to buy. Contrary to other market transactions, the buyer here does not entirely decide the quantity demanded.

D.   *Supply Conditions and Pricing Practices*

Medical care is too complicated to be fully understood by patients or persons without medical training. Therefore, there is the risk that patients will buy or receive ineffective or harmful treatment. Society deals with this risk by restricting entry into the medical profession through the licensing of care givers. In contrast to other markets, where licensing is partly meant to raise government revenue, the purpose of licensing in medical care markets is almost exclusively to ensure that patients receive good quality care. However, licensing also restricts competition. Thus, the standard competitive model is not a suitable tool for analyzing health care markets. McGuire (1983, 2000) presents monopolistic models that may be appropriate for this purpose.
An important characteristic of health care is that it is not retradable (McGuire, 2000), which makes it an ideal target for price discrimination (Kessel, 1958).

4.2. **Health Production Functions and Health Input Demand Functions**

Health is an individual-specific phenomenon. Individuals accumulate this asset in the context of a family or household. While issues related to family (Becker, 1973; 1981) are not considered here, issues related to household-level decision making are analyzed in Section 4.3. The present section describes health production by individuals using a structural model due to Rosenzweig and Schultz (1983). In the model, individuals choose health inputs. The fact that health inputs are choice variables introduces heterogeneity in the health of individuals. The heterogeneity arises from unobserved preferences and health endowments of individuals that influence their choice of health inputs. The model also recognizes heterogeneity in health status arising from unobservable influences of biological processes on health production technology. The heterogeneity of health human capital in the population is due to both unobserved behavior and technology. A procedure for dealing with these sources of heterogeneity (biology, preferences, and technology) when empirically analyzing health accumulation is one of the novelties of the Rosenzweig-Schultz model.

The model is chosen for exposition because of its generality. It incorporates key insights from models of household production and consumption (Becker, 1965), health production (Ben-Porath, 1967; Auster et al., 1969), and models of demand for health and health care (Grossman, 1972a,b and Acton, 1975). It further illustrates procedures for consistent estimation of parameters of any economic model when the behavior of agents is conditioned on unobserved variables.

Although some of the studies reviewed in this section were completed over two to four decades ago, they form the basis for modern research in health production and demand for health and health care, and provide the necessary concepts and techniques for further developments in the field. Some of these concepts and methods have not been incorporated into the current health economics literature, especially as it relates to developing countries (see Grossman, 2004). It is hoped that the review will help rectify this situation.

A. *Health Production and Input Demand Functions*

Following Rosenzweig and Schultz (1983), and adopting their notation, we assume that a household's preference ordering over health, H, n X-goods, and m-n Y-goods that affect health can be characterized by a utility function of the form

$$U = U(X_i, Y_j, H), \; i = 1,..., n; j = n +1, ..., m \tag{1}$$

Let the production of health by a household be described by the function

$$H = \Gamma(Y_j, I_k, \mu), \; k = m + 1, ..., r \tag{2}$$

where, the r - m I_k are health inputs which do not enter the utility function except through their effects on H (e.g, health care); Y_j is a subset of Y (e.g., smoking or physical exercise) that both affects health and contributes to utility directly; X_i is good i (e.g., clothing) that contributes only to utility; and μ represents unobservable household-specific health endowments known to the

household but not controlled by it, e.g., genetic traits of its members or environmental factors. It should be noted that both Y_j, and I_k in (2) are subsets of Y, i.e., all the inputs that enter health production function.

The budget constraint for the household in terms of the r purchased goods is

$$F = \sum_t Z_{ti} p_t, \quad t = 1, ..., r \tag{3}$$

where F is the exogenous money income, the p_t are exogenous prices, and Z_t is a vector of all purchased goods, i.e., all the subsets of X, Y, and I that are obtained from the market. Needless to say equation (3) can easily be modified to incorporate non-market goods with a full income constraint.

The household's reduced-form demand functions for r goods, including the r-n health inputs, derived from the maximization of equation (1) subject to equations (2) and (3), are

$$Z_t = S_t(p, F, \mu) \quad t = 1, ..., r \tag{4}$$

Similarly, the reduced-form demand function for health outcome may be expressed as

$$H = \psi(p, F, \mu) \tag{5}$$

Notice that in equations (4) and (5), the subscript t for p is suppressed because consistent with demand theory, the entire set of prices enters the demand function for each and every good. Since demand equations (4) and (5) contain unobserved health production technology, this fact should be taken into account when estimating the equations.

Yet empirical investigations of health care and health production have concentrated mainly on estimating health input functions such as (4) or health human capital demand equations such as (5). See Acton (1975) and Deri (2005) for an implementation of (4) and (5) in developed economy contexts. There is a large literature on health care demand from developing countries that is consistent with (4), but examples consistent with (5) are rare (Leonard 2003). See for example Akin et al.(1986), Akin et al. (1998), Buldoc et al. (1996), Gertler et al. (1987), Heller (1982), Lindelow (2005), Mwabu (1989), Sahn et al.(2003), Sauerbon et al. (1994), and Schneider and Hanson (2006) for applications of various version of equation (4) in developing country contexts.

Although these models are useful in providing policy relevant parameters for health care demand and for its prediction, they fail to take policy making to the next stage of connecting causally the usage of health inputs to health capital production. In developing countries where health status is low, and is in dire need of improvement, this is a major failing of the models.

B.   *Hybrid health production and health demand functions*

Motivation

9

In an attempt to causally connect usage of health inputs to changes in health status, a hybrid health production model that combines equations (2), (4) and (5) is often estimated (Rosenzweig and Schultz,1983). The hybrid form of the theoretical model is

$$H = \theta\ (Y_m, p_l, F, \mu),\ l = 1,...,\ m\text{-}1,\ m +1,\ ...,\ r \tag{6}$$

where H, Y, F and μ are defined as before, i.e., H is health status, Y_m is a health input of type m such as medical care, F is exogenous income, μ is unobservable health endowment specific to each household, while p_l is a vector of prices of type m health inputs. In contrast to (2), Y in (6) incorporates health inputs of all types. Moreover, the health input, Y_m, is endogenous because it depends on health status, H, the initial health status before Y_m is demanded. Thus, in estimating this equation the endogeneity of Y_m and the unobservability of μ should be taken into account. Several other key features of equation (6) are worth emphasizing.

First, equation (6) may be interpreted as a form of demand for *health* (Engel curve for health) since it is conditioned on exogenous income, F, with other covariates in the function being treated as shift factors. If the price of health were available in (6), the expression can be viewed as a Marshallian demand for health. However, as shown in Section 4.2, the shadow price of health is endogenous, an issue that would need to be considered when estimating the demand for health.

Second, equation (6) may be interpreted as a form of a health production function, with F being treated as a proxy for health inputs other than medical care, and with prices of health inputs, p_l, serving as background variables. Explicit specification of input prices in equation (6) allows it to be interpreted as a meta production function as in Evenson (2001) and Bindlish and Evenson (1993, 1997). A meta production function is an envelope of output response curves, each curve representing different degrees of changes in output due to an input response to price variation (see Hayami, 1969, p. 1298). The concept is useful here because it helps focus attention on changes in health input prices in an analysis of health production and health input demands, thereby showing the extent to which input prices can be used as instruments for improving health.

Furthermore, looking at equations (1) and (6) together, it can be seen that even if the health input Y_m is a source of disutility, such as tooth extraction (see equation 1), it may still be demanded because it enhances health (equation 6). In the same vein, even if a health-related input such as smoking is harmful to health, it may still be consumed as long as its marginal utility is positive. It is evident from equation (6) that to evaluate the health effects of input prices, information is needed on marginal products of health inputs whose prices have changed.

Third, equation (6) states that an individual's health status is influenced by a total of r purchased health inputs, of which m are of medical care variety, and $r\text{-}m$ belong to other types, such as food, shelter, and *behavioral* health production inputs such as smoking, recreation and alcohol consumption. These types of inputs may be extended to include non-market behavioral inputs such as the timing of marriage or the first birth. The distinction between derived demand for "behavioral health inputs" and derived demand for "commodity or market health inputs" is important because it

helps separate out policies that promote desired behavioral changes in the production of health from those that alter consumption levels of health-enhancing commodities. The distinction greatly widens the scope of the concept of demand in the analysis of policies that individuals and society may use to enhance health. Treatment of health-improving behaviors as derived non-market demands, helps stress the fact that these behaviors are choices that households make and that policy can be used to modify them.

Fourth, equation (6) states that the price vector, p_l, comprises l prices, one for each of the m-1 inputs of type m, with the price of the mth input being omitted because it is a numeraire. Further, the prices of r-m inputs are omitted because, for simplicity, only the health effects of m inputs are being considered. Fifth, it further states that health status depends on medical care, the exogenous prices of other health inputs (i.e, m-1), exogenous income, and unobservable household-specific health endowment term, μ.

Sixth, the conditioning variable of direct interest, Y_m, represents either a continuous choice or a discrete choice demand for medical care. If demand is discrete, Y_m, represents consumption of medical care by households or individuals, with the estimated coefficients being interpreted as responses of demand to its determinants at the extensive margin. In the event of continuous choice, Y_m represents the intensity of health care usage, conditional on up-take, so that the estimated parameters are demand responses at the intensive margin. Rosenzweig and Schultz (1983) illustrate the shortcomings of the hybrid model under the assumption that Y_m is a continuous variable. Dow (1999), Gertler and van der Gaag (1990) are notable examples of discrete choice models of medical care demand in developing countries while Jones (2000) contains a summary of the literature in this area.

Since medical care is a choice variable, the health effect of medical care in equation (6) is biased if estimated by ordinary least squares. Further, even if medical care is randomly provided to a particular region, the OLS estimate of its coefficient would still be biased due to unobervability of μ, apart from any omitted variables bias. Rosenzweig and Schultz (1983, p. 728) show analytically that the sign and size of the overall bias here depends on (i) the properties of the utility function, (ii) the marginal products of all health inputs, (iii) how μ affects health directly, and (iv) how μ affects the marginal products of the controllable health inputs. A statistical discussion of these issues is taken up after a further elaboration of equation (6).

 Demand for health

Equation (6) is consistent with Grossman's (1972a,b) formulation of the demand for health human capital. In his pure investment model, H is a factor of production, whereas in the pure consumption model, it is a consumer good; in his general model, H is simultaneously a producer as well as a consumer good.

Two desirable properties of Grossman's model are not captured in equations (4)-(6). First, the shadow price of health (which is nontradable), is not explicitly shown in any of the equations. Second, the overall benefit of health capital (both as a producer and as a consumer good) is not

11

derived. These contributions can be illustrated with a one period version of the utility function in equation (1), expressed in Grossman's (1972a,b) notation. The utility is maximized subject to the household income and production technology.

Max U = U(φᵢHᵢ, Zᵢ) (7a)

subject to

$P_iM_i + V_iX_i + W_i(TL_i + TH_i + T_i) = W_i \Omega + A_i = R$ (7b)
$I_i = I_i(M_i, TH_i; E_i)$ (7c)
$I_i = (H_i - H_0) + \delta H_i$
$Z_i = Z(X_i, T_i; E_i)$ (7d)

where
H_i is the stock of health in period i (time period i = 1 for all variables);
φ_i is the flow of services per unit of health stock so that $h_i = \varphi_i H_i$ is the total quantity of health services available for consumption in period i, measured in this case by the number of healthy days;
H_0 is inherited stock of health capital, and δ_i is its depreciation rate in period i;
P_i and V_i are prices of medical care (M_i) and other goods (X_i) respectively;
W_i is the wage rate in the labor market;
I_i is gross investment in health;
Z_i is an aggregate of all commodities besides health;
TH_i and T_i are time inputs associated with the production of I_i and Z_i;
TL_i is the time lost from market and non-market activities due to illness;
E_i is level of education;
A_i is non-labor income;
Ω is total the amount of time available in any period;
R is full income, the monetary value of assets plus the earnings an individual would obtain if he spent all of his time working.

Equation (7b) is the full household income constraint, where

$\Omega = TW_i + TL_i + TH_i + T_i,$

where

TW_i is hours of work. The inclusion of TL_i in (7b) modifies Becker's (1965) time budget constraint, so that it can fully exhaust the total time available in any period (Grossman, 1972a,b). Part of the 'full income,' R, a concept coined by Becker (1965), is spent on market goods, part of it is spent on nonmarket production, and the remaining part is lost due to illness.

Equations (7c &d) are production functions for health and a composite non-health commodity respectively.

The Lagrangian Function to be maximized is

$$\mathcal{L} = U(\varphi_i H_i, Z_i) + \lambda(R - (C_i + C_{1i} + W_i\, TL_i)) \tag{8}$$

where
$C_i = P_i M_i + W_i TH_i$ (Total cost of medical care)
$C_{1i} = V_i X_i + W_i T_i$ (Cost of non-medical commodities)
$H_i - H_0 = I_i$ (Gross investment in health, with its arguments M and TH suppressed) and with depreciation rate δ_i set equal to zero.

The optimal quantity of health capital demanded, H, is found by differentiating equation (8) with respect to gross investment (I) and setting the partial derivatives equal to zero to obtain the first order condition, which after manipulation yields the equilibrium condition

$$U_{hi} \bullet (\partial h_i / \partial H_i) \bullet (\partial H_i / \partial I_i) = \lambda\{(dC_i/dI_i) + W_i(\partial TL_i/\partial H_i) \bullet (\partial H/\partial I_i)\} \tag{9A}$$

where,
(a) $U_{hi} = \partial U/\partial h_i$ (is the marginal utility of healthy days, h_i);

(b $\partial h_i /\partial H_i = G_i$ (a unit increase in health capital, H_i, increases the number of healthy days, h_i, by G_i, which is the marginal product of health capital in the production of healthy days);
(c) $\partial H_i/\partial I_i = 1$ (a unit increase in gross investment raises health human capital, H_i, by 1 unit);
(d) λ (is the marginal utility of income, since from (8), $\partial \mathcal{L}/\partial R = \lambda$);
(e) $dC_i/dI_i = \pi_i$ (is the marginal cost of gross investment, the *shadow* price of health, which depends on opportunity costs of purchased health inputs, M, and non-purchased inputs, TH);

(f) $\partial TL_i/\partial H_i = -G_i$ (reduction in the number of healthy days lost to illness arising from a unit increase in health capital, H, which is the reverse of (b) above).

Expression (9A), where the effect of health on wages or income is ignored, can now be rewritten as

$$U_{hi} \bullet G_i = \lambda(\pi_i - W_i \bullet G_i) \tag{9B}$$

The left-hand side of (9B) is the marginal benefit of investing in an extra unit of health human capital, expressed in utility terms while the right-hand side is the associated marginal cost, also stated in utility terms. Lambda (λ), the marginal utility of income, converts the monetary cost ($\pi_i - W_i \bullet G_i$) on the right-hand side of (9B) into a utility magnitude. Thus, the net marginal cost of producing a unit of health capital is ($\lambda\pi_i - \lambda_i W_i \bullet G_i$). In equilibrium, the marginal benefit, $U_{hi} \bullet G_i$, must be equal to the net marginal cost, ($\lambda\pi_i - \lambda_i W_i \bullet G_i$).

Dividing both sides of (9B) by lambda (λ) to convert utilities into monetary magnitudes and rearranging the expression slightly, one obtains

$$\pi_i = W_i \bullet G_i + (U_{hi} \bullet G_i) \bullet (1/\lambda)$$

or
$$\pi_i = G_i (W_i + U_{hi} \cdot (1/\lambda)) \tag{9C}$$

where
π_i = marginal cost of producing a unit of health capital, in monetary terms;
$W_i \cdot G_i$ = the value of the marginal product of health capital (extra healthy time resulting from successful treatment of an illness);
$(U_{hi} \cdot G_i) \cdot (1/\lambda)$ = the monetary equivalent of an increase in utility due to a unit increase in health capital (i.e., a unit increase in healthy time).

Equation (9C) shows that at equilibrium, the *shadow* price of health is equal to its marginal benefit, which in this non-separable household production model, consists of two distinct parts. One part $(W_i \cdot G_i)$ reflects the monetary value of the marginal product of an extra unit of 'healthy time' in market and non-market activities, while the other part $(U_{hi} \cdot G_i) \cdot (1/\lambda)$ represents the monetary equivalent of the additional utility derived from that unit.[3]

Expression (9C) provides two insights into health human capital. The first insight is that health is jointly valued as a producer and as a consumer good, but these two distinct elements of the benefits are impossible to disentangle because the utility element is not observed. The only way to separate out these benefits is to arbitrarily assume that health human capital is either a producer or a consumer good. In that case, the equilibrium shadow price of health would be equal to one or the other of the two benefit magnitudes in (9C). The second insight from (9C) is that the shadow price of health human capital encompasses more than just the price of medical care. As can be seen from (8), the shadow price of health depends on the price of medical care and on the wage rate.

The wage rates and the medical care prices are determined in labor markets and in medical care markets respectively. An individual takes these prices as given in making health care decisions. However, even when the wage rates and medical care prices are exogenously given to an individual, the shadow price of health human capital is endogenous, because this price depends on health, which is a choice variable in the health production function. Once the shadow price of health is given, the demand for health follows directly from the theory of consumer behavior, but this price should not be taken as exogenous when estimating a model of demand for health.

A key point that is worth repeating is that health is not traded in the marketplace. Thus, at a shadow price, of say π_i, one must produce and supply health capital to one self. If π_i is constant, the

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[3] Equations (8-9) are derived under the assumption that a unit of health human capital lasts for one period only and does not depreciate. It is straightforward to introduce concepts of durable health capital, depreciation, intertemporal utility and interest rate, r, into the analysis (see Grossman, 1972a, pp. 228-230).

individual is willing to supply to self an infinite amount of health. At that price, the quantity he "buys" depends on his health demand schedule. If the demand schedule is downward sloping, a finite optimal quantity of health capital would be demanded. If the demand curve were flat, no such quantity would exist.

In Grossman's health demand model, health is endogenous, in the uncontroversial sense that individuals choose the optimal amount of it that they need so as to produce "healthy days." In other words, health status is governed by health investment and consumption activities of individuals. This is a major contribution of the model to policy making because it links health status to health maintenance activities of households and society.

However, unnecessary controversy has arisen in the literature over this feature of the model (Grossman 1998, 2000) because the optimal quantity of health capital is associated with a particular length of life or time of death. The controversy is rooted in Grossman's (1972a,b) assessment of his own model. "Death is said to occur when the stock falls below a certain level, and one of the novel features of the model is that individuals "choose" their length of life" (Grossman 1972a, p.225; 1972b, p. 1). In Grossman's model, individuals choose the optimal number of healthy days, rather than the length of life itself. The number of healthy days, can be zero without death occurring. Indeed, people can live for years in a constant state of illness. Furthermore, at the individual level, illness is a random event, which cannot be anticipated or insured against. Thus, the health capital accumulated by successful treatment of an illness in a previous period can be wiped out by a random illness in the next period, so that death can occur during a period of good health. However, this need not be so if in reducing mortality, health interventions are complementary across time periods. Specifically, if inter-temporal complementarity exists, an intervention undertaken against a specific risk to health in a prior period would reduce another risk in a future period (Dow et al., 1999).

In Grossman's model, three outputs are produced sequentially. The first output is health capital, which is produced using two inputs, namely, medical care and personal time. The accumulated health capital is then used to produce the second output, namely, healthy days. The healthy days are finally used to produce market and nonmarket commodities.

The second production activity above seems unnecessary (Muurinen 1982). Once accumulated, health capital can be used directly as a producer or a consumer good. Healthy days are simply units for measuring health. Alternative measures of health at the individual level include height, weight, body mass index, and indicators of activities of daily living. It should also be noted that in determining the optimal length of life, it is the marginal utility of an extra day of life (not of an extra healthy day) that is relevant.[4]

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[4] See Cropper (1977) and Ehrlich and Chuma (1998). The time of death in these models is exogenous due to unpredictability of illness. Even after receiving medical treatment, recovery is uncertain. "Recovery from illness is as unpredictable as is its incidence" (Arrow, 1963, p. 951). Although an individual contributes to his longevity at each age, the actual length of life he lives is outside his control.

The above are but minor criticisms of Grossman's model. The model's result that people contribute to their longevity through health maintenance activities is quite intuitive. It is also intuitive that the additions that people make to their longevity are limited by resource availability and by the technology available to produce health.

A serious shortcoming of Grossman's model is that estimation of both the health production function and demand functions relies on restrictive functional forms. Grossman's identification strategy (Grossman 1972b) that uses per capita income as an instrument for medical care can be improved. The next section discusses better methods for estimating health production and health input demand functions.

C.   *Functional Forms for Health Production*

<u>Specification Issues</u>

Ideally, the health effects of input demands depicted in equations (6) and (7c) should be estimated using a functional form that imposes a minimum of restrictions on the way the inputs are combined to produce health. The transcendental logarithmic health production technology has certain attractive properties, one of which is a second-order approximation in log form, to any production technology. The translog health production function may be stated as in equation (10); for derivations and details see Diewert (1971) and Fuss and McFadden (1978).

$$ln(H) = \gamma + \tfrac{1}{2}\,\Sigma_i\Sigma_j\beta_{ij}\bullet ln(Y_i)ln(Y_j) + \Sigma_i\beta_i\bullet ln(Y_i) + \delta\bullet Z + \mu + \varepsilon \quad (10)$$

where,
ln is a natural log operator; γ, δ, β_i, and β_{ij} are technological parameters to be estimated; Y_i is health input i, with $i \neq j$; Z is a vector of other control variables such as socioeconomic characteristics of households; ε is a random error term. As before, the term, μ, represents unobservable household-specific health endowments known to the household but not controlled by it, e.g., genetic traits of its members.

The constant term, ½, in the translog specification of the health production function comes from the local second-order approximation to any production function using the Taylor series expansion.[5]

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[5] The following generalized linear health production function can also be specified as $H = \Sigma_i\Sigma_j\alpha_{ij}\bullet(Y_i)^{1/2}\bullet(Y_j)^{1/2}$, where α_{ij} are the health effects of the interaction terms (see Diewert, 1971, p. 505). In the case of four health inputs, the model to be estimated would have sixteen interaction coefficients (including the parameters on squared terms). However, symmetry restrictions, i.e., $\alpha_{ij} = \alpha_{ji}$, considerably reduce the number of the parameters that are actually estimated. As in the case of the translog function, the generalized linear and Leontief specifications are very useful in providing second-order approximations to an arbitrary function at a given vector of covariates using only a minimal number of parameters, i.e., the coefficients of the interaction terms (Diewert, 1971, pp. 497-506; Rosenzweig and Schultz, 1982, pp. 67-81). Examples of other functional forms for continuous choice health care demands are in Deaton (1997) and Hunt-McCool et al. (1994), while examples for discrete choice demands are in Dow (1999) and Sahn et al (2003).
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That is, *any* function of unknown form is approximated by a second degree polynomial function (see Diewert, 1971).

Since μ is unobserved, the composite error term in equation (10) is the sum of ε and μ, which is likely to be correlated with Y_is so that the OLS estimates of β_i and β_{ij} parameters may be inconsistent, a situation that calls for the testing of exogeneity of Y_is.

The translog production function has two desirable properties. First, it is flexible in the sense that it allows the data to help determine the correct mathematical form of the production technology to a second-order approximation. If for example, the estimated β_{ij}s are equal to zero, equation (7) takes the form of a Cobb-Douglas production technology. Second, the symmetry restriction, $\beta_{ij} = \beta_{ji}$, can be used to reduce the number of β_{ij} parameters to be estimated, thus conserving the degrees of freedom. The generalized Leontief-Diewert specification has the same advantages as the translog function (see Diewert, 1971, p. 505).

Estimation Strategies and Some Results

Because of the problems of endogeneity of health inputs and heterogeneity of patients, the OLS parameter estimates in equation (10) are biased. If longitudinal data are available, fixed and random effects methods can be used to solve the time invariant heterogeneity problem. However, since the health inputs are chosen by households, the endogeneity problem would still remain. Thus, IV estimation methods should be used irrespective of whether or not the available data are longitudinal or cross-sectional.

The challenge in using the IV methods is to find valid instruments for the endogenous health inputs. In general, local commodity prices and measures of community level infrastructure can be used as instruments for health inputs (Strauss, 1986; Strauss and Thomas, 1995; Wooldridge, 2002). Instruments for medical care in equation (10) would include user fees at local clinics, distances and travel time to clinics, prices of staple foods, alcohol and cigarettes, distances to market centers, and to social infrastructure such as roads, schools and clinics. These factors are assumed to influence the demand for medical care, while exerting no independent effect on health.

Once valid instruments for the endogenous health inputs are available, IV parameter estimates of a health production function can be estimated consistently. Rosenzweig and Schultz (1983) illustrate with data from the United States how to deal with the problem of endogeneity of health inputs in the estimation of a health production function, when birth weight is used as a measure of health status of newborns. Using the 2SLS method, they showed large child-health effects of behavioral inputs, such as the timing of prenatal care, smoking, and mother's age at first birth. In particular, educated women and women from high-income families sought prenatal care earlier, but women from low socioeconomic background postponed such care. A mother's delay in seeking prenatal care reduced both the birth weight and the gestation period. A delay of six months in seeking prenatal care lowered birth weight by 45 grams and reduced gestation period by 1.6 weeks. Availability of family planning programs did not always affect the timing of births. In contrast to these findings, biased OLS estimates showed that delay in receiving prenatal care had no effect on the weight of the new born, confirming that estimation method matters.

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The econometric methodology proposed by Rosenzweig and Schultz (1983) can be used to generate information that policy-makers need to design and implement programs for increasing utilization of prenatal and immunization services by mothers. Effects of prenatal care programs on child health (as measured for example, by birth weight or survival probabilities at birth) can be quantified using this methodology and thus be the basis for rationalizing resource allocation in the fight against childhood diseases. This is a substantial contribution of the model because mortality and morbidity of children are some of the most pressing health problems in low-income countries. However, the severe data requirements that must be met to properly estimate health production functions must be noted. First, many household surveys do not collect information on community-level variables such as prices and social infrastructure so that instruments for endogenous health inputs like medical care and immunizations are very difficult to find. Second, even when community-level information is available, one is often restricted to using cross-section data because few national statistical offices in developing countries (the main sources of household surveys) rarely collect panel data. On the same vein, the national statistical offices take time to release data to researchers so that estimation of health production functions on current data is often infeasible. Moreover, substantial effort is required to clean survey data sets and to ensure that they are internally consistent.

4.3.   **Household Production, Consumption and Health**

Background

The household provides the environment in which individuals produce and consume health and other commodities. Becker (1965, p. 496) described this environment as follows; "a household is truly a 'small factory': it combines capital goods, raw materials, and labor to clean, feed, procreate and otherwise produce useful commodities." Becker's comparison of a household with a factory is not exact, however. As he is fully aware, there is more to a household than its production role. Another key role of the household is the distribution of intermediate and final goods such as leisure time, health care services, food and clothing among its members. This role is important because it determines the health and other welfare dimensions of each household member.

In Becker's model -- the unitary model, the intra-household distribution of commodities occurs automatically. The model predicts that in a household with a caring head, all members are motivated to pursue a common goal. Becker (1974, p. 1080) stresses this point (his Rotten Kid Theorem), as follows: "Put still differently, sufficient 'love' by one member guarantees that all members act as if they loved other members as much as themselves. As it were, the amount of 'love' required in a family is economized: sufficient 'love' by one member leads all other members by 'an invisible hand' to act as if they too loved everyone."

In this idealized setting, even a selfish family member would willingly make transfers to other members, to avoid the risk of being sanctioned by the head, the only person in the family with sufficient love and resources. In this model, the Rotten Kid Theorem ensures that the household head

would never actually have to intervene to enforce intra-household allocations. Any intra-household transfers are voluntarily done (see Bergstrom, 1989, pp. 1139-1140).

Becker's model, predicts that household members pool their resources and that the welfare of members is unaffected by the identity of the person controlling resource allocation.[6] Since these predictions hardly find support in the data (Strauss and Thomas, 1995), intra-household distributional issues should receive priority in policies aimed at improving health, especially the health of the vulnerable household members such as women and children (Dercon and Krishnan, 2000).

Intra-household distribution issues in the area of human capital are complicated. For example, in contrast to the usual case of income redistribution within a household, where income transfers are made from one person to another, health human capital itself cannot be so redistributed. Once human capital has been accumulated by an individual, part of it cannot be transferred to another. However, some categories of health inputs, such as medical care expenditures, nutrients and insecticide-impregnated bed-nets can be redistributed, but behavioral health inputs such as dietary habits, personal hygiene and sexual practices cannot be reallocated from one individual to another.

The non-transferability of health from one person to another, including an important set of its determinants (behavioral health inputs), poses a serious problem to the unitary model as a framework for understanding health effects of public policies within households. This problem arises because of missing markets. There are no markets for health or for behavioral health inputs, both of which are self-produced. For example, an individual learns methods of personal hygiene and uses them to prevent illnesses, i.e., to produce health. The individual in this context cannot use an income transfer from the caring head, or from another family member to buy personal hygiene, but can use the income to purchase inputs that help produce personal hygiene. Other things equal, intra-household income transfers would not improve health if key inputs to health production are self-produced. Briefly, it is virtually impossible to predict a priori how health of individuals within a

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[6] Alderman, et al. (1995) call the Beckerian model of the household "unitary" because all household members are assumed to act as one person, all pursuing a common goal, to distinguish it from "collective" models, where individuals who constitute a household act separately. In their model labeling, Alderman et al (1995) follow the terminology in Chiappori (1988), Bourguignon and Chiappori (1992) and Browning et al. (1994). The term "collective" in these models stresses the analysis of many individualistic behaviors of members of a group (a household), in contrast to one-person model under the same setting. Recent summaries and discussion of the large literature on household models is in Strauss et al. (2000), and Vermeulen (2002). Earlier formulations and analyses of these models are in Udry (1996), Behrman (1997) and Bergstrom (1997), while a synthesis of agricultural household models is in Singh et al. (1986).

household will be affected by re-distributive policies, without making strong assumptions about markets for health and heath care, as well as about health production technologies. Because there are no markets for health and for key health inputs, separation of health consumption and production processes is not possible, a situation that fully undermines the predictive power of reduced-form health demand functions. For precise formulation of this argument see (Pitt and Rosenzweig, 1986).

The Collective Model

The collective model (Alderman, et al., 1995; Haddad et al., 1997) is appropriate for analyzing health capital formation within a household because it can address distributional issues related to both tradable and non-tradable commodities. The various versions of the collective model that exist in the literature share one feature: they all attempt to explain intra-household allocations.

Following Thomas (2000), the welfare maximization problem of the household can be expressed as

$$\text{Max } W = \sum_m \tau_m U_m (L, X; A, e), \quad m = 1, ..., N \tag{14A}$$

Subject to

$$pX = \sum_m (w_m(T_m - L_m) + Y_m) \tag{14B}$$

where

W = Welfare derived from total household consumption;
$\tau_m U_m (.)$ is an individual utility function, weighted by τ_m, the index of bargaining power of individual m in the allocation of consumption within the household;
p is a vector of commodity prices;
w is a vector of wages for all household members;
T is total household time;
Y is total non-labor income;
X is a vector of goods consumed by all household members, including *medical care* so that commodity expenditure, pX is equal to total labor and non-labor income;
L is a vector of leisure time for all household members;
A is a vector of household-level demographics such as household size, composition, and personal characteristics such as age, gender, health status and education of all household members;[7]
e is a vector of unobserved heterogeneity in the household, such as attitudes and ability of members or more generally, efficiency parameters (Manser and Brown, 1980, p. 35).

Notice that since $\sum_m \tau_m$ is equal to 1, if the bargaining weights for all m except m-1 are equal to zero, consumption allocation within the household is determined by one person, and expression (14A)

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[7] Some of the variables here, such as household size and composition, and health status are endogenous and would require instrumentation to properly estimate the model.

simplifies to a unitary model.[8] The bargaining power index, τ, is variously known in the literature as a Pareto weight (Dercon and Krishnan, 2000; Browning et al., 2004; Duflo and Udry, 2004) and as a distribution of power index (Browning and Chiappori, 1998). The Pareto weight indicates the influence that individual m has on household market demands. It is a welfare weight, meaning that it shows the importance attached to each individual's utility when aggregating the utilities of household members.

The Pareto weight is conceptually different from the resource sharing rule (Browning et al., 2004). The sharing rule is a mechanism for *decentralizing* decision making within a household, while the Pareto weight is a utility *aggregating* device. The sharing rule specifies the share of individual m in total household income. The rule depends on individuals' options outside the household and on demographic characteristics or the preference shifters (Browning et al., 2004). Although preference shifters influence the Pareto weight, they enter it through the budget constraint. The Pareto weight (bargaining power index), τ_m, in equation (14A) amounts to the share of individual m in total household welfare. The Pareto weight and the sharing rule can be expressed as (a) $\tau_m = \tau_m(\rho_m(\theta); y, p, w)$ and (b) $\rho_m = \rho_m(\theta; y, p, w, d)$, respectively, where d is a vector of demographics and θ is a vector of extra-household environmental parameters, including any alimony rights mandated by government (Rangel, 2006). As just noted, the demographics, d, enter the Pareto weight, τ, through the sharing rule, ρ.

The key feature of the extra-household environmental variables, θ, e.g., non-earned income, assets brought into the family through marriage, sex ratios within the village, and family law is that they influence the decision making process within the household (by altering the resource sharing rule, ρ) but do not affect an individual's preference over commodities and are thus exogenous to demand functions. As expected, the sharing rule is also affected by total household expenditure, y, the commodity prices p, the wage rates, w and demographics, d. Browing et al. (2004) provide examples of general functional forms for Pareto weight, and the corresponding forms for the sharing rule. Although Browning et al. indicate that in empirical applications, interest is usually in the sharing rule, Dercon and Krishnan (2000) focus on Pareto weights in their Ethiopian study.

Subsuming e in τ and solving equation (14A) yields a set of commodity and leisure demand functions so that the demand for commodity g (such as medical care) by an individual m in a household can be expressed as

$$X_g = g(p, w, y; A, \tau(\rho(\theta))) \qquad (15A)$$

The term $\tau(\rho(\theta))$ in equation (15A) captures both the rule for intra-household allocations, ρ, and the weight, τ, for utility aggregation within a household. As expected, the resource sharing rule influences intra-household distribution of commodities. For example, holding total household

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[8] Notice that equation (14A) is a generalization of the following two-person expression
$$\text{Max } W = (1-\tau)[U_m(L, X; A, e)] + \tau[U_m(L, X; A, e)], \quad m = 1, 2.$$

income constant, modification of the rule governing intra-household allocation of commodity g (medical care) to m, will change the quantity of g demanded by m as well as m's Pareto weight.

Thomas (2000) discusses how expression (15A) can be used to test the assumptions underlying unitary and collective models, e.g., income pooling and Pareto efficiency. Since the Pareto weight in equation (15A) is unobservable, and in any case, since its magnitude can be inferred once the sharing rule is determined, it can be suppressed for convenience of notation. Accordingly, in a two-person-two commodity case (see Quisumbing and Maluccio, 2003), the test for income pooling and for Pareto efficiency can be expressed as

$$(\partial X_1/\partial\theta_h)/(\partial X_1/\partial\theta_w) = (\partial\rho_h/\partial\theta_h)/(\partial\rho_w/\partial\theta_w) = (\partial X_2/\partial\theta_h)/(\partial X_2/\partial\theta_w) \tag{15B}$$

where

$$\partial X_g/\partial\theta_m = (\partial X_g/\partial\rho_m)(\partial\rho_m/\partial\theta_m), \qquad\qquad m = h,\ w;\ g = 1, 2.$$

The first and last terms in equation (15B) are ratios of the effects of bargaining power of any two household members, say h and w on demands for commodities 1 and 2. The middle term captures the idea that a change in bargaining power of either household member changes the individuals' shares in total household income. If there is income pooling, the changes in budget shares experienced by the two individuals will be the same, irrespective of whether it is the bargaining power of h or w that has changed, so that the ratio of the two magnitudes is equal to one. Since the middle term is common, the test for income pooling or for Pareto efficiency on the consumption side of the household model involves a comparison of the first and the second terms. If both ratios are the same across the two commodities, Pareto efficiency cannot be rejected. The constancy of the ratios across the two commodities is an indication that there is no gain from the re-allocation of the commodities, i.e, the existing intra-household allocation is Pareto optimal. Equation (15B), an expression for the test of Pareto efficiency using the consumption side of the household model, is easily generalizable to a multiple commodity case.

In expression (14A), the household maximizes the weighted sum of utilities that its members derive from individual consumption, given the Pareto weights, τ_m. The equation represents only one of the ways of modeling the welfare of the household in collective decision making contexts, with a focus on consumption. The key assumptions underlying the model are that the outcome of resource allocation within the household is Pareto efficient, and that the household has a functioning resource sharing rule.

Chiappori (1988), Chiappori (1992) and Browning et al. (1994) propose collective models in which Pareto efficiency is the only assumption needed, arguing that it implies the existence of intra-household resource sharing rule. In these general collective models, each household member maximizes the unweighted version of equation (14A) subject to his own budget constraint, provided that total consumption expenditure of all members does not exceed the full household income (see Browning et al., 1994, p. 1074). The assumption that the outcome of resource allocation decisions of individuals within a household is Pareto efficient implies that for a given a resource sharing rule, welfare gains from intra-household redistribution have been exhausted.

Udry (1996) develops a general collective model capable of analyzing both consumption and production decisions of households, and uses it to test the Pareto efficiency assumption with data from Burkina Faso.[9] He finds large production inefficiencies within households, contrary to the Pareto assumption of efficiency. In particular, crop output per acre in plots managed by women was found to be 30% lower than the output on similar plots controlled by men planted with the same crop in the same year.

In African agrarian contexts, a woman's plot serves as a school as well as a farm (Udry, 1996, p. 1034). Thus, apart from allocative inefficiency, due for example to transactions costs, an alternative reason why crop output per acre on a woman's plot might be lower than on a man's plot within the same household, is that the woman's plot is being used to teach children how to farm, i.e., to produce farm-specific human capital for the household. If this were so, women's plots would have higher intensities of child labor than men's plots. Since this was not the case in Burkina Faso, Udry rejected the Pareto efficiency assumption. The assumption has also been rejected by Dercon and Krishnan (2000) with a consumption-based test applied to Ethiopian data. However, Quisumbing and Maluccio (2003) fail to reject Pareto efficiency assumption. To date, the Pareto efficiency hypothesis has been rejected only with evidence from African countries.

Udry's model can easily be adapted to analyze health production and health care utilization activities within a household. A woman's plot is not only a farm as well as a school, but also a clinic. Since a primary activity of women in agrarian communities is child rearing, small children in these settings would typically be found on women's plot. In the event of illness, the children would receive initial treatment on this plot, and if treated outside the plot, return to the same unit for subsequent care. Women may farm their plots less intensively than men, not only because they use some of their time to teach children about farming, but also to meet the health and nutrition needs of children. Thus, although women's crop output per acre on a plot may be lower than on a similar plot controlled by men, women's contribution to child health on their plot would likely exceed that on men's plot, a situation that could then be consistent with Pareto efficiency. These health human capital considerations greatly complicate the testing of Pareto efficiency within a household. Pareto efficiency failures in contexts of health production within a household, may be more severe than in agricultural production contexts studied by Udry, because information about health technology is likely to be asymmetrically held among household members. For example, information about treatment of childhood diseases such as diarrhea or malaria may differ significantly between wife and husband. This information asymmetry could account for any observed differences in child health

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[9] In contrast to expression (15B), the test was implemented using a productivity equation of the form:

$Q_{htci} = X_{htci}\beta + \gamma G_{htci} + \lambda_{htc} + \varepsilon_{htci}$

where,

X_{htci} is a vector of characteristics of plot i planted with crop c at time t by a member of household h (X_{htci} includes, along with other information, the area of the plot); Q_{htci} is the yield on the plot; G_{htci} is gender of the individual who controls the plot; λ_{htc} is the household-year-crop fixed effect that restricts attention to the variation in yields across plots planted to the same crop within a single household in a year; and ε_{htci} is an error term that summarizes the effects of unobserved plot quality variation and plot-specific production shocks on yields; see Udry (1996, pp. 1013-1015) for the complete model; notice that Pareto efficiency implies that $\gamma = 0$.

outcomes by the gender of the parent providing non-earned income for medical treatment or giving home remedies for an illness. In the case of adult health, Pitt and Rosenzweig (1985, 1986) could not reject the hypothesis that a reduced-form health production technology of the head of a farm-household in Indonesian was different from that of his wife, a finding that is consistent with asymmetry of information regarding prevention and treatment of illnesses among household members.

In general collective models (see e.g., Chiappori (1988), Browning et al.(1994) and Udry (1996), independent resource allocation decisions of household members are beneficial to the whole household. This aggregate, Pareto efficiency outcome of allocation decisions of self-interested household members is reflected by the linearity of the household-level welfare function as in equation (14A). In this framework, household members advance their collective well-being without coordinating their individual decisions. However, the fact that the household members live together and interact repeatedly, may enable them to learn the household-level needs to which each is obliged to contribute. Indeed, individuals who live together cannot succeed in some vital production and consumption activities without cooperative behavior that coordinates self-interests. In general collective models (see Chiappori, 1988), cooperative behavior is implied by the assumption that the outcomes of individual decisions within a household are Pareto optimal. This formulation of general models is in stark contrast to parallel bargaining models of household behavior where explicit cooperation among household members in their individual activities is modeled, and is key to the advancement of household welfare.

Manser and Brown (1980) and McElroy and Horney (1981) propose bargaining household models in which cooperation among household members is required for existence of a household-level welfare function. In contrast to equation (14A), where a household maximizes the weighted sum of personal utilities, in cooperative bargaining models, the household maximizes the *product* of net personal utilities of its members; see Manser and Brown (1980, p. 38; McElroy, 1997, p. 57). The household-level utility is zero whenever one of the personal utilities is zero (due for example to non-cooperation, which may be motivated by options outside the household). This was the starting point of Nash (1950, p. 155) in his analysis of the bargaining problem: "A two-person bargaining situation involves two individuals who have the opportunity to collaborate for mutual benefit in more than one way. In the simple case, which is considered in this paper, no action taken by one of the individuals without the consent of the other can affect the well-being of the other one."

Cooperation within a household facilitates production and consumption of health and health care. In cooperative household models, as in other collective models, the amount of health care consumed by an individual, and thus the health status of that individual, depends on his bargaining power within the household. The household bargaining models have fruitfully been used to analyze intra-household distribution of health care and health outcomes in developing countries (see Strauss and Thomas, 1995). However, this model has the shortcoming that it ignores conflicts among household members even in the absence of outside options that would otherwise motivate the conflicts.

Chen and Woolley (2001) propose a non-cooperative household model in which individuals act strategically within a household to maximize self-interest, taking as given the behavior of other members. In this model, an individual cooperates with other household members if the utility from

cooperation exceeds that from his selfish behavior within the household. Chen and Woolley (p. 732) use the concept of Cournot-Nash equilibrium to analyze this conditional cooperation. A threshold level utility from strategic behavior is modeled as a threat-point to cooperation, where the threat is about a return to a non-cooperative behavior in a separate sphere of a household (see Lundberg and Pollak, 1993, 1994; Duflo and Udry, 2004), rather than about quitting the household as in the cooperative bargaining models of Manser and Brown (1980) and McElroy and Horney (1981).

McElroy (1997, p. 61) uses a diagrammatic scheme to classify household models into two main overlapping groups: the bargaining models and the Pareto-optimal models. The Nash bargaining models and the unitary models (the family utility models) are depicted as special cases of the subset of the Pareto-efficient models encompassed within the bargaining models. McElroy's diagrammatic scheme is quite useful as it illustrates the relationships among the dominant household models, all of which are designed for partial-equilibrium analysis of production, consumption, saving and other activities of households.

The non-cooperative bargaining model is quite appealing if individuals are assumed to intuitively know their personal utilities in separate spheres of the household, and then use that information to change their bargaining power. These models can be used to analyze health capital formation within households where public goods serve as inputs into health and health care production. Examples of household public goods include, housing, sanitation services, the caring for chronically ill-members of the family, and other vulnerable members such as orphans and the elderly, and treatment and prevention of communicable diseases.

The collective model of the household has major advantages over the unitary model in an analysis of human capital effects of public policy and other relevant variables within a household. First, the model focuses on an individual as a unit of analysis, and thus is able to address the conflicting interests of household members. Second, in a collective model, the person controlling household resources is identified, thus allowing an analysis of direct health effects of placing resources in the hands of different household members, e.g., women with young children. Third, the collective model permits the testing of the assumptions of the unitary model, e.g., altruism, income pooling and the Pareto efficiency outcome of resource allocation decisions. Fourth, although both models are able to explain intra-household differences in incomes and human capital outcomes (Strauss and Thomas, 1995), the unitary model relies on unrealistic assumptions. For example, the unitary model assumes that unobservable factors such as "invisible hand" and "love" account for the observed intra-household allocations. In contrast, collective models explain intra-household allocations using the concept of bargaining power, which is determined by observable factors, such as unearned income, demographics, violence or threat of violence, and career or marriage options outside the household.

The collective models that address conflicts, cooperation, and other interactions within a household through a bargaining process, are better suited to analyzing intra-household allocations of health care in low-income countries than the parallel collective models that posit Pareto efficiency as the sole mechanism through which household members achieve their individual and common goals. Because of imperfection and incompleteness of health care markets in developing countries, the outcome of resource allocation based on such markets is unlikely to be Pareto efficient.

4.4. **Income and Health**

The relationship between income and health at the micro- and macro-levels is widely studied (see e.g., Audibert, 1986; Summers, 1992; Pritchett and Summers, 1996; Schultz and Tansel, 1997; and Strauss and Thomas, 1998). Interest in a two-way relationship between health and income, especially in developing countries, arises from the need to design policies based on this relationship to improve living standards (Strauss and Thomas, 1995, 1998).

There is an established literature on theoretical models of nutrition-based efficiency wages, showing strong non-linearities between calorie intake and labor productivity (Bliss and Stern, 1978a,b; Strauss, 1986; Dasgupta, 1993). Workers who consume more calories are more productive. These models imply that employers have an incentive to raise wages to give workers incomes that would enable them to consume productivity-enhancing amount of calories. At the same time, the employers have an incentive to exclude the poorest and malnourished workers from wage employment because such workers are too expensive to hire. This latter type of incentive works against poverty reduction policies of governments in low-income countries, where expansion of labor market participation rates is the main strategy used to fight poverty. However, despite this important policy implication of the efficiency wage models, little credible empirical work exists on effects of nutrition on labor productivity in low-income countries (Srinivasan, 1994).

The functional consequences of malnutrition and illnesses during childhood are felt throughout the life-cycle (Deaton, 2006; Fogel, 2004; Fogel and Costa, 1997; Strauss and Thomas, 2007). Thus, prevention of childhood diseases and malnutrition would substantially increase health and economic growth in low-income countries. Child nutrition investment, especially those that promote school feeding programs can supplement the nutrients children receive at home.

Since income has a strong impact on health (the so-called "wealthier is healthier hypothesis"), there is a need to know how health benefits of growth are distributed in the population (Pritchett and Summers, 1996; Case and Deaton, 2005). If economic growth benefits a few, its impact on health will also be limited to a few. As demonstrated in Section 4.3, such an inequality can only be corrected in the long-run because it is the distribution of health inputs, rather than of health per se, that has to be changed. Information on effects of income on health across social groups can help design health policies that promote equity in health outcomes in the population (Gakidou, et al., 2000). The ensuing sub-sections survey the literature that clarifies the above issues in the context of developing countries.

Measurement of health

Health is multidimensional and thus has a variety of measures, each of which is likely to have a different effect on productivity and labor market outcomes such as wages, nature, and duration of employment (see e.g., Strauss and Thomas, 1995; Ahlburg, 1998). As pointed out by Fuchs (1982), "there is no one measure of health status (or even one summary measure) that is best for all purposes" (p. 11). Several types of measures of health are identified and discussed below.

1.	The first measure relates to indicators of general health, typically derived from household surveys. Individuals in a probability survey are asked to report on their health status. An individual is typically asked to indicate whether his health status is excellent, good, fair or poor. The responses are then averaged to determine the proportions of the populations that correspond to each of the above discrete categories. This measure has the disadvantage that people may perceive health differently so that good health might not mean the same thing to all people. Furthermore, if health perceptions are systematically correlated with socioeconomic characteristics such as income and exposure to health care systems, self-assessed health status can be misleading. For example, sick individuals in a poor disease endemic area, with limited opportunities for medical treatment may report being in good health because some illnesses such as blindness, ringworms or malaria may be perceived as normal phenomena due to their prolonged, widespread occurrence in the area. Such people might be adapted to the sickness that they experience (Banerjee et al., 2004).

Other self-reported measures of general health include illness restricted activity days, and disease symptoms, which are also subjective. A less subjective measure is self-reported or externally observed performance in activities of daily living (ADLs). The ADLs are used mainly to measure the health status of the elderly rather than of the youth or prime-age adults (Strauss and Thomas, 1995; Schultz, 1999). The most commonly used measures of general health include mortality and morbidity rates, life expectancy at birth and various indicators of disease burdens, e.g., disability adjusted life years and quality adjusted life years (Culyer and Newhouse, 2000). However, these measures can be constructed only for large populations (not individuals) and then only if vital events in the populations are well recorded.

2.	Ability to move out of birthplace to take advantage of health care services elsewhere, to avoid disease endemic areas, or to find employment may be a proxy for health status. However, this form of health status is difficult to measure. For example, a migration dummy, that takes a value of one if a person migrated from his birthplace and zero otherwise, could capture a non-health attribute such as educational attainment. However, if information is available about types of diseases prevalent in a particular area, a migration dummy to the healthy location could be interpreted as a measure of health status. In particular cases of river blindness and malaria, persons migrating to cities from rural areas endemic to these diseases are likely to be healthier in cities than their rural counterparts. Thus, migration status needs to be combined with specific health information both about the birthplace and current area of residence to serve as a measure of health status.

3.	Biomedical evidence suggests that calorie intake is correlated with increases in oxygen uptake (Strauss and Thomas, 1998). Measures of calorie consumption include daily per capita calorie availability or intake. The availability measure has the obvious disadvantage that the available quantity of calories may not be consumed due to wastage and other reasons, while the intake measure is prohibitively expensive to obtain because it requires an intrusive household survey. The most common approach to obtaining calorie intakes of household members is to ask them to recall ingredients that went into meals consumed, usually over the previous 24 hours. This is a time intensive but feasible method. The calorie intake is a measure of the quantity of nutritional inputs used to produce health. The outcomes of these inputs, expressed in terms of anthropometrics, such as height, weight, arm circumference, and body mass index (the ratio of weight in kilograms to height in meters squared) are other accumulative nutrition-based measures of health. However, caution

should be exercised in using calorie consumption data from short time intervals to make conclusions about nutrition and health status because energy intake of an individual can vary within a homeostatic range without impairing health and without changes in body mass (see Srinivasan, 1992).

4.   Hemoglobin levels of individuals can be measured through household surveys to provide information about prevalence of anemia in children and adults. Anemia occurs when the blood does not have enough hemoglobin, a red pigment in red blood cells that helps carry oxygen from lungs to all parts of the body. Anemia might be caused by iron deficiency, malnutrition, worm infestation and other health problems. The hemoglobin level in the blood can easily be measured by paramedical personnel. Typically, 11-13 grams per deciliter (g/dl) are used as the cutoff points for adequate levels of hemoglobin in the blood (see Banerjee et al., 2004; Gertler, 2004; Thomas et al., 2006). Persons with hemoglobin levels below the thresholds indicated above are likely to suffer from fatigue, low body temperature, rapid heartbeat, shortness of breath, chest pain, dizziness, headache, irritability, numbness and cold in the hands and feet, conditions that can cause death or interfere with schooling or work performance.

5.   The type of disease (e.g., acute or chronic) is a good indicator of health status of an individual. A long-term disability such as diabetes does not have the same effect on health as an acute condition such as an injury. Disease categories constitute notable measures of community and personal health, and are suggestive of clinical and other interventions that are required to improve health at various levels of society (Jamison, et al., 2006). However, since diseases are diagnosed after individuals have had contacts with health care system, they reflect health care treatment choices of individuals and households. Thus, disease types cannot be treated as exogenous in the measurement of economic effects of health, a caution that also applies to health measures indicated in (1)-(4) above.

Health and labor market outcomes

What regularities exist between health and labor market outcomes such as wages and labor force participation? Strauss and Thomas (1998) show a strong and positive correlation between adult height and hourly wages in the United States and Brazil in the 1970s. Taller men in both countries earn more, especially in Brazil. In Brazil, a 1% increase in men's height is associated with about 8% increase in hourly wages. However, with controls for other dimensions of health human capital, the height elasticity of Brazilian wages declines to about 4% (Strauss and Thomas, 1997).

In a more recent study, in which wage effects of height are corrected for endogeneity, Schultz (2002) reports large differences in effects of height on wages between United States and two low-income countries (Brazil and Ghana). At the margin, wage returns to height in Brazil and Ghana are about three times larger than the returns in the United States. A centimeter increase in height in Brazil and Ghana is associated with 8-10% increase in wages for both men and women, i.e., a 1% increase in height raises wages by 13-17%, a large increment, compared with the earlier upper bound estimate of 8% reported by Strauss and Thomas (1998).

The differences in wage returns to nutrition between developed and low-income countries reflect nutrition levels, as well as the types of work available in the two societies. The mean height in the

United States is higher than in Brazil and Ghana. Thus, because of diminishing returns to nutrition, the wage effects of height are lower in the United States. The larger share of white collar jobs in the United States may also contributes to lower returns to height there, because height may not be a good proxy for the type of human capital required to perform white collar jobs.

Strauss and Thomas (1998) show a persistent correlation between the fraction of urban men who are not working and their nutritional status. In urban Brazil, short men earn less than tall men, and are less likely to be working. A similar profile emerges from the same data that combines height and weight. The probability that a man is not working decreases until his BMI reaches around 24, at which point the employment probability becomes essentially flat. In other words, in Brazilian urban areas, overweight and obesity are negatively correlated with men's chances of finding employment. However, in rural areas where malnutrition is still prevalent, overweight and obesity may not be a problem. These results suggest different returns to nutrition (as proxied by height and body mass index) in Brazil and the United States.

Wage Effects of Health

Is health the source of the observed variation in wages among workers? This is a difficult question to answer because health is endogenous to wages. To help answer this question, Strauss and Thomas (1998) specify the following wage function

$$w = w(H, A, S, B, I, \alpha, e_w) \tag{20}$$

where,

w is an individual's log of real wage;
H is an array of measured health human capital;
A is a vector of demographic characteristics;
S is education human capital;
B is family background of the individual, which includes education human capital of parents;
I is local community infrastructure such as electrification or road density;
α is an array of unobservables such as ability;
e_w is measurement error.

In estimating equation (20), problems of simultaneity, heterogeneity, measurement errors, and omitted variables must be addressed. The problem in equation (20) is to isolate the effect of health on wages. The α-variables play a major role in the estimation of equation (20). Some of these variables include time-invariant natural ability, while others may include the social network of individuals, which varies over time. If available, panel data can be used to deal with the individual-specific, time-invariant α-variables. However, the time-varying αs would continue to be a source of heterogeneity in wages. Strauss and Thomas (1998, p. 778) suggest that local community infrastructure, the disease environment, prices of food and prices of health inputs can be used as valid instruments for health. Although these factors would normally affect health status without directly influencing labor productivity, there may be some important exceptions. For example, low-productivity workers might migrate to locations with high densities of roads and clinics or such facilities may be selectively placed to serve low wage workers. In either case, local densities of

roads or clinics would be weak instruments for the health of workers. Moreover, which variable is used as an instrument for health depends on how health is measured. If health indicators are nutrition-based, food prices as well as fees for medical care services would be potential instruments for health (see Strauss, 1986).

Effects of Health on labor supply

The next issue concerning the relationship between health and labor market outcomes is whether better health increases labor force participation and labor supply. The welfare effects of health manifest themselves through labor force participation. To analyze labor supply (and participation) effects of health, Strauss and Thomas (1998, p.780) specify the following model of labor supply, conditioning it on health and wages.

$$L = L(H, pc, w\{H; S, A, B, I, \alpha, e_w\}, S, A, B, V, \xi) \tag{21}$$

where,

L is labor supply or labor market participation;
pc is a vector of prices for consumer goods;
$w\{.\}$ is the real wage;
V is non-labor income;
ξ is the taste parameter, with the other variables being defined as in equation (20).

Estimation of equation (21) permits the separation of the labor supply effect of health, H, from the effect of preferences, ξ. Apart from the problem posed by the joint determination of wages and labor supply, isolating the role of health and preferences in the labor supply is complicated, because preferences are unobserved, and health is measured with error, and its identification based on local infrastructure is subject to debate. The measurement error in health forces one to use IV for H, i.e., to look for instruments for health. There are other factors complicating the estimation of (21). As can be seen from equation (21), health status influences labor supply and labor market participation by changing wages, inducing both income and substitution effects that may bear opposite signs. In addition, the workers' preferences for leisure and goods affect labor supply directly, as well as indirectly through the substitution effect induced by wages. As in equation (20), local social infrastructure and prices can be used as instruments for health. The instruments should be designed to purge the covariance between health status and the error term of the labor supply equation.

In addition to the above individual-level labor supply effects of health, household-level effects may also be important. Thirumurthy et al (2005) show that the effect of an individual's health on his own labor supply can be quite different from its effect on the labor supply at the household level. An improvement in BMI and in the CD4 count of AIDS patients receiving ARV therapy in Western Kenya, led to an increase in individual labor supply, but to a reduction in the labor supply of other household members at the household level, notably among adult women and young boys.

Effects of health on self-employment profits

Equations (20) and (21) focus on effects of health on labor market outcomes. The same equations can be modified to investigate effects of health on profits generated from self-employment as well as the number of hours worked. Local prices and infrastructure remain valid instruments for health.

However, if labor and credit markets exist, health has no effect on self-employment output or profits because sick family labor can be replaced by market labor (Pitt and Rosenzweig,1986). Pitt and Rosenzweig showed that although illness reduced the farm-labor supply of males in Indonesia, it had no effect on farm profits because ill farm-workers could be replaced by hired labor. Strauss (1986) showed in the case of rural Sierra Leone that the marginal product of family labor was practically the same as the marginal product of hired labor. In such a setting, profit or output from self-employment can be written as

$$\pi = \pi(pf, pm, wh, wf, I, F, S, \varphi, e_\pi) \tag{22}$$

where,
π is profit from self-employment;
pf is the price of output, say, food;
pm is the price of non-labor input;
wh is the wage of hired labor;
wf is the wage of family labor;
I is local infrastructure;
F is a vector of fixed factors;
φ is a vector of unobserved factors affecting the production of self-employment output;
e_π is measurement error in profits.

The essential point in equation (22) is that health is not a determinant of profits or output when labor supply and production decisions are separable. However, if a well-functioning labor market does not exist, or if family and hired labor are not perfect substitutes, profits and outputs generated from self-employment activities will depend on health status. Equation (22) illustrates that the nature of local labor markets is important in specifying the role of health in self-employment production functions. In the above setting, separability of a household's decisions in production and consumption is assumed. "That is, the household acts as though its decisions can be divided into two steps: first, maximize self-employment profits subject to the available technology, and second, maximize utility subject to the budget constraint augmented by the value of profits" (Strauss and Thomas, 1998, p. 782). See Singh et al. (1986) for an analysis of separability of consumption, production and labor supply decisions of farm households.

Separability in farm household models means that production decisions can be made independently of decisions as to consumption of goods and leisure, but the reverse is not true. That is, consumption decisions must involve production decisions, because consumption is constrained by income. Production decisions are independent of consumption here because all the production inputs required are obtainable from labor and credit markets, which are assumed to exist (see Singh et al, 1986).

Productivity Effects of Health in subsistence agriculture

In farm environments without the separability feature, Strauss (1986) in Sierra Leone, and Audibert (1986) in Cameroon find substantial effects of health on farm productivity. In Cameroon, a reduction in the incidence of bilharziasis increased rice production, but a reduction in malaria prevalence had no output effect. However, these results were not corrected for endogeneity of health status. In Sierra Leone, where instruments for health were used, calorie intake was found to have large effect on productivity. The instruments for calories (proxy for health) included local infrastructure and food prices. Dercon and Krishnan (2000) cite evidence that body mass index (Quetelet index) is positively correlated with ability to perform strenuous work and with farm productivity of women.

However, there is need to recognize that the relationship between nutrition and productivity is not a simple one because "energy intake could vary in the short-term within a homeostatic range without affecting the health and work capacity" (Srinivasan, 1994, p. 1851). Srinivasan (pp. 1848-49) explains the non-linearities between nutrition and farm productivity as follows: "The efficiency wage is based on the relationship between nutritional status and productivity of the laborer. In this relationship the requirement of energy for maintaining basic metabolic processes (and for performing activities other than agricultural work) serves as a threshold: if food intakes do not provide enough energy to exceed this threshold, it is impossible (physiologically) for the laborer to engage in agricultural work."

Taken together, the above two ideas indicate that for well-nourished workers, energy intake can vary up or down within some range without affecting farm productivity, while for severely malnourished laborers an increase in energy intake would not affect their productivity, if the amount of energy consumed remains below the threshold that an individual needs to engage in farm work.

Effect of income on health

The macro level effect of income on health (the wealthier is healthier hypothesis) is documented in Pritchett and Summers (1996). There is strong micro level evidence that income affects health. In Brazil, Thomas (1990) estimates large effects of mother's non-earned income on child health. In Sierra Leone, Strauss (1984) finds significant effects of income on nutritional status. Deaton and Subramanian (1996) report similar findings for South India. Despite the controversy surrounding the calorie-income relationship (Behrman and Deolalikar, 1987), the micro level evidence shows that increases in incomes raise caloric consumption, especially at low levels of income (see Strauss and Thomas 1995). However, the elasticity of calories with respect to income is less than unity. Pitt et al (1990) show that the income calorie elasticity in Pakistan is low during harvest time.

4.5.   Identifying Causal Effects of Better Health: Examples

Measurement of effects of health on income, and vice-versa, is complicated by endogeneity of both health and income in the estimated equations, and by the externalities of health human capital.[10] The

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[10] Duflo (2004) has shown that while education increased the wages of the workers who were exposed to a

instrumental variables methods, and experimental treatment designs, are two approaches that have been developed to deal with this problem. A study by Schultz (2003) in West Africa (Cote d'Ivoire and Ghana) is used to illustrate the IV approach, while studies by Thomas et al (2006) in East Asia (Indonesia), Miguel and Kremer (2004) in East Africa (Kenya) and Gertler (2004) in Central America (Mexico) illustrate the experimental treatment approaches.

The IV methods and the randomized designs are employed to solve differently the same problem, namely, identification and estimation of a causal effect. The causal effect in both approaches is identified through a two-stage process. In the first stage, variation of the hypothesized causal factor is made exogenous; in the second stage, the impact of the causal factor on outcome of interest is measured. The studies illustrated in this section attempt to answer the question: what is the effect of better health on economic or social prosperity?

In solving this problem, the first stage in the IV method is to find exogenous variables that affect health without influencing the outcome of interest (e.g., wage income). Similarly, in the experimental approach, the first stage involves organizing a field experiment that varies health exogenously without affecting the outcome variable of interest, i.e., wage income. The factor that causes health to vary exogenously is called an instrumental variable in the IV approach, while in the experimental approach, it is known as treatment assignment mechanism (see Todd, 2007). In the IV approach, estimation of a causal effect is through regression methods, whereas in randomized experiments, simple differencing of values of the outcome variable across treatment and control groups may suffice (see Duflo et al., 2007).

There are important caveats about the two approaches which need to be stated: the results from experimental designs are not easily generalizable, and the experimental designs themselves may not meet strict criteria of randomness (Rosenzweig and Wolpin, 2000). In the same vein, the IVs results may not apply outside the study site, and the IVs themselves may be weak and correlated with the outcomes of interest.

To overcome the problem of specification bias associated with the IVs, Rosenzweig and Wolpin (2000, p. 828) recommend the use of "naturally random events as instrumental variables" whenever possible, and provide five examples of such instruments, linking them to particular studies, namely: twin births, monozygotic twins, birth date, gender, and weather shocks.

The problem of experimental designs in field settings not meeting criteria of randomness, usually due to selection bias, may be overcome by advance planning of field activities and by working closely with local communities to ensure that study subjects comply with experimental designs (see Thomas et al., 2006; Parker et al., 2007).

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large school construction program in Indonesia, the wages of unexposed workers fell or increased less rapidly. Thus, the aggregate effect of education expansion can be negative, if for example, a complementary factor of production such as infrastructure cannot be adjusted. Similarly, although a malaria control program may increase farm yields per hour in a particular area, its economy-wide effect may be negative if market inputs remain fixed as health human capital increases and the marginal product of labor declines.

The four cases below illustrate the use of IV methods and randomized experimental designs in the estimation of causal effects of better health. The approaches themselves are generic even though they are drawn from applications in specific countries.

The IV Methods

Schultz (2003) uses the IV regression method to estimate the income effect of health in Ghana and Ivory Coast using survey data. In the first stage regression, instruments for major forms of human capital (not just health capital) are identified; see also Thomas and Strauss (1997). In the second stage regression, the coefficients on endogenized human capital variables, with a focus on health, are estimated. The first step is accomplished by estimating the following input demand equations.

$$I_{ij} = \alpha_j \bullet Y_i + \beta_j \bullet X_i + \varepsilon_{ij}, \quad j = H, E, B, M \qquad (23)$$

where,   i = 1, ..., n

I_{ij} is human capital investment of type *j* embedded in individual i;
H = Adult height, a dimension of health human capital that indicates childhood nutritional status;
E = Years of schooling (a measure of education human capital);
B = Body mass index, an indicator of adult nutritional status and current health;
M = Migration status (whether the individual has migrated from region of birth), an indicator of combined elements of education and health.
Y = a vector of variables that affect the demand for human capital partly through its impact on wage structures and through other channels;
X = a vector of variables that affects the demand for human capital without modifying the wage structure;
ε_{ij} = the iid error term.

In the second stage, the predicted values of the various forms of human capital (I^*_{ij}) are used to estimate the wage or productivity equation of the form:

$$w_i = \Sigma_j \gamma_j \bullet I^*_{ij} + \delta Y_i + v_i \qquad\qquad j = H, E, \text{B}, M \qquad (24)$$

where
w_i = the logarithm of the wage rate for individual i;
Y_i = a vector of variables that belong to the wage function for individual i;
γ_j = the effect of human capital *j* on log wage;
δ = log wage effects of covariates, *Y*.

Equation (24) conveys the key idea that when estimating the wage effect of one form of health human capital, e.g., body mass index, the effects of all the other forms of human capital must be held constant. Failure to do this results in a mis-specification of the wage function, because the omitted human capital variables are relegated to the error term, thus biasing the wage effect of health. For

example, to estimate the impact of BMI on the wage rate using equation (24), controls are needed for education, migration, and height.

Because all forms of human capital must be included in the wage function, and not just the health status variable of interest, a generic set of instruments for human capital has been identified in the literature (see Strauss, 1986). Since this set of instruments has become standard, an example from Schultz (2003) is presented in Table 1. (The results for Ivory Coast and for female sub-samples are omitted).

The human capital variables shown in Table 1a are for adults at the time of the survey collection, while the family background variables refer to earlier periods when human capital of the adults was being formed. The instrumental variables in Table 1a affect demands for human capital inputs in the direction predicted by theory. For example, distances to social facilities are negatively associated with measures of human capital. Employment in agriculture is negatively correlated with household demands for human capital, a result that may be attributed to low incomes in agriculture. Although a number of coefficients on instrumental variables are statistically insignificant, the hypothesis that the joint effect of all the instrumental variables is non-zero cannot be rejected (see Schultz, 2003, Table 5), a finding that enhances the validity of the instruments (Bound et al. 1995).

The diagnostic results that must be reported include the first stage F-statistics and incremental R-squareds on exclusion restrictions. Following the first stage regression, exogeneity and overidentification tests should also be carried out and their results reported (see Schultz, 2003, pp. 347-8). The stata estimation command, *ivreg2*, has two options (*ivendog* and *overid*) that may be specified to carry out these otherwise computationally burdensome tests.

Table 1a

Instruments for Endogenous Human Capital Variables in a Wage Function (Selected Results from the First Stage Regression for the Male Sub-sample, Absolute *t*-Statistics in Parentheses), Ghana

Instrumental Variables	Various Forms of Human Capital			
	Education[b]	Migration[b]	Body Mass Index[b]	Height[b]
Mother's years of education	0.779 (2.13*)	-0.0007 (0.27)	-0.0300 (1.45)	0.0006 (1.03)
Father's years of education	0.122* (5.63)	0.0025 (1.72)	0.0452* (3.67)	0.0000 (0.15)
Father's education unknown (=1)	0.687 (0.81)	-0.0147 (0.26)	-0.684 (1.41)	0.0215 (1.66)
Mother employed in agriculture (=1)	-0.725* (3.97)	-0.0774* (6.24)	-0.605* (5.81)	-0.0216* (4.54)
Father employed in Agriculture (=1)	-0.424* (2.03)	-0.0014 (1.10)	0.108 (0.91)	-0.0019 (0.60)
Rural Resident[a] (=1)	0.101 (0.46)	0.921* (61.4)	0.165 (1.32)	0.0069* (2.09)
Rainfall[a] (annual mm)	0.0063 (0.46)	-0.0059 (6.32)*	0.0153 (1.96)*	-.0003 (1.68)
Malaria (=1)	-0.7690* (2.47)	-0.0647* (3.06)	-0.339 (1.92)	-0.0143* (3.03)
Diarrhea (=1)	-0.204 (0.78)	-0.137 (7.62)	0.117 (0.79)	-0.0000 (0.01)
Measles (=1)	0.506 (1.70)	-0.141* (6.96)	-0.239 (1.41)	0.0065 (1.43)
Sanitation and water problem (=1)	0.274 (1.00)	0.142* (7.62)	0.0397 (0.26)	-0.0031 (0.76)
Immunization campaign in last 5 years (=1)	-0.231 (0.64)	-0.0922* (3.74)	-0.180 (0.88)	-.0019 (0.35)
Distance to nurse or doctor	0.0282	-0.0021	0.0131	0.0005

(km/miles)	(1.22)	(1.37)	(1.00)	(1.32)
Distance to permanent market (km/miles)	0.0069 (0.38)	-0.0106* (8.65)	-0.0031 (0.37)	0.0002 (0.79)
Distance to primary school (km/miles)	-0.198 (1.69)	-0.0503* (6.32)	-0.0031 (0.37)	0.0002 (0.79)
Distance to middle school (km/miles)	-0.199* (3.85)	-0.0373* (10.6)	-0.0399 (1.36)	0.0007 (0.87)
Distance to secondary school (km/miles)	-0.283* (2.49)	-0.0076* (9.86)	0.0011 (0.16)	0.0005* (2.66)

Notes: * = Statistically significant.
a: These variables are also in the wage function, and thus do not belong to the set of the identifying instrumental variables, i.e., they are in *Y* but not in *X* in equation (23).
b: Other control variables include 4 age dummies (in *Y*), 6 ethnic groups (in *Y*), 10 regions (in *Y*), 10 local food prices (in *X*), state level per capita expenditure on curative and preventive health programs in 1987 (in *X*), and malaria campaigns in the last 5 years (in *X*).
Source: Extracted with minor changes from Schultz (2003, Table A.2).

The IV estimates of the effects of human capital variables on wages for male workers in Ghana are shown in Table 1b; the OLS estimates are reported for comparison purposes. The results are from a linear wage function (estimates from non-linear specifications have been omitted). It should be noted that if equation (24) is estimated with endogenized human capital inputs as the regressors, the standard errors of regression coefficients must be adjusted (see Wooldridge, 2002, p. 568).

As can be seen from the table, the IV estimates (column 2) are substantially larger than those obtained via OLS (column 1). Focusing on the IV estimates, it is evident that there are large returns from better health: a unit increase in mean BMI raises the log wage by 0.079, whereas an extra meter above the mean height increases the log wage by 5.69. In other words, a unit increase in BMI in Ghana increases male wages by 7.9%, while a male worker whose height is 1 cm taller than the mean height earns about 1.3% higher wage than a worker of average height. Height also effects labor productivity through its effects on choice of occupation (see Fogel, 1986).

The effects of other human capital variables on wages are also large, and statistically significant. The IV results show that a year of schooling increases male wage rate by 4.45% while migration increases it by 21.8% above the mean wage at the place of birth. It is important to emphasize that without controls for education and migration, estimates of returns to health investments would be biased. The stocks of human capital in Ghana in the 1980s are shown in the last column of Table 1b (see Schultz, 2003, Table 2). Although the BMI for males in the 1980s was within the normal range of 18.5 to 25 (see Fogel, 2004), the mean education level at around 7 years was quite low. The migration dummy shows that one third of male workers had moved from their birthplace prior to the household survey. The estimated coefficients on human capital variables in columns (1) and (2) of Table 1b show the response of the mean log-wage to marginal changes in the

sample means of these variables (column 3). Thus, descriptive statistics are an essential part of estimation results and should always be reported.

Wage Effects of Four Human Capital Inputs in Ghana: Dependent Variable is Log Hourly Wage for Adult Males (absolute *t*-statistics in Parentheses)

Variables	Estimation Method (N=3414)		Sample Means
	OLS (1)	IV (2)	(3)
Education (Years of Schooling Completed)	0.0437* (9.86)	0.0445* (2.46)	7.13 (5.47)
Migration (Moved from Birthplace =1)	0.348* (6.75)	0.218* (2.26)	0.333 (0.471)
BMI (Body Mass Index (kg/m^2)	0.0530* (6.80)	0.0793* (1.95)	20.9 (2.52)
Height (Meters)	1.48* (5.02)	5.69* (3.45)	1.69 (0.066)
Mean of Dependent Variable (Standard Deviation)			5.77 (1.38)

Notes: Controls in all the regressions include region of birth, ethnicity, age group, and season of interview; * = statistically significant.
Source: Constructed from Schultz (2003, Tables 2 and 4).

Randomized experimental studies

Indonesian study

Thomas et al. (2006) present a randomized field experiment on how consumption of nutrients affects nutritional status, and how better nutrition in turn, affects labor earnings, employment, leisure, physical health, and psycho-social health. WISE (Work and Iron Status Evaluation) is the name of the field experiment, implemented in Java, Indonesia, over the period October 2001 to December 2004. The experiment randomly assigned iron tables and placebos to households, with the households that received placebos serving as the control group. Differences in the outcome of the experiment between the control and the treatment groups was then evaluated. The results presented below relate to the outcome of the experiment over the first 12 months. A brief account of the experiment may be helpful in interpreting the results, which are also highlighted elsewhere (see Todd, 2007).

The health input studied was supplementation of the normal household diet with iron tablets. In September 2001, a screener study was carried out to select the households and obtain baseline information for the study sample. Over 37,000 individuals in 9,500 households were screened for hemoglobin deficiency by pricking their fingers to obtain the blood required for the test. All screened persons, i.e., those for whom hemoglobin levels were established, were pooled and an experimental sample randomly drawn from them. Since benefits of iron supplementation accrue mainly to iron-deficient persons, males age 25-75 and females age 40-75 with low hemoglobin levels were over-sampled, generating sample of over 17,500 persons living in 4,300 households.

Once an individual was selected, his or her entire household was included in the sample so that randomization to treatment and control groups was at the household level. Adults in treatment households received 120 mg of purple-colored iron tablets and children age 5 years or less received orange flavored syrups while counterpart individuals in control households received placebos of the same appearance. These tablets and placebos were taken once a week, over a period of one year. Randomization at the household level averted the sharing of tablets among members of the same household, and avoided the necessity of keeping records of who in the household took what tablets. Randomly assigning a study sample (which need not be random) to treatment and control groups is the decisive step in a field experiment (see Duflo and Kremer, 2007).

The pre-baseline survey was conducted between January and April 2002, four months before the baseline study in May 2002. Subsequently, households were interviewed every four months to record changes in socio-demographics, health status, work and leisure activities. The intervention commenced in August 2002 and ended in December 2003, but the four-month interviews continued through December 2004, with the final interview for all households taking place in 2005. Attrition from the study sample was minimal, and compliance with treatment instructions was high. See Thomas et al.(2006, pp.12-15) for details of the experiment.

Table 2a shows the effect of treatment on health status, measured by the difference in hemoglobin level between the treatments and controls, 12 months into the intervention. The table shows the intent-to-treat-effects, i.e., it compares hemoglobin levels among individuals assigned to the treatment group relative to hemoglobin levels among persons assigned to the control group. Focus is on effect of intent-to-treat because it represents the outcome of the experiment when everyone fully complies with treatment instructions.

In this study, compliance rate for treatment group as well as for the control group was 92 percent. Division of the intent-to-treat effect (obtained via differencing) with the compliance rate for the treatment group yields the average effect of treatment on the treated (see Ravallion, 2007). If the compliance rate is equal to one, the intent-to-treat effect is the same as the average effect of treatment on the treated (TT). If experimental data are available, the TT, the average treatment effect (ATE), the local average treatment effect (LATE) and the marginal treatment effect (MTE) can all be derived using local IV regression methods and the control function approach (see Wooldridge, 1997; Heckman and Vytlacil, 2000; Card, 2001; Todd, 2007). Tables 2a and 2b illustrate the simplicity of computing TT when implementation of a randomized experiment is almost ideal, as in Thomas et al. (2006).

Table 2a
Selected Results for Hemoglobin Status: Intent to Treat Effects, Indonesia
(Standard Errors in Brackets)

Indicator	Sample	Status at 8 Months		Diff (T-C) (3)	Diff at baseline, 4 Months before T) (T-C) (4)	8 Months *minus* Baseline (Diff-in Diff) (5)	Adj. Diff-in-Diff (6)
		Treatment (T) (1)	Control (C) (2)				
Hemoglobin	Male	13.250* [0.040]	13.127* [0.040]	0.123* [0.057]	-0.059 [0.059]	0.183* [0.057]	0.181* [0.057]
	Female	11.974* [0.033]	11.819* [0.033]	0.156* [0.046]	.040* [0.048]	0.116* [0.048]	.117* [0.048]
Hemoglobin (<12g/dl) (Proportion)	Male	0.179* [0.009]	0.206* [0.009]	-0.027* [.013]	-0.004 [0.014]	-0.023 [0.015]	-0.023 [0.015]
	Female	0.461* [0.011]	0.499* [0.011]	-0.038* [0.016]	-0.024 [0.016]	-0.014 [0.019]	-0.014 (0.014)
Sample size	Male	1804	1759	3563	3563	3563	3563
	Female	2021	2042	4063	4063	4063	4063

Notes: Column (3) = (1)-(2); (5) =(3)-(4); (6) adjusts for age effects; * = statistically significant.
Source: Thomas et al (2006, Table 3).

In the first panel of Table 2a, columns (1) and (2) report Hb at eight months into the study for treatment and control groups, respectively. The difference in hemoglobin levels between these two groups is in column (3), while column (4) reports the baseline difference, i.e., the pre-existing difference, prior to the start of the intervention. Column (5) presents the Hb difference-in-difference between the treatments and the controls after the intervention, i.e, the Hb difference at eighth month of the intervention between the two groups, minus the corresponding difference at baseline. In column (6) the estimates are adjusted for differences in ages of the subjects.

Column (6) shows that prior to the intervention, the hemoglobin levels for the treatments and the controls were about the same for males (diff = -0.059; se = 0.059), which is as it should be, because the two groups had been randomly selected from the screened population, and randomly assigned to receive iron-tablets or placebos. Column (3) shows that by the eighth month into the intervention, hemoglobin level of the treatment group among males exceeded that of the control group by 0.123 g/dl, a difference that was statistically significant (se = 0.057). This is the first glimpse of evidence that the iron tables were effective in fighting anemia.

However, this is not yet the intent-to-treat effect of the intervention, because the pre-existing differences among subjects have not been taken into account. Column (5) does this by subtracting the difference in Hb levels for the two groups at the start of the intervention (column 4) from the difference in Hb levels at the eighth month (column 3) to yield an intent-to-treat effect of 0.183 g/dl for males, which is statistically significant (se = 0.057). In other words, since the Hb for the treatments (T) was 0.059 g/dl lower than that for the controls (C) at baseline, this amount must be added to 0.123 g/dl (the excess of Hb for T over C at the eighth month) to obtain the full amount by which iron tablets increased hemoglobin in the male sub-sample. Similarly, in the female sub-sample, where the reverse situation prevailed at baseline, 0.040 g/dl (column 4) is subtracted from 0.156 g/dl (column 3) to yield 0.116 g/dl (se = 0.048). In column (6) an adjustment for age differences in Hb response to treatment leaves the estimate in column (5) practically unchanged for both sub-samples.

Biomedical evidence suggests that subjects with low HB levels would gain most from consumption of iron tablets (Thomas et al., 2006). If this is so, the intent-to-treat effects shown in columns (5) and (6) for both sexes are understated. This hypothesis can be tested by subtracting from column (3), the difference in column (4) for subjects with low Hb prior to the start of the intervention (i.e., Hb < 12 g/dl at the baseline survey). For this group, Thomas et al. (2006, Table 3) report large Hb gains for both sexes (0.4 g/dl for males and 0.2 g/dl for females). The conclusion from the first panel of Table 2b is that the iron-supplementation increased Hb levels in the study sample, with males gaining substantially more than females.

The second panel of Table 2b depicts the effects of the intervention among iron-deficient subjects (i.e., those with HB < 12 g/dl at baseline). The results show that 20% of males and 50% of females (column 2) were iron-deficient at the start of the intervention. Eight months into the intervention, the prevalence of iron-deficiency among males had dropped by 2.7% compared with a drop of 3.8% among females (column 3). However, the adjustment for pre-existing differences between treatment and control groups shows that by the eighth month of the intervention, the prevalence of iron-deficiency in the study sample had not changed (columns 5 and 6). That is, although the hemoglobin level of the sample had increased, it was still below the cut-off point of 12 g/dl for both sexes.

Table 2b shows the intent-to-treat effects of better health (improvement in Hb level) on selected measures of economic prosperity after eight months of iron-supplementation. Columns (1) and (2) of Table 2b parallel columns (5) and (6) of Table 2a. Specifically, column (1) reports the change in each labor market outcome for the treatment group between the eighth month of the intervention and the time prior to the start of the intervention (the baseline), relative to the change in the same outcome for the control group. The effects computed were mainly generated during the first four months of the intervention because hemoglobin levels take time to build up. Moreover, the intent-to-treat effects are calculated for subjects with low Hb levels at baseline (Hb < 12.5 g/dl), under the assumption that the subjects were the ones most likely to benefit from iron-supplemented diets. Similarly, column (2) reports effects for persons with high Hb levels at baseline (Hb > 12.5 g/dl), which are mostly negligible, as expected. Panel 1 shows that among low Hb persons, probability of working during the eighth month of the intervention had increased by 3.6% for males, with the increase for females being smaller and statistically

insignificant (column 1). As expected, working probabilities for persons with baseline Hb levels greater than 21.5 g/dl did not change (column 2).

Table 2b
Intent-to-treat Effects on Work, Earnings and Hours Worked by Hemoglobin Status at Baseline, Indonesia (Standard Errors in Brackets)

Indicator	Sample	Change in Outcome for Treatment Group *Minus* Change in Outcome for Control Group	
		If Low Hb at Baseline (DinD) (1)	If High Hb at Baseline (DinD) (2)
1. Pr(not working in Month of Survey Interview)	Male	-0.036* [0.012]	-0.003 [0.007]
	Female	-0.020* [0.014]	0.029 [0.020]
2. Quartic Root Earnings (Rp 000) Last 4 Months	Male	0.576 [0.299]	-0.012 [0.173]
	Female	0.163 [0.091]	0.033 [0.127]
3. Hours Spent Working (Last 4 Months)	Male	-12.968 [36.368]	-144.185 [21.027]
	Female	9.644 [15.264]	30.137 [21.425]
4. Quartic Root Hourly Earnings (Rp 000) (Last 4 Months)	Male	0.126* [0.066]	0.007 [0.038]
	Female	0.034 [0.025]	-0.009 [0.035]
5. Quartic Earnings (Rp 000) if Self-Employed (Last 4 Months)	Male	0.113* [0.040]	-0.006 [0.040]
	Female	0.056 [0.026]	-0.21 [0.037]

Source: Thomas et al. (2006, Table 5); * = statistically significant.

Panels (2) through (5) report similar effects for wage earnings, hours spent working, hourly earnings, and earnings from self-employment. The striking findings here relate to effects of better health on hours worked and on hourly earnings. The number of hours worked remained

the same for both sexes (because the changes shown in panel 1 are statistically insignificant). Thus, the increase in total earnings for males shown in panel (2), is due to the increase in earnings per hour (panel 4). Although the females experienced an increase in total earnings, the increase in their hourly wage is statistically insignificant. Increases in male earnings among the self-employed substantially dominate the increases in female earnings (see Thomas et al. 2006).

<u>Kenyan Study</u>

Miguel and Kremer (2004) report on results of a field experiment, conducted in Kenya to investigate effects of deworming on pupils' health, school attendance and test scores. Their findings show that deworming increased school attendance through better health, but had no effect on pupils' test scores.

The experiment, known as the Primary School Deworming Project (PSDP), was implemented in western Kenya between 1997 and 2001 in an area with high prevalence of helminth infections – Hookworms, Roundworms, Whipworms and Schistosomiasis. Practically all primary schools in the project site (75 schools), with a total enrolment of about 30,000 pupils were sampled. The seventy-five PSDP schools were randomly divided into three groups of 25 schools each, and randomly assigned to a treatment or control status, with randomization taking place all at one time. The schools were first stratified by geographic zone, then listed alphabetically, and then counted off 25 times in the order 1-2-3, 1-2-3, etc. The "1"s became the Group 1 schools, the "2"s became the Group 2 schools, and the "3"s became Group 3 schools. Group 1 schools were treated in 1998 while Group 2 schools were treated in 1999.[11]

Thus in 1998, Group 1 schools were the treatment group, and Groups 2 and 3 were the comparison schools. In 1999, when Group 2 schools were phased into treatment, Groups 1 and 2 schools became the treatment group, and Group 3 schools, which did not begin treatment until 2001, were the control group. This experimental design had the advantage of exposing the whole population of schools to treatment over time in a random fashion.

The consent of the community and parents was sought before children were given deworming drugs. It is important to stress that although treatment was at the individual level, randomization was at the school level. That is, the unit of observation was a school rather than a pupil. Randomization at the school level permitted estimation of the following direct and indirect effects of treatment: (a) the direct effect of deworming on infection rate among treated and untreated children within a school; (b) the cross effect of the schools treated in 1998 on the infection rate among children in untreated schools in 1999; (c) the cross effect of treated schools on infection rate among children in the nearby schools; (d) the direct effect of treated pupils on their own infection rates within a school in subsequent periods.

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[11] I am very grateful to Edward Miguel for clarifying, in an email communication, the randomization of schools into treatment and control groups.

In addition to receiving drug treatment, the children and teachers were taught how to prevent worm infections. However, no worm prevention behaviors were observed among children in either the treatment or control schools. As a result, Miguel and Kremer (2004, p. 175) concluded that the health effects of the PSDP were through helminthic drugs rather than through health education.

Table 3a shows the status of infections, pupils' health, and worm prevention behaviors in treatment and control schools after one year of program activities. As can be seen from Panel A, worm infection rates were lower in the treatment group than in the control group, indicating that the program was generally effective against helminths. The differences in infection rates between treatment and control schools were statistically significant (last column of Table 3a). The prevalence of moderate-to-heavy infections in treatment schools (.27) was 25% lower than the prevalence in comparison schools (.52). Panel B shows that pupils' in treatment schools had better measures of general health than pupils in comparison schools. In particular, self-reported cases of sickness were 3-4% lower in treatment than in comparison schools.

Panel C shows that there was no difference between the treatment and control schools in behaviors associated with helminth-infection prevention, such as wearing shoes and general cleanliness. This finding shows that the health education messages given to children were not effective in inducing behavioral changes necessary to prevent worm infections, and is suggestive of the need to explore alternative mechanisms for doing so. One obvious reason for children not wearing shoes despite the associated benefits stressed by health education messages is that their parents could not afford to buy them shoes.

Table 3a
Health and Health Behavior Differences Between Treatment and Comparison Schools
(Standard Errors of Differences in Parentheses)

Variables	Treatment Schools (Group1)	Comparison Schools Group 2)	Treatment *minus* Comparison
Panel A: Helminth Infection rates			
Any moderate-heavy infection, January-March 1998 (baseline)	0.38	-	-
Any moderate-heavy infection, 1999	0.27	0.52	-.25* (0.06)
Hookworm moderate-heavy infection, 1999	0.06	0.22	-0.16* (0.03)
Roundworm moderate-heavy infection, 1999	0.09	0.24	-0.15* (0.04)
Schistosomiasis moderate-heavy infection, 1999	0.08	0.18	-0.10 (0.06)
Whipworm moderate-heavy infection, 1999	0.13	0.17	-0.04 (0.05)
Panel B: Other Nutrition and Health Outcomes			
Sick in the past week (self-reported), 1999	0.41	0.45	-0.04* (0.02)
Sick often (self-reported)	0.12	0.15	-0.03* (0.01)
Hemoglobin concentration (g/L)	124.8	123.2	1.6 (1.4)
Proportion anemic (Hb < 100g/L), 1999	0.02	0.04	-0.02* (0.01)
Panel C: Worm Prevention Behaviors			
Clean (observed by field worker), 1999	0.59	0.60	-0.01 (0.02)
Wears shoes (observed by field worker), 1999	0.24	0.26	-0.02

			(0.03)
Days contact with fresh water in past week (self-reported), 1999	2.4	2.2	0.2 (0.3)
Sample size information: Parasitological Results: 2328 (862 Treatment Group, 1467 Comparison Group); Hemoglobin Results: 778 (292 Treatment Group, 486 Comparison Group); Pupil Questionnaire Health Outcomes: 9,102 (3562 Treatment Group, 5540 Comparison Group).			

Notes: Moderate-to-heavy infection thresholds for the various intestinal helminths are: 250 egp for *S. Mansoni*, 5,000 for Roundworm; 750 epg for Hookworm; and 400 epg for Whipworm; * = statistically significant.
Source: Miguel and Kremer (2004, Table V).

Table 3b shows the effect of deworming on school attendance between the treatment and comparison schools over the period May 1998 and March 1999, approximately one year into the the intervention. As is clear from the table, school attendance increased significantly in treatment schools relative to comparison schools. Among young girls and all boys, school attendance in treatment schools exceeded the comparison schools by 9.3% [0.841-(0.731+0.767)/2] in the first phase of the project. Moreover, gains in school participation among the treated pupils were larger among boys than girls (8.8% and 7.6%, respectively).

The smaller gain in school participation (5.7%) among teenage girls is consistent with the fact that a small proportion of these girls received helminthic drugs (Miguel and Kremer, 2004, p. 190), and is indicative of a causal effect of deworming on school attendance. The causal effect of deworming on school participation is confirmed in the last column of table 3b, which shows that the difference in attendance rates in the two control schools (Groups 2 and 3) was not significantly different from zero at the 5% level.

Next, Miguel and Kremer (2004) investigate the effect of deworming on pupils' test scores in school examinations. Deworming was hypothesized to improve tests scores by increasing the total amount of time spent in school and by improving learning while pupils are in school. However, the study's hypothesis – that better health resulting from a reduction in parasitic infections would increase test scores was not supported by the data (Miguel and Kremer, 2004, Table X). Citing Strauss and Thomas (1998), Miguel and Kremer (p. 202) claim that there is an analogous result in the labor market literature where the impact of poor health on labor productivity is inconclusive. However, these two findings are not comparable because the labor market result, where it exists (see Section 4.4), relies on separability of production and labor supply (Singh et al., 1986) so that the work of sick family members can effectively be done by hired labor. In the present context of a test score (an approximate indicator of the output of an education production function, where child health is an exogenously varied input) this is not the case. That is, labor supply (school participation) is not separable from acquisition of cognitive skills (production of education human capital). The most likely reason for the reported test score result is that the experimentally induced change in school participation rate was not large enough

to produce an effect, or it was not well measured – issues that Miguel and Kremer discuss in detail.

Table 3b

Effects of Deworming (via Better Health) on School Participation, School Level Data

Variables	Group1 (25schools)	Group 2 (25Schools)	Group 3 (25 Schools)	Differences	
	Treatment	*Comparison*	*Comparison*	Group 1- (Groups 2 & 3)	Group 2- (Group 3)
First year post-treatment (May 1998 to March 1999)					
Girls < 13 years, and all boys	0.841	0.731	0.767	0.093* (0.031)	-0.037) (0.036)
Females	0.855	0.771	0.789	0.076* (0.027)	-0.018 (0.032)
Males	0.844	0.736	0.780	0.088* (0.031)	-0.044 (0.037)
Girls >=13 years	0.864	0.803	0.811	0.057* (0.029)	-0.008 (0.034)

Notes:* = statistically significant.
Source: Miguel and Kremer (2004, Table VIII, Panel A).

The differences in health status (Table 3a) and in school attendance rates (Table 3b) between the treatment and control schools exclude the externalities and long-run effects of deworming. Miguel and Kremer illustrate the strength of combining experimental designs with regression methods in their identification of within-school externalities. "Although randomization across schools makes it possible to experimentally identify both the overall program effect and the cross-school externalities, we must rely on non-experimental methods to decompose the effect on treated schools into a direct effect and within-school externality effect"(Miguel and Kremer, p. 175).

In addition to using the regression method as a decomposition tool, Miguel and Kremer also use it to estimate direct and externality effects of deworming on health and school participation controlling for school and pupil characteristics. As to externality effects, each one thousand pupils in treatment schools that were 3 kilometers away from untreated schools reduced moderate to-heavy infections in the untreated schools by 26%. Similar externality effects were calculated for school participation rates (see Miguel and Kremer, 2004, Tables VII and IX).

Todd (2007) presents econometric methods for estimating causal effects when data from randomized experiments are not available, or when such data suffer from sample selection, attrition and other problems.

<u>Mexican Study</u>

An evaluation of health effects of a large, nationally representative experiment in Mexico, known as *Progresa*, is presented in Gertler (2000, 2004). Like the Kenyan and Indonesian experiments above, Progresa was demand-oriented. That is, it provided subsidies which were administratively targeted to particular groups (the poor) within communities. This is in contrast to supply-driven approaches, that transfer increased amounts of resources to communities, e.g. in form of clinics.

The program was started in 1997 by the Government of Mexico to address the problem of extreme poverty in rural communities. Within three years of implementation, Progresa extended benefits to about 2.6 families in 50,000 villages, comprising 40% of rural families. However, the villages were phased into the program randomly due to budgetary and logistical constraints as in the Kenyan study above. The government chose 320 treatment and 186 control villages, a total of 506 study villages, during the first phase of the program. The treatment villages began receiving benefits in August 1998, while the eligible households in the control villages waited for two years before receiving benefits. Although Progresa benefits were targeted to households, the program was randomized at the village level. The structure, objectives and implementation of *Progresa*, i.e., a program on education, health and nutrition (see Ravallion, 2007) and similar programs in Latin America are described in Parker et al.(2007).

In an effort to improve health in rural communities, Progresa made cash transfers worth 20-30% of family income every two months to mothers of participating families in treatment villages if one or more of the following conditions held (see Gertler, 2004, p. 336-337).

(i) Children of age 0-23 visited immunization and nutrition monitoring clinics;
(ii) Children age 24-60 months attended nutrition monitoring clinics;
(iii) Pregnant women visited clinics to obtain prenatal care;
(iv) Lactating women visited clinics to obtain postpartum care, nutrition supplements and education about health, nutrition and hygiene;
(v) Other family members visited clinics for physical check-ups;
(vi) Adult family members participated in regular meetings at which health, hygiene, and nutrition issues and best practices were discussed.

Gertler (2004) evaluated consequences of Progresa on child health by comparing health status of children between the treatment and control villages. The technical problem was the estimation a child health production function, the market inputs of which included immunizations, nutrition supplements, medical care, and a set of behavioral inputs such as breast feeding and other best practices, e.g., avoidance of alcohol consumption and smoking during pregnancy (see Rosenzweig and Schultz, 1983). Items (i)-(vi) above provide a flavor of the types of inputs that belong in a child health production function; the inputs however, are endogenous to child health.

The inputs into child health were varied exogenously by randomization of Progresa's benefits across treatment and control villages. This was the first stage in the evaluation, accomplished by the Mexican Government at the design and start of Progresa. It is worthwhile to contrast the Mexican study with the Indonesian and Kenyan studies, where researchers had to implement their own randomized designs to permit identification of impacts.

The second stage in the evaluation of the impact of Progresa on child health is the measurement of the change in health status of children following implementation of the program, which can be accomplished through a simple calculation. "The randomization and the fact the control and treatment samples are well balanced in characteristics imply that a simple comparison of mean outcomes post-intervention will likely provide an unbiased estimate of program impacts" (Gertler, 2004, p. 338). However, to better account for effects of heterogeneity among households, regression methods were used. The post-intervention data were collected through a household survey from a subset of the original 506 experimental communities (320 treatment villages and 186 control villages).

The dependent variables in the regressions were (a) illness (whether child was reported ill four weeks prior to the household survey); (b) stunting (defined as being two or more standard deviations below the age-sex standardized height of a health reference population); and anemia (defined as hemoglobin less than 11 g/dl). The controls in the regressions included family background variables and village dummies (Gertler, 2004, pp. 338-9).

The evaluation results are in Tables 4a and 4b, which show, respectively, the baseline descriptive statistics, and the effect of *Progresa* on the odds ratio of illness (probability of being ill if in treatment villages, divided by probability of being ill if in control villages) in a sample of children. Since the sample is random, the results from the experiment apply to all rural children in Mexico.

The last column of Table 4a shows that at baseline, all of the characteristics of children were statistically indistinguishable between treatment and control villages. Also notable, is the fact that at baseline in 1997, the parents of children had very low levels of education, 3-4 years of schooling. However, both the control and treatment villages seem to have had good access to social infrastructure at baseline because about 70% of households in which the children lived had electricity.

**Pre-intervention Descriptive Statistics for Sample of Children,
Age 0-35 months at Baseline, 1997**

Variable	Treatment	Control	*p*-value for difference
Child was ill in the last 4 weeks (=1)	0.330	0.323	0.771
Age (months)	1.625	1.612	0.914
Male (=1)	0.511	0.491	0.091
Father's years of education	3.803	3.840	0.980
Mother's years of education	3.495	3.829	0.062
Father speaks Spanish (=1)	0.942	0.929	0.276
Mother speaks Spanish (=1)	0.935	0.917	0.443
Own house (=1)	0.93	0.917	0.465
House has electricity (=1)	0.644	0.711	0.091
Hectares of land owned	0.809	0.791	0.553
Male daily wage rate (pesos)	30.483	31.219	0.370
Female daily wage rate (pesos)	27.258	27.844	0.493
Sample size	4,519	3,306	

Source: Gertler (2004, p. 339, Table 1).

Table 4b reports estimates of the impact of Progresa on the odds ratio of illness in a combined sample of children from treatment and control villages. The odds ratio of an illness is equal to 1, if the probability of illness in treatment villages is equal to probability of illness in control villages. Thus, if the odds ratio is equal to 1, the program has no effect on probability of being ill.

The first two columns of Table 4b show the estimated odds ratio from logistic regression estimates of the coefficients on dummy variables indicating whether the child was in a treatment village and also eligible to receive Progresa benefits. The third column reports results for the effect of length of time that the child could have been in Progresa on odds ratio. The results

strongly reject the hypothesis that Progresa had no effect on illness probabilities in treatment villages.

Table 4b

Estimates of Health Impacts of *Progresa*: Dependent Variable is Children's Odds Ratio of Illness (the *p*-value for the Test of the Hypothesis that the Estimated Odds Ratio is Equal to 1 is in Parentheses)

Variable	Newborns	Child Age 0-35 Months at Baseline	
		Model 1	Model 2
Progresa eligible (=1)	0.747 (0.013)	0.777 (0.000)	
Progresa eligible for 2 months (=1)			0.940 (0.240)
Progresa elogible for 8 months (=1)			0.749 (0.000)
Progresa eligible for 14 months (=1)			0.836 (0.005)
Progresa eligible for 20 months (=1)			0.605 (0.000)

Notes: Control variables include the socioeconomic variables reported in Table 41a, measured at baseline prior to intervention.
Source: Gertler (2004, Table 2).

The estimates indicate that the newborns in treatment villages were 25.3% less likely than their counterparts in the control villages to be reported as being ill in the previous month (odds ratio = 0.743, *p*-value =013).[12] Similarly, the second column of the table shows that the 0-3 year-olds in treatment villages were 22.3% less likely to be ill compared with the same age-group in control villages. The third column reports results for effects of duration of exposure to the program on odds ratio of illness. Two months into the program, illness probabilities were not different in treatment and control villages (odds ratio =0.940, *p*-value = 0.240). That is, children in treatment villages were 6% less likely to be reported ill, relative to those in the control villages, but this

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[12] Gertler (2004, p. 339) uses the term log-odds to refer to the estimates presented in Table 4b (his Table 2). Instead, we use the term odds ratio because an estimate such as 0.747 is simply the ratio of the probability of illness in treatment villages divided by probability of illness in control villages; however, the logarithm of 0.747 is the log-odds ratio, which is not required here.

difference was not statistically significant. However, 20 months into the program, the probability of illness in treatment villages was 39.5% lower than in comparison villages. A similar analysis shows that children in program areas were less likely to be stunted or anemic relative to children in control villages (see Gertlrer, 2004, Table 3, p. 340). In particular, treatment children were 25.3% less likely to be anemic and grew about a centimeter taller during the first year of the program.

Summary and discussion

The four studies above illustrate structural (IV) and experimental approaches to the measurement of benefits from health improvements, focusing on advantages of a particular approach in specific contexts. Glewwe and Miguel (2007) provide a detailed account of strengths and weaknesses of using IVs and randomized experiments to measure the impact of child health on education in low-income countries; see also Ravallion (2007).

Schultz's study in Ghana and Ivory Coast shows how IV methods can be applied to survey data to provide consistent estimates of returns to health investments. Miguel and Kremer 's study in Kenya illustrates how a deworming program, randomized at the school level, can be used to evaluate the impact of children's health on school attendance and on test scores in the presence of treatment externalities. The study by Thomas et al. in Indonesia shows how supplementation of household normal diet with iron tablets (randomized at the household level to avoid the sharing of tablets among treatment and control subjects from the same household), can be used as a source of exogenous variation in health, thus permitting measurement of its impact on earnings, productivity, and leisure. Gertler's study exploits exogenous variation in utilization of health care services across village (due to *Progresa*), to estimate the impact of the services on child health using regression methods. Progresa was randomized at the village level because of the broader geographic nature of some program benefits, such as improvements in local health facilities, and because it was perceived that randomization within a village would be politically difficult (see Behrman et al., 2005).

The outcomes of randomized social programs are dependent on compliance with various program requirements, and in this regard, Progresa's conditionality of cash transfer is noteworthy. The government transferred cash to families only after they had used the services availed by program, such as immunizations, health education and prenatal care. Becker's (1965) and Grossman's (1972a,b) models show that the opportunity cost of the time spent to seek health care can be a major deterrent to health care utilization, even when the money price of care is fully subsidized. Paying families to use basic health care, as in Progresa, recognizes this fact, and amounts to reimbursing families for the time cost of seeking care, which the very poor can hardly afford. Indeed, even without treatment externalities of the type documented by Miguel and Kremer (2004), such payment is justified on grounds of the opportunity cost of the time individuals must incur to seek and use health care. There is a strong case for extending subsidies to the poor to cover health care costs beyond the money cost of care.

4.6.   **HIV/AIDS and Development**

Introduction

The relationship between HIV/AIDS and development is a special case of the interdependence
between income and health (Section 4.4). The methods and concepts discussed in Section 4.4 can
be applied to investigate the relationship between HIV/AIDS and development. The term
development encompasses more than economic growth. It also includes progress in reducing
inequality and poverty and increasing the expected length of life.

HIV/AIDS has characteristics that distinguish it from many others diseases. It is pandemic,
chronic, fatal, and highly stigmatized. A virus called HIV (human immunodeficiency virus)
causes AIDS (acquired immune-deficiency syndrome) when it destroys white blood cells that are
essential to the disease fighting ability of the body (the immune system). In most low-income
countries, HIV among adults is heterosexually transmitted. After transmission, infected
individuals enter a clinical latent stage during which health status declines gradually without
signs of disease symptoms. In east Africa, the median time from infection to AIDS is 9.4 years
(Thirumurthy et al. 2005). Over time, the immune systems of almost all infected individuals
become too weakened to fight diseases. This period of immune deficiency is associated with
substantial weight loss, and opportunistic diseases such as cancers, pneumonia and tuberculosis.
In resource poor countries, individuals usually die within one year after progression to AIDS
(Thirumurthy et al., 2005). ARV (antiretroviral) therapy can reduce the likelihood of
opportunistic diseases and prolong life (WHO, 2004). However, to implement ARV therapies,
information is needed on health care demand behavior of individuals. Also important in the
context of HIV/AIDS control, is information on demand for VCT (voluntary counseling and
testing) services (see Glick and Sahn, 2006). Use of VCT services allows early detection of
AIDS, so that ARV therapy can be initiated before the HIV substantially weakens the immune
system. The results of VCT may also motivate behavioral change to prevent further transmission
of the virus. Individuals who know their HIV status have an incentive to change sexual behavior
to avoid infection or exposure of others to the virus.

The world profile of HIV/AIDS

Since the reporting of the first five cases of HIV/AIDS on June 5, 1981 in Los Angeles 25 years
ago, the disease has reached virtually every corner of the globe, infecting more than 65 million
people, of whom 25 million have died (Fauci, 2006). The world profile of HIV/AIDS (Bloom
and Sachs, 1998; World Bank, 2005) shows that the poorest regions in the world have the
highest incidence of HIV/AIDS. Africa for example bears the burden of two-thirds of AIDS
deaths worldwide, and about three quarters of new HIV/AIDS infections. Furthermore, for every
8 people living with HIV/AIDS worldwide 5 are in Africa (Table 5).

Table 5

**Estimates of HIV Infections and AIDS Mortality by Region
as of December 2004**

Region	Persons Living with HIV/AIDS	Number of New Infections in 2004	Number of AIDS Deaths in 2004
Sub-Saharan Africa	25.4 million	3.1 million	2.3 million
South and Southeast Asia	7.1 million	890,000	490,000
Latin America and Caribbean	2.1 million	293,000	131,000
Eastern Europe and Central Asia	1.4 million	210,000	60,000
East Asia	1.1 million	290,000	51,000
Middle East and North Africa	0.5 million	92,000	28,000
North America, Western Europe and Oceania	1.6 million	70,000	23,200
Total	39.4 million	4.9 million	3.1 million

Source: World Bank (2005, p. 5).

Although the social and economic losses associated with HIV/AIDS have so far been limited mainly to Africa, the epidemic is in a nascent stage in other developing regions of the world, notably Asia (World Bank, 2005). If policy measures are not taken to stop the spread of HIV/AIDS in Asia, the worldwide regional HIV/AIDS profile will, within several decades, mimic the current world poverty profile. That is, the highest incidence of HIV/AIDS will continue to be in Africa, but the largest number of HIV/AIDS deaths and infections will occur in Asia because of that continent's large share in the world population. Thus, while implementation of extensive prevention and treatment programs are required in Africa to control HIV/AIDS, widespread prevention measures and focused treatment campaigns are required in Asia to stop the emergence of the epidemic there on the scale observed in Africa.

Effects of HIV/AIDS on growth

The evidence in table 2, that poor regions in the world have high HIV/AIDS prevalence can be interpreted to mean that HIV/AIDS causes poverty as intuition suggests. There is a large literature that examines the causal effects of HIV/AIDS on macroeconomic growth (Kambou et al., 1992; Cuddington, 1993; Bloom and Mahal, 1997; Arndt and Lewis, 2000). Many

macroeconomic studies find small negative effects of HIV/AIDS on growth (see e.g., Bloom and Mahal, 1997), while a few report large negative effects (see McDonald and Roberts, 2006; Roe and Smith, 2006). A positive long-run growth effect of HIV/AIDS has been simulated for South African economy (Young, 2005).

HIV/AIDS reduces growth by increasing depreciation of health capital and therefore reducing life expectancy (Grossman, 1972a). This foreshortening of life expectancy undermines individuals' incentives to accumulate education human capital. Thus, HIV/AIDS may cause poverty by reducing both the health and education human capital, the two key determinants of income (Couderc and Ventelou, 2005). However, the positive relationship from poverty to HIV/AIDS is not causal, because a virus is the reason for the disease. The positive relationship from poverty to AIDS reflects the correlation between poverty and a cluster of behavioral patterns that put individuals at high risks of infection by human immunodeficiency viruses. For instance, since use of condoms among the poor is limited, the virus and the associated sexually transmitted diseases spread rapidly among the poor.

Policies to control HIV/AIDS
To design effective AIDS prevention strategies, a good understanding of the dominant modes through which HIV is transmitted is needed (Feachem and Jamison, 1991; World Bank, 1997). Globally, HIV is spread through (a) unprotected sex with an infected partner, (b) the sharing of infected injection equipment, (c) childbirth and breast-feeding (mother-to-child transmission), (d) transfusion of contaminated blood and blood products and through (e) health facilities that do not take precautions to protect their patients and staff against HIV (World Bank, 2005, p. 5). Since all these correlates of AIDS are behavioral in nature, information on demand for behavioral health inputs is critical in the design of policies to prevent the epidemic and treat the infected persons.

Acquisition of information about own HIV status can help slow down transmission of HIV by motivating infected individuals to seek early antiretroviral (ARV) treatment. Early uptake of ARV considerably reduces the intensity of HIV infection. The effectiveness of ARV therapy when received before the CD4 count drops below a threshold level has recently been demonstrated in Kenya. AIDS patients who received ARV therapy when their CD4 count was too low (below 35) had small survival probabilities but those who began treatment earlier experienced rapid recovery (Thirumurthy et al. (2005).

Provision of formal and informal education could increase VCT for expectant mothers and increase the uptake of ARVs that reduce the mother-to-child transmission rate. Nevirapine, a relatively low cost ARV drug, can be briefly administered to a mother at the onset of labor and to her child at the time of delivery to reduce the likelihood of transmission of HIV from mother to child by a substantial amount (see Canning, 2006). But to achieve this low-cost reduction in transmission, a pregnant woman must undergo VCT and decide to have this locally provided therapy. Without information on her HIV infection status, a woman may not consider the pros and cons of this intervention. In the last few years, Nevirapine has become more widely available in Africa and elsewhere. But the results of routine testing for HIV at prenatal clinics and birthing hospitals have been treated as confidential information, and the results of testing are

provided only to women who request them. Many women are not informed of the likely benefits which they could obtain for their child and their own future health if the test determined they were infected by HIV. With the recent decline in the cost of ARV drugs and the growing capacity of health care systems to dispense them, it is important to design new educational programs which publicized the private benefits of testing and of timely treatment of HIV/AIDS, and evaluate the effects of these programs on prevention behavior and on transmission of the epidemic.

A desired change in demand for behavioral health inputs can be achieved through public subsidization of such inputs. In particular, demand for VCT services can be increased through subsidization of VCT visits or health education programs. However, even when services at voluntary counseling and testing centers (VCT) are provided free of charge, time cost and lack of appropriate information can be a major barrier to using them (see Gersovitz, 2005). Thus, in addition to reducing financial and time costs of making VCT visits, information about the value of such visits need to be generated and disseminated widely. Policies for controlling HIV/AIDS in low-income countries should include making basic education widely available to the youth, age 15-19.. Ainsworth et al. (1996) show that schooling is positively correlated with demand for contraceptives in Africa, behavior that is likely to be negatively correlated with the risk of exposure to HIV. There is some evidence that educated persons are more likely to be tested for HIV than persons without any schooling (Gersovitz, 2005).

Support for fighting HIV/AIDS should be given to projects that have the greatest potential for reducing the scale and effect of the epidemic, relying on locally generated information to design, implement and manage them (World Bank, 2005). The material in this sub-section complements this view because it shows the types of health inputs that can be made available by the projects to reduce the transmission of HIV/AIDS and to mitigate its health and economic impacts within the infected population. Supporting community-based organizations and the health ministries to deliver cost-effective treatment, preventive, counseling and testing services to populations, as suggested by the above evaluation, would go along way in effectively attacking the epidemic. However, as emphasized in this paper, care should be taken to design projects that address demand-side as well as the supply-side barriers to health care utilization, paying attention to the linkage between utilization and health outcomes. At the policy level, the link between health outcomes and the services for HIV/AIDS prevention, treatment, and for the care and support of the persons living with HIV/AIDS, can be made by periodic monitoring and evaluation of the effectiveness of the projects, especially in improving the health status of the poor.

4.7. **Industrial Organization of Health Care**

The nature of health care industry determines the cost of medical care, its quality and accessibility by the population.[13] In developed countries, private methods of health service

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[13] An analysis of industrial organization "focuses on individual, imperfectly competitive markets and seeks to understand the behavior of firms that compose them and the resulting efficiency, i.e, performance of those markets" (Dranove and Satterthwaite, 2000, p. 1095).

financing and provision include employer-based health insurance schemes, medical group practices, and a variety of not-for-profit health care providers, commonly known as health maintenance organizations. The essential feature of these privately organized health care entities is that they operate mainly through a variety of markets. Prominent among these, are markets for health care, pharmaceuticals, drugs, and medical supplies and equipment. Industrial networks of health care firms (health care providers and payers for the services provided) are supported by an elaborate system of social institutions such as the laws relating to medical insurance, public health, contracts, patents, development of pharmaceutical products, and training of health professionals.

In contrast to the above case, health care in low-income countries is directly provided to the population with financing from general taxation. In a few countries, government-provided health insurance exists for certain categories of public sector employees. However, efforts to implement large-scale, national health insurance programs in low-income countries, especially in Africa, have met with great difficulties (see World Bank, 1993).

The industrial organization of health care in developed countries is analyzed in Parts 5 and 6 of the *Handbook of Health Economics* (Culyer and Newhouse, 2000). In their study of industrial organization of health care in the United States, Dranove and Satterthwaite (2000) limit themselves to topics such as agency, decision-making under uncertainty, non-price competition, market entry and exit, and product differentiation. The *Handbook of Industrial Organization* (Schmalensee and Willig, 1989) provides examples of the range of similar and parallel topics that are usually studied under the general subject of industrial organization. These topics include determinants of firm and market organization, transactions costs, collective bargaining, price discrimination, politics of regulation, and the environment.

Dranove and Satterthwaite (2000) note that health care markets fail to satisfy the key requirements of a perfect market, namely, large numbers of consumers and firms; free entry and exit; marketability of all goods and services including risk; symmetric information with zero search costs; and no increasing returns, externalities or collusion. Thus, while the perfect market model, may still serve as benchmark for optimal performance of health care markets, it cannot be used to illuminate how these markets actually function. Instead, a monopolistically competitive model has been recommended for that purpose (Stiglitz, 1989; McGuire, 2000). Dranove and Satterthwaite analyze health care market structures (provision and financing of health services) in contexts of independent and regulated physicians and under managed care insurance plans. They show that health care quantity, quality, price, and outcome vary across different market structures and industrial organizations (see also Rizzo, 2006). For example, health care prices are lower under managed care health insurance plans (where large groups of health care payers negotiate price discounts for treatments on behalf of their clients) than under settings of independent hospitals or physicians (where patients undertake such price negotiations individually). In the United States, managed care has become a dominant mode of organizational form of health care because it offers payers and providers alike an incentive to be cost conscious (Glied, 2000; Cuellar and Gertler, 2006). Managed care health insurance plans (health care payers such as a network of insurance firms) have an incentive to reduce the costs that patients pay for health care to limit the insurance benefits paid out, while health care providers (hospitals,

physicians or health maintenance organizations) have an incentive to reduce health care costs to attract more patients. However, there is evidence that in United States, health care providers such as hospitals and physicians have formed consortia to neutralize the price reducing power of managed care (Cuellar and Gertler, 2006).

In industrialized countries, government is one of the payers for health care that patients obtain from the market (Chalkley and Malcomson, 2000). Thus, types of contracts that ensure that patients get quality health care is a major concern in the industrial organization of health care in such countries. In low-income countries, the government simply provides health care directly to patients free of charge or at a nominal fee, although in many settings private patients make side payments to providers to receive services and drugs or to avoid waiting; or to receive better quality care (see Banerjee et al., 2004). In these countries, private health insurance is limited, and the national health insurance has not taken root. However, fragmentary schemes of community-based health insurance exist (Schneider and Hanson, 2006).

The industrial organization of health care in low-income countries is in formative stages because the social institutions conducive to its emergence and growth are lacking. However, since developing countries are globalizing at a rapid rate, the policymakers in these economies may soon face issues of managed care in private health sectors. Already, managed care is a notable feature of the private health sector in South Africa (Van den Heever, 1998). Readers interested in managed care and related issues may consult Culyer and Newhouse (2000), McGuire and Riordan (1994) or the general industrial organization literature (Schmalensee and Willig, 1989).

5. **CONCLUSION**

This paper has reviewed a large literature on economic methods and concepts that can be used to effectively address health policy issues in low-income countries. Health policy-making in developing countries can be strengthened through a better understanding of five issues: (A) How can the health of the population, especially of the poor be improved? (B) What are the economic effects of investments in health and how do investments that increase income from various sources affect different dimensions of health? (C) What are the effects on health of demand for behavioral inputs such as smoking and personal hygiene and of demand for market inputs such as food, medical care, nutrition, and vaccinations? (D) How important are the intra-household allocations in influencing these demands? (E) What analytical work exists on HIV/AIDS that could be used to inform prevention and treatment of this disease in low-income economies?

A. Improving health outcomes
The review finds that developing country literature is largely silent on policies to improve health. This is because in this literature, health production is not properly linked to patterns of demand for market inputs and to behavioral changes. An integrated conceptual and empirical framework for analyzing policies that can be implemented to improve health (Rosenzweig and Schultz, 1983) has been presented in this paper and linked to related literatures. Despite estimation challenges, the framework can still be used with available data to illuminate health policy issues in low-income countries. In applying the framework, it is important to recognize that the inputs that go into health production are choices of economic agents, and thus the estimated effects of

the inputs are likely to be biased by preferences, and by unobserved components of health endowments and household constraints.

B. Economic effects of better health

The paper shows how health improvements affect wages, labor supply (number of hours worked and whether or not individuals enter the labor force), farm productivity (yields per hour) and fertility. The empirical evidence thus provided can guide investment of scarce resources in ways that best improve the economic well-being of the population through improvements in health. However, to avoid misguided policies, it is important to also consider the effect on income of health of individuals *not* exposed to health-improving interventions. See Duflo (2001, 2004) for examples of effects of education on wages and employment in Indonesia that go beyond the individuals exposed to schooling opportunities.

More generally, caution should be exercised in extrapolating micro level findings to the macro level because general equilibrium adjustments can undo the findings derived from a localized experiment. Increases in health human capital may not be accompanied by economic growth due to bad macro policies or corruption that misallocate or dissipate the additional capital.

C. Behavioral changes as health inputs

A behavioral change such as quitting smoking, changing a sexual practice or overcoming addiction to drugs, alcohol, or diet is associated with better health, just as is the use of market inputs such as medical care and vaccinations. Thus, a behavioral change serves as an input into health production. Indeed, health effects of changing behavioral inputs may be as important as effects of altering market inputs of medical care.

In the United States, approximately 50% of deaths in 1990 could be attributed to behavior-related health problems (McGinnis and Foege, 1993; Cutler and Glaeser, 2005). The low-income countries also suffer many diseases that are rooted in behavioral choices, and in psycho-social problems as in the US case analyzed by McGinnis and Foege. The paper has highlighted structural models and statistical methods for estimating without bias, the effects of behavioral changes and market inputs on health outcomes.

D. Intra-household distribution of health inputs

Equity in health outcomes is an important consideration in health care at the household and national levels. Health outcomes such as height, longevity, or the number of healthy days (Grossman, 1972a,b) cannot be re-distributed from one individual to another. However, market inputs into health production, such as medical services, drugs, vaccinations, food, housing, and clothing can be re-allocated to change the existing distribution of health outcomes. Society can improve equity in health outcomes by designing policies directed at changing the distribution of health inputs within households in a particular way. For example, policies that give control of child care resources to mothers can be implemented with good outcomes. An instance of this in the education sector, is a family law in Brazil that extended alimony rights to women living in informal marriages, with the consequence that the schooling of their children increased without altering the amount of family resources (Rangel, 2006). Specifically, the schooling of first born-

daughters of less educated women in informal marriages (whose bargaining power was strengthened the most by the law) increased compared with that of first-born daughters of mothers in formal marriages.

The paper has reviewed unitary and collective household models that may be used to evaluate such policies. If for example, the key assumption of the unitary model (income pooling) is valid, it is not necessary to place child care resources in the hands of mothers to improve child health. Effort instead, should go into increasing household income, without regard to the identity of the person controlling the additional income. However, if the main assumptions of collective models are accepted (Pareto efficiency or bargaining), and the unitary model is not maintained, policies that promote control of resources by particular household members may be needed to improve equity in health outcomes within households.

The main problem in choosing between the above alternative health policy designs is that unitary and collective models are empirically difficult to distinguish one from another. For example, a unitary household may still distribute a market input (e.g., food) like a collective household. In a unitary household, a healthy individual might get greater allocation of calories than an unhealthy individual on efficiency grounds (Pitt, Rosenzweig and Hassan, 1990). The same allocation pattern may also be observed in a collective household, because the assumption of Pareto efficient outcome of individualistic behavior guarantees that everyone in the household is better-off when greater allocations go to a healthy person.

The assumption that resource allocation decisions are Pareto efficient may be too strong for health care markets. The income pooling assumption is even stronger. The collective household models with a bargaining element do not rely on either the pooling or the efficiency assumption. They are more suited than the unitary or the general collective models to the analysis of intra-household distribution of a non-market outcome such as health status. Within a household, the bargained resource sharing rule seems to play the role of an internal market mechanism.

E. HIV/AIDS and Development
Previous research on this topic in developing countries has focused on effects of HIV/AIDS on macroeconomic performance. This line of research needs to be complemented by studies at the household level that provide information on demand for both behavioral and market inputs and their consequences. Behavioral changes can dramatically affect risks of exposure to HIV infection. Information on preventive behavior is critical to designing policies directed at the root cause of HIV transmission. In particular, information derived from demand for VCT services can be used to improve the timing of ARV therapies, to target prevention measures to individuals according to their HIV status, make treatment more effective in increasing the longevity and productivity of those living with AIDS.

Increasing longevity of HIV-parents, has the additional social benefit of reducing the number of AIDS orphans. A decline in the number of orphans in countries suffering from AIDS pandemic would reduce household and government expenditures on orphans and likely increase saving. Since life-prolonging ARVs are still unaffordable in many low-income countries, a dilemma may arise as to whether, in a program designed to reduce the number of AIDS-related orphans, ARVs

should be targeted to mothers or fathers. Although information on the impact of ARVs on survival probabilities by gender may help illuminate the efficiency issue involved here, it would not resolve the ethical dilemma. This example brings to light an instance of the limit of economic analysis in health care policy making.

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