THE IMPACT OF TUBERCULOSIS ON ECONOMIC GROWTH

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ABSTRACT

Tuberculosis remains one of the most devastating diseases in the world, affecting people of all ages across the globe. This paper reviews the economic literature dealing with the theoretical and empirical links between poor health and low productivity, both at an individual and national level. It then conducts panel data analysis covering 91 countries, using an augmented Solow growth model and notification data of tuberculosis incidence from 1981 to 2000. Taking into account other national characteristics it finds that countries with a lower burden of tuberculosis grew faster than those which were more heavily afflicted. Although tests of robustness suggest that some of this effect may be due to reverse causality, there remains a persistent effect of between 0.2 and 0.4 percent lower growth for every 10 percent higher incidence of tuberculosis. This corresponds to an annual loss of between US$ 1.4 and 2.8 billion in economic growth worldwide.

RÉSUMÉ

La tuberculose demeure une des maladies les plus dévastatrices au monde, affectant des personnes de tous âges à travers le globe. Cet article consiste en une revue de la littérature économique traitant des liens théoriques et empiriques entre une santé fragile et un bas niveau de productivité, tous deux au niveau individuel et national. Ensuite il mène une analyse de données panel regroupant 91 pays, utilisant le modèle augmenté de croissance de Solow ainsi que l'incidence de tuberculose provenant des données rapportées de 1981 à 2000. En prenant en considération d'autres caractéristiques nationales, il apparaît que les pays avec un plus bas niveau de tuberculose se développent plus rapidement que ceux étant affectés plus gravement. Même si les tests de robustesse suggèrent qu'une partie de cet effet pourrait être due à une causalité renversée, un effet persistant de croissance plus faible variant de 0.2 à 0.4 pour-cent demeure pour chaque 10 pour-cent d'incidence plus élevée de tuberculose. Cela correspond à une perte annuelle de 1.4 à 2.8 milliards de dollars américains en terme de croissance économique à travers le monde.

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1. INTRODUCTION[1]

Despite rumours of its demise, tuberculosis remains one of the most deadly, and disabling, diseases in the developing world. According to the World Health Organization's Global Burden of Disease project (Mathers 2002), in 2000 it was the eighth highest cause of death, and the tenth highest cause of disability adjusted life years (DALYs).[2] Still more disturbing is that unlike most other major diseases, with the notable exception of HIV/AIDS, its burden is spread across all age groups and is responsible for the deaths of many productive individuals in the core 15 to 44 age group.[3]

In addition to being a significant disease, tuberculosis is also increasingly a high profile issue. Its recent resurgence, due in part to the rise of multi-drug resistant (MDR) strains, and its intimate connection with HIV/AIDS in the developing world – it is often the first opportunistic infection to strike AIDS patients – have led to increased attention in medical and media circles. This is evidenced by the launch in January 2002 of the Global Fund to Fight AIDS, Tuberculosis and

[1] A glossary of acronyms used throughout this paper can be found in appendix 3.
[2] DALYs reflect the period of healthy life lost to illness, as measured by time incapacitated multiplied by the severity of incapacity, including premature mortality. Deaths from Mathers et al (2002) table 13, p 35; DALYs from Mathers et al (2002) table 18, p39.
[3] In 1990 it was the second greatest cause of death in this age group for men, and the leading cause of death among women. The only other infectious disease among the leading ten causes was HIV (Murray, Christopher JL and Lopez, AD (1996) Vol 1, table 3.13, p 182).

Malaria, a public-private partnership that aims to raise and direct funds towards the prevention and treatment of these three diseases.[4]

This funding drive was buttressed by academic research aimed at uncovering the costs and benefits to society of successfully reducing the world's disease burden. Much of this work came under the banner of the Commission on Macroeconomics and Health (CMH), a World Health Organization (WHO) study of the relationship between health, economic growth and poverty reduction, the key results of which are set out in section 2.7 below.[5]

In light of this and other efforts to raise expenditure on tuberculosis treatment it seems useful to study in detail the potential macroeconomic impact of a reduction in this disease, in order to justify both current outlays and potential future spending. While detailed studies of each strand of the disease are needed, this paper will focus on the overall burden of disease: is there a demonstrable effect of lower gross tuberculosis incidence on economic growth at the national, and therefore the worldwide, level.

The paper is set out as follows: the remainder of this section contains a brief summary of current tuberculosis incidence, known impact and treatment; section 2 reviews of the literature in the fields of economic growth modelling, health and productivity, and the likely impact of increased health spending on outcomes;

[4] For details, see http://www.globalfundatm.org.
[5] The final report of this commission is available at http://www.cmhealth.org or as Commission on Macroeconomics and Health (2001).

section 3 presents the methodology for this study and an outline of the data used; section 4 reports the results; and section 5 draws some tentative conclusions and makes suggestions for further work.

1.1 An overview of tuberculosis

Tuberculosis is a disease which is found throughout the world, although the overwhelming majority of cases are found in poorer countries. Although 1.86 billion people are infected with the tuberculosis bacillus,[6] rather fewer individuals actually become ill in a given year, the most recent estimate being 8.46 million cases in 2001.[7] Of these 6.77 million, or 80 percent of the total, occurred in just 22 high burden countries located primarily in South and East Asia and Sub-Saharan Africa.[8] While China and India between them account for some 40 percent of worldwide cases, the highest case rates are believed to be in Zimbabwe, South Africa, Kenya and Cambodia. Such estimates are uncertain since reporting of tuberculosis undershoots what is believed to be the true world burden by around half.[9]

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[6] Dye, Christopher, Scheele, S, Dolin, P, Pathania, V and Raviglione, MC (1999) p 681
[7] World Health Organization (2003) table 4, p 10
[8] Figures from World Health Organization (2003) table 4, p 10; a full list of these high-burden countries can be found in Appendix 1.
[9] In 2001 there were a total of 3.81 million reported cases of tuberculosis: see World Health Organization (2003) table 10, p 19.

Tuberculosis is caused by *Mycobacterium tuberculosis* and is spread largely through the coughing and sneezing of infectious patients.[10] Once infected around one in ten people develop the disease. The incubation period of the disease ranges from weeks to years, although it is rarely more than five years, and after this point any incidence is usually due to further contact with an infectious source. While tuberculosis can affect many areas of the body, by far the most common location is the lungs. Untreated pulmonary tuberculosis leads to death in between 60 and 70 percent of cases; however an uncomplicated infection, if treated by a course of oral chemotherapy has a very high success rate. In the developed world deaths from non-MDR- or HIV-TB are rare, and in the developing world an intensive, "short-course", treatment regime completed under supervision reduces fatality rates to around five percent.[11] Such treatment regimes have been found to have cost-effectiveness rates from a provider perspective as low as US$ 1-4 per DALY saved,[12] although this depends on the cost of a programme and the type of tuberculosis being treated: in a middle-income country with high HIV infection rates this figure is likely to be closer to

[10] Details for this paragraph are taken from Borgdorff, Martien W, Floyd, K and Broeckmans, JF (2001), which provides an excellent introduction to tuberculosis, treatment options, their efficacy and cost-effectiveness, and constraints to a broad "scaling up" of treatment, in particular DOTS (see next footnote).

[11] This is the WHO standardised practice called "Directly Observed Treatment, Short-Course" or DOTS and involves six to eight months of treatment with a combination of drugs. During the first two or three months on an intensive regimen is followed, which a health professional or other responsible individual watches patients self-administer to ensure compliance. This approach is intended to significantly reduce the incidence of MDR tuberculosis, which usually arises through partially completed chemotherapy regimens.

[12] Original figure from the 1993 World Development Report published by the World Bank; quoted in Borgdorff et al (2001) p 30.

US$ 40 per DALY.[13] While there are a number of other potential strategies for treating tuberculosis, including BCG vaccination in infancy, prophylactic chemotherapy and active case finding, they do not appear to be so cost-effective.

While this partial equilibrium approach is helpful in understanding the potential benefits of improved tuberculosis treatment in individual cases, a more global picture is also useful. Holger Sawert and colleagues (1997) take this approach in creating an epidemiological model to measure the costs and benefits of a comprehensive national tuberculosis treatment programme in Thailand. They find that over a twenty year period improved control of the disease through the use of DOTS should reduce by 45 percent the number of new cases arising each year. In direct provider costs this amounts to a saving of US$ 8.3 million over twenty years,[14] but the overwhelming gain from such a programme is a 32 percent reduction in DALY's lost to the disease, the benefit of which, costed at the current wage rate, would be US$ 2.4 billion. This is both a huge saving and an indication of the size of the current impact of the disease, even in a middle-income country such as Thailand.

A further aspect of tuberculosis treatment that is often overlooked is the cost of the disease to the individual. Although many nations offer nominally free

[13] This is because higher income countries also have higher staff costs for treatment, and because a treated HIV-TB case adds an average of two years to an individual's life, as opposed to around 40 years in the vase of a non-HIV positive patient.

[14] There are two factors driving provider costs here: an upward force due to a more comprehensive treatment programme, which is large in the early years of the programme, but declines over the years; and a downward one from a reduced caseload for treatment, which grows stronger as time passes, eventually becoming the dominant factor.

treatment of the disease, this is often not reflected in patients' experiences. R. A. Croft and R. P. Croft (1998) find in a brief survey of in a rural Bangladeshi village that loss of income, and out-of-pocket expenditures prior to reaching treatment, averaged US$ 245 – more than 30 percent of an average family's income. This was primarily due to medicine costs and time off work sick.

In a larger study P. Kamolratanakul and colleagues (1999) find that tuberculosis-related costs in Thailand were similar across different income groups, and thus most heavily felt among those living beneath the poverty line. For these individuals expenditures and lost income amounted to more than 20 percent of annual income. The likelihood of this reducing other investment activities can be seen in the fact that 16 percent of those living below the poverty line were forced to sell property, and a further 35 percent had to take loans, either from relatives or banks, to pay for their treatment.

In comparison to healthcare costs, loss of income, even amongst those who recover from tuberculosis, can be considerable. In a study of household costs in Tanzania, Kaspar Wyss, Peter Kilima and Nicolaus Lorenz (2001) find that the cost of the illness to families amounted to more than five times the cost to providers of treating the patient.[15] As all these studies demonstrate, an approach which considers only the benefits to the provider of reduced tuberculosis

[15] The median estimate for household costs was US$ 481, of which US$ 431 was lost productivity costs, while the median estimate for provider costs was US$ 90.

incidence would miss a crucial aspect of the benefits which accrue due to reduced disease burden.

Tuberculosis has long been a disease associated with poverty and poor public sanitation, and in the nineteenth and early twentieth centuries was a significant issue across what is today the developed world. This high incidence of disease dropped steadily as incomes increased and sanitation improved, and after 1950 decreased more rapidly to today's low levels, due most likely to the introduction of anti-tuberculosis chemotherapy.[16] While this may give cause to hope that a rise in average income may reduce tuberculosis without direct action to treat the disease, this is not borne out by patterns of incidence in the developing world. The global burden of cases has been rising, by 0.4 percent in 2001,[17] and in the light of the rise in HIV it was estimated in 1993 that case number might rise in the future, even given a decreasing risk of infection.[18]

HIV-TB has in recent years become a serious issue, given that tuberculosis is often the first opportunistic disease to strike AIDS patients, raising the risk of active disease in a bacillus infected individual from between ten and twenty percent over a lifetime to around ten percent per year. Recent estimates by Elisabeth Corbett and colleagues (2002) are that nine percent of adult tuberculosis

[16] This argument is advanced by Borgdorff et al (2001), p 11.
[17] World Health Organization (2003), p 1
[18] This is because the reduced resistance to disease raises the percentage of bacillus-infected individuals who suffer from the disease (Murray, Christopher JL, Styblo, K and Rouillon, A (1993) p 240-1).

cases, and 13 percent of tuberculosis deaths, are attributable to co-infections with HIV, with the figures for Africa being considerably higher.

MDR-TB is defined as new cases of tuberculosis which are resistant to at least isoniazid and rifampicin, the two mainstays of tuberculosis treatment, and such resistant strains of the disease have come into existence largely as a result of uncompleted drug regimens to solve uncomplicated infections. Patients in such cases cannot be treated using the DOTS formula, but must undergo far longer and more expensive regimens, which cost tens of thousands of dollars at market prices. Thankfully recent moves to create a worldwide joint-purchasing arrangement have significantly reduced the cost to providers, but the burden of treating such cases continues to be considerable: even on the best terms MDR-TB drugs remain some 50 times more expensive than a standard DOTS regimen. A recent study by Pedro Suárez and colleagues (2002) in Peru estimated that in a well-run programme for MDR-TB patients the cost per DALY gained would still be at least US$ 165. Christopher Dye and colleagues (2002) estimate that some 3.2 percent of all tuberculosis cases were MDR-TB in 2000, and so long as many countries, and individuals, take an ad-hoc approach to tuberculosis treatment these figures, and thus the average cost of tuberculosis treatment, are likely to rise.

Given this potential combination of lower benefits (HIV-TB) and higher costs (MDR-TB) to treatment, outlays to restrain such developments may pay

considerable dividends. Katherine Floyd and colleagues (2002) estimate that approximately US$ 1.2 billion is required each year to treat tuberculosis worldwide, and that at least US$ 300 million of this is not available from existing sources. These costs consist almost equally of tuberculosis specific inputs such as medicines, and demands made by treatment on general health services and facilities, such as hospital beds and staff. While there are arguments for providing such funding on humanitarian grounds, the purpose of this study is to determine whether such expenditures are likely to prove justified on an economic basis.

2. LITERATURE REVIEW

In order to study the impact of disease on economic growth two aspects of the Economics literature need to be brought together. The first of these is the economic growth literature itself, its origins and developments to the present. The second is the literature concerning the impact of health, either positively or negatively measured, on productivity and thus ultimately growth. This section proceeds by describing first the theoretical and empirical literature on economic growth; followed by some concerns relating to the measurement of health and microeconomic evidence on the impact of health on productivity; and then an overview of the empirical literature on the macroeconomic effects of health on national output. Demonstrating a connection between health and growth is only beneficial to those in need if policy change actually leads to a reduction in suffering, and thus a rise in incomes, and thus finally an outline of the literature linking health policy and health outcomes is provided.

2.1 Theoretical models of economic growth

While many people are responsible for the process which led to it, much of today's growth literature derives from a single paper. Robert Solow (1956) outlined a single-sector model of the economy with two factors of production, labour and capital, operating under constant returns to scale, and extended this by adding neutral technological change to create a model of the economy as:

$$Y = A(t)\, F(K,L) \qquad (2.1)^{19}$$

where Y is output; A is technological change; K is capital stock; and L is the size of the labour force.

This model leads to a neat division of any growth in the economy into changes in the levels of factors of production, and the efficiency with which they are used, that is technical change. Over the following two decades the field of growth accounting arose to perform just this task, determining what economic growth could be explained by changes in factor levels, and what remained unexplained, 'total factor productivity' (TFP). Solow's work also implies that so long as savings, technological progress, and population growth rates are equal across nations, countries would converge to a single level of income per capita, due to diminishing returns to capital arising from the Cobb-Douglas nature of the production function.

During the 1980's there were suggestions that the constant returns to scale assumption of Solow was unrealistic when one took into account the effect of knowledge and its diffusion. Romer (1986) proposed the existence of externalities for knowledge accumulation as a form of human capital, and thus viewed technological change as arising primarily from "the accumulation of

[19] This is not the equation with which Solow began his paper, but one which he considered in his extensions (Solow (1956) Equation 13, p 85).

knowledge by forward-looking, profit-maximising agents",[20] rather than an exogenous process. Lucas (1988) also focuses on skill levels, and constructs a model where current earnings are foregone in preference for higher future earnings through a learning-by-doing mechanism.

Although Romer saw externalities as existing across space between individuals, while Lucas believed they exist through time, both authors used this approach to escape the constant returns to scale result – that all nations would come to rest at a single growth rate – which was at odds with available evidence that countries' incomes have diverged over the known history of the world.[21] While evidence on this matter has tended not to support such direct claims of endogenous growth, as shall be shown below, the insights raised on the nature of learning and skills were crucial to the empirical growth work which followed.

2.2 Empirical models of economic growth

In order to test these two theories, in the early 1990's several authors sought to test both the exogenous and endogenous growth models across countries. The first, and probably most famous, of these were Gregory Mankiw, David Romer and David Weil (1992) and Robert Barro (1993). While their results are similar, their underlying assumptions are very different. Mankiw, Romer and Weil

[20] Romer (1986), p 1003
[21] Romer (1986), p 1008-13

(hereafter MRW) take Solow's model seriously, and transform it into a set of testable hypotheses which maintain the assumption of constant returns to scale. Their first efforts are constrained to using only capital and labour as inputs, but when these provide inconsistent results, the authors broaden their model to take into account the human capital suggested by Romer and Lucas:

$$\ln y = \ln A(0) + \frac{\alpha}{1-\alpha-\beta} \ln s_k + \frac{\beta}{1-\alpha-\beta} \ln s_h - \frac{\alpha+\beta}{1-\alpha-\beta} \ln (n + g + \delta) \qquad (2.2)^{22}$$

where y is per worker output; s_k and s_h are respectively savings invested in physical and human capital; α and β are the elasticities of income with respect to these investments; n is the population growth rate; g is the annual improvement rate of technology; and δ is the depreciation rate.

Estimating this model, assuming (g + δ) to be constant worldwide and using the proportion of working aged individuals who are of school age as a proxy for human capital investment, MRW find that the returns to physical and human capital are each around 0.3, reflecting their shares in national income, and thus rejecting the thesis of externalities to capital investment.[23] The authors then continue to show that although absolute convergence in incomes had not been seen historically across countries, when variation in savings and population

[22] This is based on equation (11) from Mankiw, Romer and Weil (1992) p 411.
[23] MRW (1992) table 2, p 420

growth rates are allowed for, a slow but significant conditional convergence is observed. Such a model looks at the dynamic path of equation (2.2) around the steady state,[24] testing the convergence rate to this steady state, $\lambda = (n + g + \delta)(1 - \alpha - \beta)$ as:

$$\ln y(t) - \ln y(0) = \Theta \frac{\alpha}{1-\alpha-\beta} \ln s_k + \Theta \frac{\beta}{1-\alpha-\beta} \ln s_h$$

$$- \Theta \frac{\alpha+\beta}{1-\alpha-\beta} \ln (n + g + \delta) - \Theta \ln y(0) + \varepsilon \qquad (2.3)^{25}$$

where y(t) and y(0) are current and initial income per effective worker; and $\Theta = (1 - e^{\lambda t})$.

A negative regression coefficient on ln y(0) signifies the existence of convergence, the magnitude of which can be measured by calculating λ. The slow rate of convergence discovered by MRW, 1.37 percent per year for the broadest sample,[26] made far more sense in the human capital augmented Solow model, when $\alpha = \beta = 1/3$, than in the original model where $\beta = 0$ and thus growth should be twice as fast.

While MRW's work, based on the strict structure of Solow's model, succeeds in roughly halving the unexplained variation in countries' growth rates,[27] Barro

[24] It is generally accepted that the assumption that all nations are at all times at, or even near, their steady states is a strong one in individual cases, but not so unreasonable in the round.
[25] This is equation (16) from MRW (1992) p 423
[26] MRW (1992) table 5, p 426
[27] Taking the case of Non-Oil countries: in MRW (1992) table 1, which reports results for the original Solow model, the R^2 is 0.59; in table 2 where the augmented model is displayed the R^2

believes that a broader approach was needed to uncover which other factors contributed to growth, that is, which play a part in TFP creation. In order to do so he takes a less structured approach, merely regressing per capita growth over the period 1960 to 1985 on initial per capita income; initial school enrolment rates; government consumption expenditure levels; measures of political instability, such as the number of revolutions and political assassinations; and measures of price distortion. In common with MRW, Barro finds that holding initial investment through schooling constant, conditional convergence does occur. These 'Barro regressions' have been broadened over the past few years, and now regularly include both proxies for input growth such as investment and human capital, and possible determinants of TFP.[28] In an excellent survey of the 'New Growth' field, Jonathan Temple (1999) devotes a section to evidence on the impact of input growth on income growth, and another to evidence on other factors.[29]

All of the foregoing was performed as cross-sectional analysis, considering a single time period. Given the dynamic nature of the issues such as the convergence of growth rates, the use of a panel data approach would seem to offer potential for greater explanatory power, and beginning with a paper by Nazrul Islam (1995) this has become the standard manner in which to consider

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rises to 0.78. This is admittedly a rough measure of goodness of fit, but acts as a reasonable yardstick.

[28] See Barro (1997) for an exposition of his approach to growth regressions.

[29] Temple (1999) sections 7 and 8, p 137-48

empirical economic growth questions. Islam compares three potential forms for the error term: the first where all errors are randomly distributed, regardless of the country considered – a pooled model; the second where errors is correlated with the regressors in a specified manner, within a panel framework – a Generalised Least Squares correlated-effects model; and the third where each country has its own error term – a Least Squares Dummy Variable, or fixed-effects model. In an initial model without human capital, the author finds that the use of a panel format gives rates of convergence and elasticity of output with respect to capital more in line with ex-ante expectations than previous work. The inclusion of human capital does not change the results, and indeed the measures of education used prove unhelpful in explaining growth in a panel format.[30] Islam's key conclusions are that the use of panel of data allows us to avoid 'convergence club' analysis which has historically suffered from endogeneity,[31] and that individual country effects have a significant impact on parameter estimates in international production function estimation.

To sound a note of caution on the use of macroeconomic growth regressions such as those mentioned above, it is instructive to listen to the progenitor of all this work, Solow himself. In a 2001 paper he questions the use of his original model

[30] This may be due to the use of gross school enrolment rates; however as is noted in section 3.2 the importance of human capital in growth regressions is a longstanding and unresolved issue.
[31] Convergence club analysis entails studying groups of similar nations, since convergence in such a setting is often considerable. As Bradford De Long (1988) points out however this approach suffers from considerable selection bias. Islam notes that a fixed-effects model allows for individual country effects, taking the convergence club analysis to its logical conclusion, but avoiding selection issues.

in the manner it is commonly found today, on the grounds that it assumes that the economy is constantly at least close to its maximum potential level of output. As he notes this is often not true in developed countries, let alone developing, agriculturally-based ones where a bad year can have a huge effect on per capita income. Solow also suggests that rather than a single technological function to measure A(t) it may be that many exist, both across countries and between factors of production, such that a realistic production function might look something like:

$$Y_{it} = F_i (A(t)K, B(t)L) \qquad (2.4)^{32}$$

where A(t) is now technological change in capital investment, and B(t) is technological change as it affects changes in labour productivity, and the overall function F_i may vary for each and every nation.

Solow's conclusions focus on the need for country-specific studies, and he does not reject the use of cross-country studies, but he does urge caution in their use.

2.3 Theoretical issues in health measurement

While it had been accepted for some time that education was a form of human capital which augmented labour inputs, it was not until the mid-1990s that it became fashionable to link health to growth in the same manner. There are

[32] This is based on Solow's suggestion in Solow (2001) p 285.

intuitive links between health and productivity, both directly through work time not lost to illness, and indirectly through education time not lost or wasted through inability to concentrate. Before diving into the empirical effects of health on income and productivity, it is perhaps prudent to discuss some theoretical issues, in particular the manner in which health can be measured, and how well this measurement is performed.

One immediate issue to bear in mind if we are hoping to improve the economic wellbeing of individuals through reduced illness or increased physical ability is that almost any measurement of health comprises of two effects: nature and nurture. In order to separate these effects it would be necessary to know an individual's genetic predisposition to good health. This might be observable based on family characteristics, with some personal error term, but it does not appear that studies have been undertaken to date which take parental endowments, such as height, or morbidity history, into account in this way. This problem is particularly important when considering anthropometric measures of health such as those mentioned below.

In considering measures of health, either as a stock or as a flow, four main approaches suggest themselves, as outlined by John Strauss and Duncan Thomas (1998) and Paul Schultz (2001). Each appears to have its own problems: three of them are subjective and the other one is almost certainly viewed with error. The first candidate is self-assessed general health status, usually carried out through a

survey. These would seem very open to personal bias, but in the United States such measurements have been found to be highly correlated with subsequent reported illness and death. The second approach to measuring health is that of functional limitations, such as the Activities of Daily Living approach.[33] Generally this is a good measure within relatively homogenous groups, and is often used amongst the elderly in the developed world, but it is harder to quantify the benefits across age and income groups, where consistently relevant questions may be difficult to come by.[34]

A third, and well-explored approach, is that of nutrition, either measured through daily intake, or anthropometric measurements. While certainly objective, the former group can be difficult to measure, particularly if individuals are being asked to recall everything consumed over even a moderate period of time, and the latter suffers from the difficulty of distinguishing between genetic and environmental factors, as outlined above. The fourth approach is to use reported morbidity rates, which might seem more concrete. This however depends on common knowledge and perception of illness, both among patients and doctors, and internationally this may be biased by the lack of an effective reporting infrastructure often seen in the developing world

[33] The Activities of Daily Living questionnaire covers changes in capacity to care for oneself, communicate with others, and otherwise conduct routine behaviours.
[34] For example, questions for geriatric patients may revolve around the ability to move themselves, while those for younger individuals might include regularity of intense physical activity. Differences in lifestyles between a wife in rural Africa and a professional woman in a western city may be almost as extreme.

In the absence of objective methods for measuring illness it is likely that there is a positive impact of income on reported morbidity which may affect results.[35] It is this potential for measurement error to be correlated with other variables likely to be included in the regression which is particularly worrying, since this may well bias any results. It may be that in order to gain a clear picture of the effect of health on growth several of these measures will need to be considered at once.

In the field of health and productivity only anthropometric and morbidity factors have been seriously considered as proxies for health. On the macroeconomic growth front, the former has generally been sidelined in favour of mortality measures such as life expectancy and adult survival rates, while the latter have been used sporadically. There seems to be scope for broader use of health measurements in this work.

Although poor measurement is in and of itself unhelpful, since it introduces more noise into any equation we may wish to estimate, thus generating larger standard errors than would otherwise be found, there is a potentially more serious issue. As John Strauss and Duncan Thomas (1998) point out, systematic measurement error linked to income, for example through richer individuals being more likely to report themselves unwell for a given level sickness, will negatively effect estimates of the impact of health on income.[36] At the

[35] This is due to those with higher incomes being better able to both report illness, and take time off to recover, compared to those who are below the poverty line.
[36] Strauss and Thomas (1998) p 797

microeconomic level this latter effect has been avoided in some cases through the use of instruments which are not related to income, but which are linked to individual, or village, health. In undertaking macroeconomic work this is not however feasible, and thus we have two downward biases on any coefficient of health measures which might determine income.

2.4 Empirical considerations of health and growth

Recently there been a profusion of work which has attempted to compute the exact benefits to individuals and society, usually in terms of wage rates,[37] that better health brings. One of the leaders in this field is Robert Fogel. His work on the on the effects of nutrition on economic growth has shown that for much of Europe's history, and quite probably much of the developing world today, the bottom deciles of the population are effectively unable to conduct serious work given their nutritional intake. He notes that at the end of eighteenth century the bottom fifth of the French and English population could manage only between three and six hours of light work per day, and ten percent of the French population could not perform any work at all, using all of their energy just to perform core bodily functions.[38]

[37] This is because in its most basic form an unrestricted wage is thought of as a reflection of an individual's marginal productivity.
[38] Fogel (1994) p 373-4

Fogel proceeds to combine evidence on height distribution, an indicator of childhood nutrition, and relative mortality and chronic disease risks in later life. He shows that the very shortest individuals in various populations had a threefold relative risk of suffering from conditions such as hernias, and were two and a half times more likely to die at a given age, compared to the population average.[39] Fogel estimates that over the past two centuries, improved nutrition alone accounts for around thirty percent of increased labour input adjusted for intensity,[40] and suggested that in the developing world today both poor childhood nutrition and poor adult nutrition are likely to combine to lower productivity. Such events undoubtedly reduce output, but also reduce the incentive to invest in other factors of production, including human capital.

This macroeconomic evidence suggests a three-way link between output, human capital as education, and human capital as a stock of health, especially at younger ages. This is backed up by microeconomic evidence such as that presented by Strauss and Thomas (1998). They show positive relationships between height and wages, and between Body Mass Index and wages,[41] in Brazil and the United States. These relationships are rather more pronounced in Brazil, where the constraints of health might be considered more frequently binding, and within Brazil are stronger for those who have had schooling than for those who have

[39] Fogel (1994) table 2, p 375
[40] Fogel (1994) p 383
[41] An individual's Body Mass Index is their weight in kilograms divided by their height in metres, squared.

not.[42] Since both of these approaches focus on correlations rather than causations, a theoretical approach might be reassuring. One approach to this is to experiment through treatment of pre-existing illnesses. Strauss and Thomas mention two experiments, one in Tanzania and another in Cameroon, both among sugarcane workers. In the former those treated for schistosomiasis, a tropical parasitic disease, achieved higher earnings thereafter, although they did not reach the wage level of those who were never infected. In the latter there was no baseline difference in wages, and no change after treatment.[43] Schultz (2001) presents a swathe of evidence on the links between height and BMI, and higher wages using evidence from Brazil, Côte D'Ivoire, Ghana, China and Vietnam, which suggests that greater height and body mass are reflected in higher wages. It should be borne in mind however that the connection between anthropometric instruments and health is necessarily a long-term one, and may be clouded by genetics and societal changes.

In order to see more clearly the connection between current illness and current pay, we can turn to surveys of individual's reactions to illness, as measured by some form of morbidity. Schultz and Tansel (1997) measure both the wage and the labour supply impact of illness in two West African nations, Côte D'Ivoire and Ghana. In a limited sample, which considers only those who are earning

[42] Strauss and Thomas (1998) figures 2 and 3, p 773-4. The evidence for BMI is bell-shaped rather than linear, reflecting poor health among seriously overweight individuals as well as among the undernourished.
[43] Strauss and Thomas (1998) p 799-800

formal wages, the authors find that although OLS estimates returned significant changes in earnings due to days ill, this reflected fewer hours worked, rather than lower wages in general. When instrumental variables are used to allow for endogeneity in the number of days disabled, wages appear to be more than ten percent lower for each day ill.[44] So long as wages are a reasonable proxy for marginal productivity, as theory suggests, then this study suggests that being ill not only prevents one from working, but is also reflected in reduced productivity while not disabled. This second effect is harder to pick up from standard survey questions.

This evidence, especially in the case of males, is supported by work in Latin America reported by Schultz (2001). He reports that hourly earnings for men in urban Peru were found to be significantly lower for those who predicted to have recently taken time off work due to illness; that elderly workers in Mexico who had not taken a day off sick in the past six months received higher earnings amounting to 96 percent of their wages; and that Colombians reporting a day disabled in the past month suffered between a 13 and 33 percent reduction in their hourly wage. Interestingly both the study in Mexico and the one performed

[44] The instruments used were food prices and health service availability. This is to allow for the possibility that richer people are able to be 'disabled' more often than poor ones, and thus the wages of those taking time off work may be upward biased.

in Peru find that it is the poorest sections of society who gain most from not being ill, suggesting that there may be diminishing returns to good health.[45]

Despite this indirect evidence that various measures of health affect productivity, this does not prove that better health leads to economic growth. Weil (2001) takes a more direct approach, and attempts to measure the returns to health, as measured by height and adult mortality rates. Assuming returns to each are linear in the log of health, and using a prior microeconomic estimate of the return to height of six percent,[46] the author determines the return to a one percent reduction in mortality to be 1.68 percent.[47] He then proceeds to use a development accounting methodology to examine what proportion of the variation in per capita incomes worldwide can be attributed to changes in health. Weil concludes that 17.4 percent of all output variation is due to human capital as health, compared to 21.4 percent for education, and 18.5 percent for physical capital. This suggests that any work on growth which ignores health measures may be significantly biasing its other coefficients.

Working with two colleagues, Sebnem Kalemli-Ozcan and Harl Ryder (2000), Weil has also considered the link between health and growth from a macroeconomic standpoint. Economists have recognised for some time the potential for longer life expectancy to boost the rate of return to educational

[45] This reflects Schultz (2001) p 28-9.
[46] Weil takes this figure from Schultz (2001).
[47] This summarises Weil (2001) section 3.2, p 8-10.

investment, since it also provides a longer period over which to repay these expenses and to reap their returns. Kalemli-Ozcan et al use a continuous time, overlapping generations model, which provides both theoretical reasoning and empirical support for the argument that not only does better health improve production directly, but also an expectation of better health leads to higher investment in education. Furthermore, using a general equilibrium model the authors show that this effect remains even in the face of the whole population improving their life expectancy.[48] The link between the two forms of human capital is thus further strengthened, reinforcing the omitted variable bias in any growth equation which does not take health into account.

One reason that causality on the issue of health and growth is so hard to prove is that the relationship is two-way. Higher incomes allow for better healthcare, preventative expenditure and better living environments; better healthcare allows for higher incomes to be earned and makes skill acquisition a more profitable affair. Unlike Weil, Lance Pritchett and Lawrence Summers (1996) focused on the former, estimating the effect of income on infant mortality in low-income countries from 1960 to 1985, allowing for the level of schooling. Using instruments such as terms of trade, investment ratios and the black market exchange premium in place of income, the authors verify that an impact does appear to flow from income to health, giving an income elasticity of infant

[48] In contrast to a partial equilibrium model, if everyone is living longer then the demand for, and thus cost of, education would rise. This might have lead to a decline in the amount of education undertaken, but the authors suggest that this is not the case.

mortality of 0.2 in the short run, and 0.4 in the longer term. Since any effects would most likely take some time to become apparent in the population, this stronger long-term effect seems plausible. This acts as a reminder that caution must be taken to account of reverse causality in any regressions involving health and income.

2.5 General health in economic growth models

As noted above, there is no one single measure of health which is agreed to be a measure of the stock of wellbeing in a nation. Nevertheless there have been a number of growth regressions which have used initial life expectancy as a proxy for health stock, and as David Bloom, David Canning and Jaypee Sevilla (2002) show these have generally found positive and significant coefficients across a range of countries, time periods and functional forms.[49] One recent advance in this area has been the move from cross-sectional to panel studies, such as that performed by Scott McDonald and Jennifer Roberts (2001). McDonald and Roberts regress output per worker on the lag of output per worker, mean years of education, investment and population growth rates across 77 countries and six five-year periods from 1960. They report that country specific effects are appropriate for all samples except OECD countries, for which they use a pooled data model, and that a fixed rather than random-effects is appropriate in all other

[49] See Bloom, Canning and Sevilla (2002) table 1, p 2

cases. The authors find that adding health capital, as measured by life expectancy or infant mortality, to the equation provides evidence that the level of health stock, but not that of education, significantly affects growth in most less developed countries. In the OECD and Latin American nations their findings are reversed – education levels are a significant factor, while health levels are not.

This implication that there are diminishing returns to improved health is supported by work by Alok Bhargava, Dean Jamison, Lawrence Lau and Christopher Murray (2001). These authors focus on adult health through the use of adult survival rates (ASRs) rather than life expectancy.[50] They regress five year average per capita income growth rates on the investment ratio (INV), fertility rates (FERT), the degree of trade openness (TRA), proportion of a nation's landmass which is tropical (TROP), lagged per capita income and lagged ASRs in a random-effects panel model covering 92 countries and five five-year periods from 1965. The ASR measure and other right hand side variables were lagged to counteract any reverse causality that might be present, and their core regressions are of the form:

$$\Delta y_{it} = \alpha + \beta_1 TROP_i + \beta_2 TRA_{it} + \beta_3 \ln(FERT_{it-1}) + \beta_4 \ln(INV_{it-1}) + \beta_5 \ln(ASR_{it-1}) + \beta_6 \ln(ASR_{it-1}*GDP_{it}) + \beta_7 \ln(y_{it-1}) + \delta_i + \varepsilon_{it} \qquad (2.5)$$

[50] Adult survival rates estimate the proportion of a cohort of fifteen year olds who will still be alive at age 60, on the basis of mortality data, sometimes augmented by demographic projections.

Using both World Development Indicators and Penn World Table data Bhargava et al consistently find that increased adult survival has significant positive effect on output, along with positive effects for investment and greater openness, and negative ones for high fertility and tropical location. They also introduce a variable which interacts the lag of the adult survival rate and income (ASR*GDP), which appears in their results with a significant negative coefficient. Interpreting this result jointly with the positive coefficient on ASRs the authors suggest that improved adult survival improves economic growth while income per capita is low, but that as income rises the effect diminishes, and at very high income levels it may even be correlated with a reduction in growth.[51] The predicted level of per capita income at which this effect no longer significant is US$ 907 using data from the Penn World Tables, and it reaches zero effect at US$ 2,123. The estimates using data from the World Development Indicators suggest that this point is reached at rather lower income levels.

While these results are undoubtedly important they are not directly related to the productivity and health literature described in section 3.4. This link can however be found in the recent paper by Bloom, Canning and Sevilla (2002) which sets out specifically to explore the relationship between returns at the micro level and those in macroeconomic growth regressions. Noting the results found by Weil (2001), Bloom et al use data from the Penn World Table to estimate a production

[51] Bhargava et al (2001) note that this is unlikely to be a causal relationship, merely the conjunction of European nations with lowish recent growth rates and large elderly populations.

function for national economies which intentionally mirrors that of Weil. Core variables include the stock of investment in the economy as measured by a perpetual inventory method; the stock of education capital as measured by average years of schooling; a term which allows for technological catch-up by less-developed nations; and the workforce's average experience and square of average experience, based on its age structure. Due to technical and theoretical problems with fixed-effects models the authors used Non-linear Least Squares (NLS) estimation, including tropical landmass and the openness of the economy to trade to account for some of the systematic country-specific effects.[52] The authors find that a percentage point increase in the ASR raises labour productivity by around 2.8 percent, and that the figure is not inconsistent with Weil's reported return of 1.68 percent, suggesting that the microeconomic evidence that health affects growth is reflected at the macroeconomic level.

2.6 The impact of specific diseases on economic growth

While the above literature suggests that health improvements in general may well improve income growth, there has also been a growing interest in the possibility that certain diseases may have considerable effects on the productivity of workers. Infectious diseases are central to this interest, since there exists both

[52] Experimenting with fixed-effects models using dynamic panel General Methods of Moments approach the authors found large standard errors were plaguing their results. Additionally, their intention to use lagged variables as instruments for contemporaneous ones would lead to issues of low precision and uncertain inference. The authors do not explain how the approach they did use avoids these issues.

considerable externalities to their treatment and often existant cures which are only in need of competent distribution, and it is therefore not surprising that these are the diseases which have been focused on. Concrete evidence of the potential benefits to curing patients of such diseases would very likely lead to a considerable rise in funding. To date the two conditions which have been seriously considered in this field are HIV/AIDS and malaria.

There is little doubt that HIV has had a huge impact on the world in the past 20 years, and also that it has reduced population levels significantly across Africa, leaving a generation of AIDS orphans in its wake. The difficulty with understanding this epidemic is vast, given the variation in reporting standards. Many nations have at one time or another denied the existence of the disease within their borders, and even today consistent reporting figures are hard to come by. As a result those who have tried to estimate the impact of AIDS at the macroeconomic level have tended to rely on estimates and projections of illness.

In the two key studies using a Solow framework to date John Cuddington (1993) focuses on one country, Tanzania, while Bloom and Ajay Mahal (1997) cover 51 nations worldwide. Noting that the rise of HIV morbidity has reduced labour productivity, raised health care costs, and thus reduced savings and other consumption expenditure, Cuddington links this to reduced human capital investment at the family level due to reduced capacity to pay. The author also explains that rising mortality will reduce the average age, and thus experience, of

the working population which may well reduce average productivity, and that pressures on government social programmes are also likely to rise as the dependency ratio rises due to the deaths of many working-aged individuals. In the Solow model Cuddington sees several effects: reduced productivity will shift the production function down, reducing output per capita; reduced population growth will flatten the required replacement locus, improving potential growth; and reduced savings will steepen this same locus, lowering the steady-state income level. The interplay of these effects are simulated using country-specific data, and Cuddington concludes that while per capita effects are less than those on total GDP, the presence of AIDS in Tanzania may lower per capita income by up to 11 percent by 2010.

Bloom and Mahal accept the arguments of Cuddington and others, but note that there are several reasons why earlier estimates of the impact of AIDS may be overstated. First, it is poor women, rather than rich men, who now bear the brunt of the epidemic in Africa. Second, the existence of social adjustment mechanisms, such as the extended family and community-based organisations, may mitigate health care costs through the provision of informal services. Third, AIDS costs are unlikely to only drawn from savings funds as some have argued, but also from consumption expenditure, thus offsetting some of the expected lower investment. Finally, in the face of the epidemic many people are likely to adjust their sexual behaviour, and therefore early estimates of cases may well be

overly pessimistic. Using available national prevalence data and estimates of prevalence by organisations such as the WHO and the United States Bureau of the Census, the authors estimate cumulative AIDS cases in each country through an epidemiological package, EPIMODEL.[53] Using figures thus derived in a Solow style regression Bloom and Mahal find that AIDS prevalence is not significantly related to growth in their sample: that low income countries have high HIV prevalence rather than vice versa. While at an individual level HIV/AIDS clearly has a catastrophic effect on families and lives, this did not appear to have yet made itself apparent at the national level by the early 1990's, at the end of these author's period of study.

While there is a debate over the effect of AIDS on growth, perhaps largely due to the high mortality, but relatively short morbidity, of the disease, the effect of another disease with almost the opposite profile, malaria, is little disputed. This is a disease where mortality for native populations over the age of five years is rare, but where suffers are at least partially incapacitated once or twice year for around two weeks at a time. The productivity effects of the disease are therefore recurrent, and significant in countries where large proportions of the population are patients, which today lie almost exclusively in the tropics. Two different studies, one by John Gallup and Jeffrey Sachs (2001) and the other by Desmond

[53] Bloom and Mahal model the process in three steps: income growth is a function of AIDS cases and other factors; AIDS rates are a function of initial establishment of HIV, current HIV prevalence and expected peak year of HIV prevalence; and HIV prevalence is a function of income and other variables affecting transmission.

McCarthy, Holger Wolf and Yi Wu (2000) consider the effect of this disease through different data but the same basic methodology. Both attempt to discover if there is a residual effect of malaria on growth, over and above general mortality effects.

Gallup and Sachs focus on the most virulent and serious form of the disease, falciparum malaria. Using 1955 malaria risk maps, the authors create a malaria index for each country which reflects the proportion of the population living in high risk areas at a given point in time. From the 1965 and 1990 population distributions they create an initial and final index for each country. The initial index figure (IMAL) and a measure of the change in the index (Δ MAL) over the intervening period are entered into a standard Solow cross-sectional regression along with measures of the initial level of secondary schooling (SEC), geography (TROP; COAST), good governance (TRA; INST) and initial life expectancy (LE) covering the period from 1965 to 1990:[54]

$$\Delta y_i = \alpha + \beta_1 \ln(\text{initial } y_i) + \beta_2 \ln(SEC_i) + \beta_3 \ln(LE_i) + \beta_4 TRA_i + \beta_5 INST_i + \beta_6 TROP_i + \beta_7 COAST_i + \beta_8 IMAL_i + \beta_9 \Delta MAL_i + \varepsilon_i \quad (2.6)$$

Gallup and Sachs find that the difference between an initial index value of one and one of zero was 1.3 percent growth per capita per year. Further, a decrease in the index over the 25 year period from one to zero, although this happened in

[54] COAST refers to the proportion of a country's population living within 100 kilometres of the coast; INST refers to an index of public institutional quality as measured by business attitudes within a country.

no country, would have increase the growth rate by 2.6 percent per year. In order to account for the potential of omitted variable bias on the malaria coefficient, indices for 20 other tropical diseases are also included, and a separate regression is run using prevalence of anopheles mosquitoes, the most potent vector for the disease, as a instrument to avoid possible reverse causation from higher income to reduced malaria incidence. In neither case were the results significantly altered.

McCarthy, Wolf and Wu extend Gallup and Sachs' work by using a panel approach over three five-year periods beginning in 1983 and by separating out the direct impact of malaria on growth, and its indirect impact through human and physical capital investment. The authors use average morbidity figures per 100,000 of population (MAL) as reported to the WHO in pooled and seemingly unrelated regressions in the form:

$$\Delta y_{it} = \alpha + \beta_1 \ln(\text{initial } y)_{it} + \beta_2 INV_{it} + \beta_3 PRIM_{it} + \beta_4 SEC_{it} + \beta_5 TRA_{it} + \beta_6 GOVT_{it} + \beta_7 FREE_{it} + \beta_8 REV_{it} + \beta_9 ASS_{it} + \beta_{10} MAL_{it} + \varepsilon_{it} \quad (2.7)$$

where PRIM is primary school enrolment, GOVT is government consumption expenditure as a percentage of GDP, FREE is a measure of political freedom, and REV and ASS refer to the number of revolutions and political assassinations which occurred during the period in question.

The authors find a steadily significant negative coefficient on malaria morbidity throughout, which for almost a quarter of the countries they sample implies that the effect of eliminating malaria would be greater than 0.25 percent additional per capita growth each year. In the most extreme case, Malawi, eradication is estimated to be worth 3.22 percent faster growth per year.[55] The results for regressions to find the indirect impact of malaria on other forms of investment are in no case significant at the 10 percent level, suggesting that indirect effects, if they exist, are too weak to be picked up in this way.

2.7 Linking health expenditures and health outcomes

While it is important to establish the theoretical and empirical links between reduced disease burden and increased output, it is also important to understand that often there are considerable practical problems in moving from such evidence to a situation where disease burden is indeed decreased. This is often due to the difficulty of implementing programmes, especially in those countries where they are most needed. Considering Brazil as an example, Werner Baer, Antonio Campino and Tiago Cavalcanti (2001) explain that although standards of health have improved over the past century there remain core problems due to a high burden of curable infectious and parasitic diseases whose continued presence is largely a result of a weak healthcare infrastructure. This situation has

[55] The authors urge caution in accepting these point estimates since the link between income and malaria is likely to be non-linear in some manner that cannot easily be judged.

occurred despite a considerable rise in health expenditures as a percentage of GDP, from one percent in the 1950s to around six percent in the 1980s. The authors note that this coincided with a move from expenditures on public preventative healthcare toward a curative framework.

The causes and effects of such policies are studied in some detail by Dean Filmer, Jeffrey Hammer and Pritchett in two companion papers (2000, 2002). In the context of primary health care (PHC), or health care for all, the dominant approach to developing world health care over the past twenty years,[56] Filmer et al (2000) posit a four stage model for determining public health care provision:

1. Composition of public spending: where is the money spent. This might be guided by policies such as PHC, or cost-effectiveness evidence;
2. Output of the public sector: what is created by this expenditure. This can be measured in terms of new clinics or equipment, or through more capable personnel;
3. Net impact of public sector supply on overall consumption: to what degree does additional public expenditure reduce private provision of services;

[56] The approach is described by the WHO, quoted in the second article, as being focused on universally accessible, locally delivered care catering to essential needs such as health, education, clean water, immunization, maternal and child health, and other basic treatment. This is often carried out through health clinics based in small towns and villages. It is absolutely not secondary or tertiary curative care.

4. The health production function: what concrete changes in health outcomes result from this change in health inputs.

The authors show a range of evidence from which it is apparent that increased government expenditure at a national level, on local health clinics, and on childhood healthcare often do not produce any measurable net benefits. They argue that this is due to steps two and three in the chain above. In the case of public sector output, the poor quality of public provision, and the shortage of equipment, drugs and workers means that it is often in the population's interest to pay for private provision, bypassing services which are nominally free-of-charge. Filmer et al note that in the few cases where high quality public provision exists in a low-income setting there have been successful programmes to motivate staff to work, and to work well, often through either implicit or explicit threats of unemployment or physical violence if they fail.

Over and above poor provision of services, there is also the problem of undertaking services which are already well provided by the private sector. The authors suggest that when there exist low price and income elasticities of demand for a service; when there exists a large private sector; when public and private provision are highly substitutable by geography, price or time; and when the private sector responds strongly to changes in government policy, it may well not be in the country's best interest to provide many services publicly, even at low income levels.

Filmer et al (2002) then continue to discuss which areas of primary health care are most in need of government intervention, taking into account these two failures, and the need to address issues of poverty and inequality – which Baer et al also found to be crucial in explaining the poor performance of Brazil's health care system. Not surprisingly the authors find the greatest need for public assistance to be in those fields where externalities and public good effects are common: vector control, health information generation, infectious disease control, vaccinations and educational campaigns. They particularly stress the need for improved public sanitation, which has an impact in several of these fields. The authors also consider the failure of private health insurance, and in the absence of a comprehensive public plan, they cautiously recognise the need to provide public hospital care.[57]

Instead of accepting PHC as a package to be promoted, Filmer et al try to divide up the services in order to discover where best the public sector might get involved. Considering health interventions on two axes, those of distortion through externality and responsiveness to public policy, they note that routine curative care tends to be market provided and change little when PHC programmes are rolled out, while those issues mentioned in the previous paragraph tend to be underprovided and respond well to government intervention. A different approach is to consider which policies really play a role

[57] Cautiously since such care is disproportionately taken up by those who are not poor, but by those who are richer and thus have greater access to the health care system.

in reducing poverty, and which do not. Studying general health care expenditure Filmer et al point out that PHC policies are relatively pro-poor, compared to secondary and tertiary care, but not absolutely so. Noting that communicable diseases are the most differentially burdened health conditions they suggest that a reallocation from clinical PHC services to population-based ones would in all likelihood be an absolutely pro-poor measure.

These arguments are supported by the difficulty of managing a decentralised network of rural clinics as compared to an immunisation or sanitation improvement programme. In the former case shirking is far harder to monitor, and the need to be permanently in a rural environment will probably be a deterrent for many able professionals. The authors conclude that a more nuanced approach to developing world public health care is needed.

This conclusion is supported by the work of the CMH (2001). Focusing on all members of low-income countries, and the poorer sections of middle-income nations, the CMH outlines areas of avoidable deaths, mostly infectious diseases and maternal/child health, and recommends a programme in many ways similar to that of Filmer, Hammer and Pritchett. The commission's report begins by outlining the links and effects of health on development, in a manner similar to this paper. The CMH links disease to low growth through direct productivity effects both on output and on educational achievement, through intergenerational effects of reduced income leading to reduced investment, and

through societal spillovers of epidemics such as those currently seen in Sub-Saharan Africa from AIDS.

Noting the current low levels of health spending in low-income countries at present,[58] the CMH estimates that health expenditures in these nations would need to rise by two-thirds to achieve these essential treatments. The positive side to this is that this amounts to only US$ 14-18 per capita per annum.[59] Almost all this expenditure is focused in community level, or 'close-to-client' services outside of complex hospital environments. In order to achieve this 'scaling-up' of services the commission recommends that increased taxation in poorer countries go hand in hand with a more direct link between local payments and local services, and an increased role for donor financing for public good provision.

This move towards localised, and also categorical,[60] provision is largely seen as a reaction to non-financial constraints to improved health care, largely those of politics and capacity. The CMH believes that for every dollar spent on improved health care, a further 15 cents needs to be allocated to improving national management structures, another 15 cents to improve the capacity of the health system to absorb such incremental funding, and an additional 10-25 cents to bring the current quality of care up to best-practice levels. Finally, in

[58] The CMH (2001) defines low-income as all Sub-Saharan nations plus other nations with income of less than US$ 1,200 per person per year. This would include all countries in the World Bank's lowest category, low-income, and some of those in its second category, lower-middle income. For more details on the World Bank categories, see section 3.3 and/or appendix 1.
[59] Commission on Macroeconomics and Health (2001) p 54-5
[60] That is to say, a vertical focus on a specific disease or family of interventions which bypass the inefficient health-care system.

acknowledgement of the current low level of incentives for health care professionals the report recommends a doubling in all health worker wages in low-income countries.[61] Adding such expenditures to direct treatment costs are believed crucial in actually improving health outcomes, but the degree to which nations are constrained by such factors varies greatly within the group of low-income nations. Thus such additional spending will be far more important in some countries and settings than in others.

Assuming that these processes are efficiently enacted, the commission estimates that these policies will save some eight million lives per year by 2015, which translates into some 330 million DALYs. Using a partial equilibrium outlook, and calculating each year as being worth per capita income in low-income countries, the CMH suggests that this would provide a direct benefit of US$ 186 billion per year by 2015, as well as additional dynamic benefits flowing from reduced poverty in these nations. While these figures are considerable it should be borne in mind that the general equilibrium effects are likely to be less, and this is against a counterfactual of no improvement in healthcare over the next 15 years.

The conclusion of work on the links between health expenditure and outcomes is that well-focused and well-planned spending can certainly improve outcomes; the issue becomes one of whether sufficient political, institutional and financial

[61] This paragraph summarises the findings of the CMH, Appendix 2 "Analysis of the Costs of Scaling Up Priority Health Interventions in Low- and Selected Middle-Income Countries".

willpower exists to enact these policies. This work strongly supports the provision of healthcare through categorical programmes which focus on the eradication of infectious diseases – such as the DOTS programme against tuberculosis. Indeed the CMH includes this programme in its list of key policies to be enacted. The rest of this paper will seek to discover whether this theoretical backing of such programme can be buttressed by empirical evidence of a link between lower tuberculosis incidence and higher economic growth.

3. METHODOLOGY AND DATA

Taking a path somewhere between the extremes of MRW and Barro, this study will regress differences in logged per capita, and per worker, GDP on logged values of both factors of production – physical and human capital stocks in per person terms – and generally accepted determinants of total factor productivity (TFP). This section proceeds by first considering which data to use, then some basic characteristics of this data, and finally the most appropriate way in which to model them in a regression.

Although in many, but not all, cases annual data is available for series, in order to reduce the effect of poorly measured data, in particular for disease variables where reporting error is likely to be considerable, this study follows many of its predecessors in using five year periods as the base unit of time, beginning in 1981 for twenty years. These data cover a range of countries, however the core of the empirical work in this paper covers 91 countries for which a balanced panel of data exists across a number of sources. Details on the countries included can be found in appendix 1, and on the series used in appendix 2.

3.1 Economic data

The two main sources of cross-country macroeconomic data are the Penn World Table (PWT), originally produced by Robert Summers and Alan Heston (1991)

and now overhauled as version 6.1 by Heston, Summers and Bettina Aten (2002), and the World Development Indicators (WDI), published by the World Bank (2002). The former provides output data in per worker and per person terms and in purchasing power parity exchange rates, which are intended to accurately reflect costs of living in a given country.[62] The latter provides data only in per person effects, using nominal exchange rate comparisons. Both series are indexed for price increases such that data are presented in constant 1996 and 1995 US dollars respectively.

In a strict Solow regression, as in any production function, one should consider output and inputs in terms of workers, rather than the population level as a whole – in converting equation (3.1) into per person terms, the denominator is the labour force, not the population as a whole. Nonetheless Bhargava et al find that using per person data does not significantly affect the results of their equations, and Temple notes that the quality of available data on labour force participation and worker hours is measured with not insignificant error, particularly in the developing world.[63] In this study core data will be taken from WDI, but regressions will also be conducted using PWT data for comparison purposes.

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[62] The PWT data makes use of a number of benchmarking studies to determine purchasing power in 115 countries worldwide. Other countries' spending patterns are inferred from those of similar benchmarked nations. Details are available in the appendix to PWT (2002).
[63] A fuller discussion of problems relating to output measurement can be found in Temple (1999) p 118.

For each of these two sources data was compiled for GDP per person in the final year of each period (GDP_{it}),[64] and the difference between the log of this figure and that of GDP per person at the end of the final year of the previous period (ΔGDP_{it}). The average level of gross capital formation or investment (INV_{it}) as a percentage of total national income was used to measure the flow of physical capital being added to the economy, assuming constant rates of depreciation across countries and time.

The level of Government expenditure ($GOVT_{it}$) as a percentage of income, the level of openness of the economy (TRA_{it}) measured as the sum of exports and imports as a percentage of income, and the level of population growth ($POPG_{it}$) were also computed from both databases as explanitors of TFP variation. Government expenditure, in particular consumption rather than investment expenditure, might be thought to crowd out private investment or just generally be indicative of inefficiencies, especially at high levels in developing countries. The openness of the economy may also be seen as a sign of the competitiveness and efficiency of a nation. Population growth is a more complex question, and the stock measure that is the age structure of the nation is probably more important than the flow measure of net births and deaths. Nonetheless population growth acts as a proxy for fertility, and as such may well reflect some facet of a nation's changing character through time.

[64] For the sake of brevity, per person can be taken to refer to both per person and per worker when referring to data, depending on the source being used, unless otherwise specified.

Table 3.1 outlines the economic data used, both from the WDI and the PWT. The trends in the data are broadly similar: population growth and government expenditure fall over time; openness to trade and initial GDP rise; gross capital formation holds steady; and GDP growth is high in the latter part of each decade. The differences in levels between data sources for government spending and investment are most likely a result of different definitions: the pairs of series have very similar growth paths.

3.2 Human capital data

The question of how to best represent human ability capital in a macroeconomic regression is complex one. Following microeconomic approaches and including experience is difficult at a national level, since no consistent or sufficiently nuanced measure of experience exists.[65] Emphasis has therefore been placed on education. Early work used gross school enrolment rates due to their broad availability, but it was recognised that this is a blunt instrument, and one which considered only the flow of education, and not its stock. These measures were thus supplanted by measures of the average years of schooling held by the adult population, in particular those compiled by Barro and Jong-Wha Lee (1993). Barro and Lee (2001) have since updated their data to cover the period 1960 to 2000. Unfortunately both of these approaches failed to provide significant and

[65] For example raw data will not give information on whether the experience gained relevant to the job held; and a national mean may well cover a multitude of cohort differences.

Table 3.1

Economic Data (Balanced Panels)

		All Periods				1980-1985	1986-1990	1991-1995	1996-2000
Source		Mean	Standard Deviation	Maximum	Countries	Mean			
WDI	Growth in GDP	678	1,472	11,769	119	368	822	530	993
	Initial GDP	6,263	9,432	45,951	119	5,444	5,812	6,634	7,164
	Gross Capital Formation	22.6	7.39	60.4	119	23.3	22.4	22.5	22.4
	Gov't Consumption Exp.	16.2	6.26	42.2	119	17.1	16.4	15.8	15.4
	Openness to Trade	76.9	49.7	380.3	119	73.6	73.1	78.1	82.8
	Population Growth	1.77	1.13	5.69	119	1.89	1.83	1.71	1.65
PWT	Growth in GDP	1,339	3,152	23,109	103	438	1,413	1,366	2,136
	Initial GDP	16,426	14,580	80,027	103	15,049	15,487	16,901	18,267
	Investment	15.2	7.62	40.3	103	15.5	14.6	15.1	15.6
	Gov't Expenditure	20.0	11.1	61.4	103	22.1	21.0	19.0	17.9
	Openness to Trade	66.9	39.6	293.7	103	61.7	61.4	67.6	76.9
	Population Growth	1.81	1.11	6.01	103	1.91	1.85	1.80	1.67
BL	Years of Schooling	5.51	2.86	11.89	109	4.97	5.23	5.74	6.06
CS	Years of Schooling	5.94	3.21	12.47	95	5.27	5.72	6.18	6.57

Sources: WDI refers to the World Development Indicators 2002; PWT to the Penn World Tables 6.1; BL to the Barro and Lee education dataset; CS to the Cohen and Soto education dataset. See text for further details.

GDP and growth in GDP are measured in constant 1995 US dollars; Gross Capital Formation, Government Consumption Expenditure, and Openness to Trade are measured as percentages of annual GDP; Population Growth is measured in percent.

robust results comparable to those found at the microeconomic level.[66] In a famous paper Pritchett (2001) asks where all the education has gone. He suggests that the imposition of a single, linear rate of return on education across countries may hide considerable international variation in returns to schooling; and that institutional constraints, low demand elasticities to increases in educated labour, and the poor quality of much of the schooling provided may explain the lack of an observed return to increased education. In response Daniel Cohen and Marcelo Soto (2001) at the OECD have produced a new dataset for 95 countries from primary sources which they claim significantly out-performs the Barro-Lee set, particularly when considered in differences rather than levels. In a second paper Soto (2002) argues that issues of colinearity between physical and human capital, and of endogeneity and measurement error, are often the cause of low estimates for human capital. Using the Cohen-Soto dataset and adjusting for colinearity he finds macroeconomic estimates for the return to schooling close to those of the microeconomic literature.

In this study the series from both Cohen-Soto and Barro-Lee which record the average number of years of schooling for the population over 15 will be used.[67] Since the Cohen-Soto data is only available at ten year intervals, the figures for 1985 and 1995 were formed by interpolation, assuming that changes in schooling stock were linear over each ten year period. Given this limiting assumption the

[66] See for example Islam (1995).
[67] It is generally accepted that worldwide those over 15 reflect the workforce more closely than those over the age of 25, the other generally available sample.

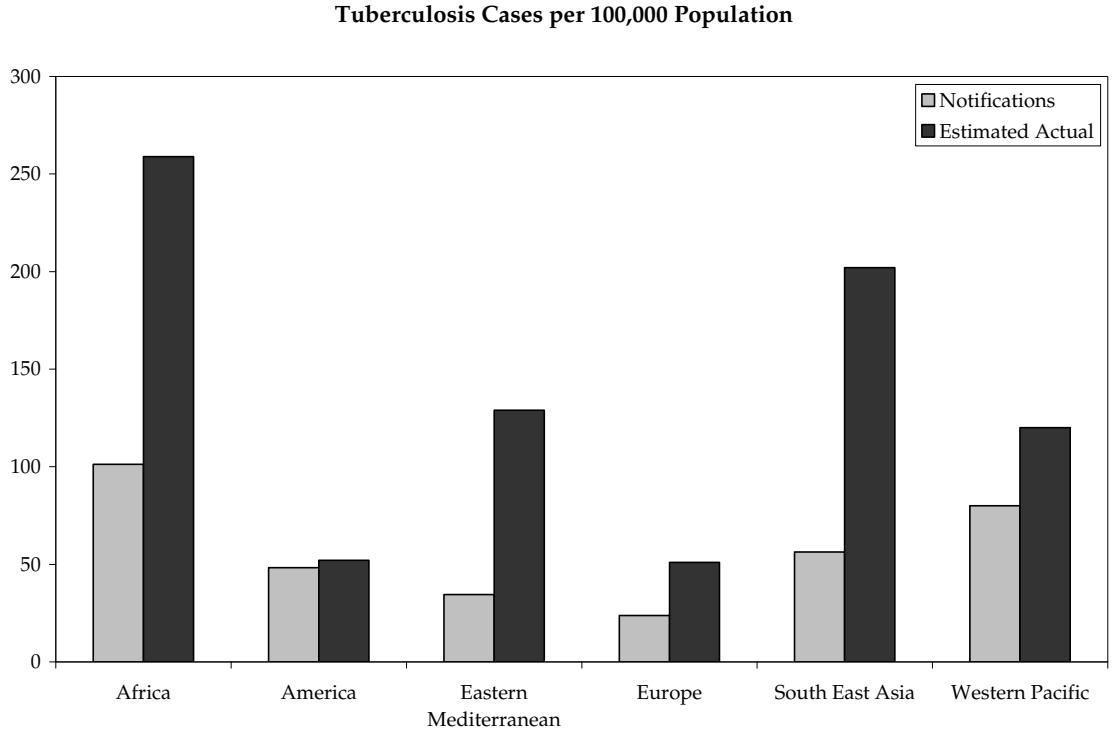
Barro-Lee (BL_{it}) data will be used as the primary source for educational attainment, and the Cohen-Soto (CS_{it}) data used as a comparison case. As table 3.1 shows, both the levels and trends of these data are very similar, although Cohen and Soto find a somewhat higher initial level and growth rate in the years of schooling held than do Barro and Lee.

This study focuses on tuberculosis, and thus information on the incidence of illness for this disease is crucial. Unfortunately long series of data on incidence do not exist. Notification data is available from 1980 to 2000 from the World Health Organization (2002), while estimates for actual incidence, made largely on the basis of this data, are available from 1996 onwards from the same source. This paper will use notification rates per hundred thousand population averaged over five year periods (TB_{it}), to allow for some of the variation in reporting, although it is accepted that this variation is a potential weakness of the work. A simple correlation coefficient between estimated and notified incidence for the 91 core countries in this study was 0.837, indicating that while notification data represents a reasonable measure of actual incidence it should still be treated with some caution.[68] This caution is supported by figure 3.1, which shows average notification and estimated incidence rates by United Nations region. It appears that the most highly infected regions underreport relative to less infected regions,

[68] This is for averaged data over the period 1996-2000. The correlation coefficient for annual data for 91 countries is 0.811. The comparable coefficients for all available data are 0.760 and 0.785 respectively.

and this may bias any results upwards since any variation in the reported figures is likely to represent a larger variation in the true figures.[69]

Figure 3.1



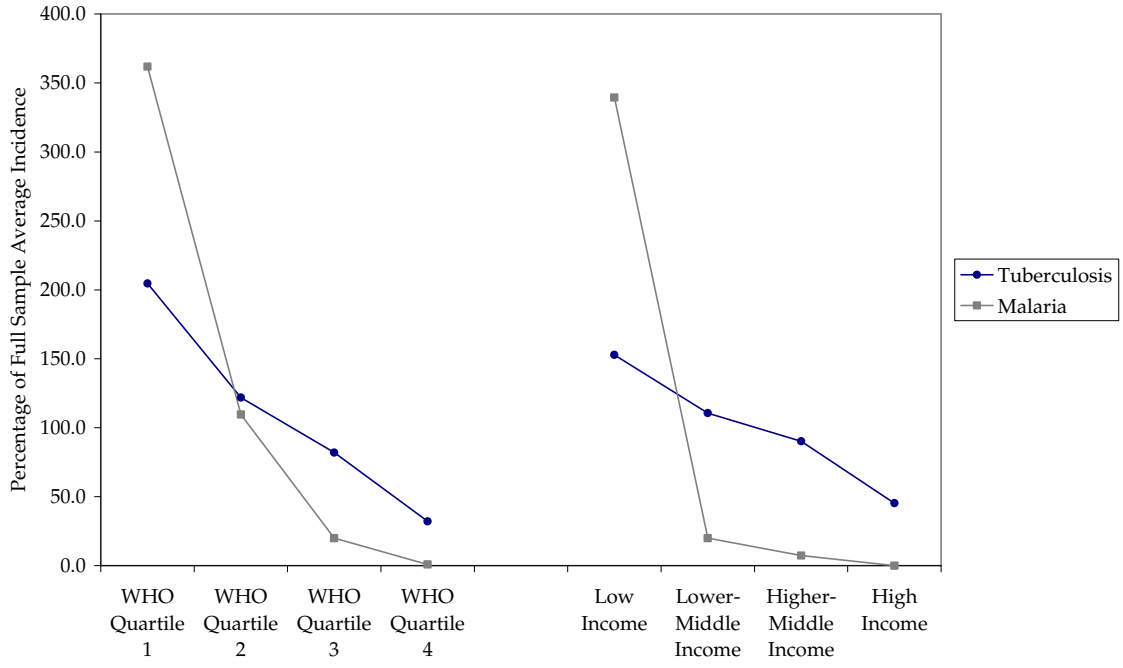
Several broader measures of health exist, as discussed in section 3.3, and this study includes three measures of mortality as proxies for other health issues. It would clearly be preferable to also include morbidity figures, but the author is not aware of any such series with sufficiently broad and deep coverage to be

[69] This upward bias in the coefficient on tuberculosis is due to the negative correlation between the true value (estimated incidence) and the error term (which is always negative in figure 3.1). This covariance enters the denominator of the coefficient estimate for the observed value (reported incidence), and thus reduces it, raising the size of the coefficient. This is however offset to an unknown degree by the larger than usual variance in the error term, due to the measurement error, which enters this same denominator positively. The net impact of poor measurement in this case is thus uncertain, but less negative than if the measurement error was random.

appropriate in this situation. Figures for average population life expectancy (LE_{it}) and infant mortality per thousand live births (IMR_{it}) are taken from the WDI. The other widely used mortality measure is adult survival rates. The ASR figures in the WDI are limited in their coverage, however a more complete series created from the unweighted average of male and female ASRs (ASR_{it}), as used by Bhargava et al, is used here.[70]

Figure 3.2

The Global Distribution of Tuberculosis and Malaria



[70] These were provided by Bhargava directly. The original source is unclear since the source quoted for the ASRs in Bhargava et al (2001), a World Bank technical report compiled by Eduard Bos (1998)entitled *Basic demographic, health and health systems data* does not appear to be available from the Bank, and Bos was not aware of such a publication when contacted (Personal Communication, December 2002).

In addition to these mortality figures this study also includes notification data for malaria cases per hundred thousand population (MAL_{it}) for comparison, since its omission may bias results for tuberculosis and it provides a useful counterpoint to tuberculosis. This data comes from two sources.[71] As figure 3.2 illustrates, tuberculosis is far more widely spread across the globe than malaria, which is largely constrained to the tropical, and poorest, parts of the world. Classifying the world either by income groups,[72] or by health system performance,[73] malaria places a far larger proportion of its burden on the poorest and on those least capable of dealing with it. This suggests that reducing tuberculosis in the poorest nations may not significantly raise productivity if a larger burden of malaria remains; but it also suggests that such a reduction may well have a considerable impact on the growth rate of medium-income countries. These two effects suggest that reduced tuberculosis may increase incomes, but it may not reduce absolute poverty as much as might have been hoped for.

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[71] For the years from 1962 to 1983 data is available from the World Health Statistics Annual for 1983, published by the World Health Organization (1983) p 791-5. From 1983 to 1997 data can be found in the Weekly Epidemiological Report (1999), table 1, p 267-70. This study takes average reported incidence for those years within each period for which data is available.

[72] These are the classifications used by the World Bank and are recalculated every six months. The classification used here was performed on the basis of 2000 Gross National Product per capita and the cut-off levels are US$ 755, US$ 2,995 and US$ 9,265 using the Atlas method. More details are available from http:// www.worldbank.org/data. The list of which countries fall into which categories is part of Appendix 1.

[73] These classifications were made by the World Health Organization (2001) Annex table 10, p 200-3 and are based on criteria of health outcomes and inequalities, system responsiveness, and the distribution of the system's financial burden. While such work was controversial in its findings, the results found using this classification are very similar to those found using the gap in potential Disability Adjusted Life Expectancy (DALE) which are found in the same table, and reflect the difference between achieved and potential DALE given existing levels of health expenditure in a country. The quartile cut-off values for health system performance are 0.483, 0.657 and 0.807; those for the DALE index are 0.554, 0.723 and 0.824.

Table 3.2

Health Data (Unbalanced Panel)

	All Periods				1980-1985	1986-1990	1991-1995	1996-2000
	Mean	Standard Deviation	Maximum	Average Countries	Mean			
Tuberculosis	63.0	77.6	653.6	196	63.9	56.0	61.2	70.8
Malaria	1,555.0	5,204	50,318	207	1,259	2,042	1,853	1,064
Adult Survival Rate	768.1	124.0	926.0	180	743.0	764.5	778.1	783.8
Infant Mortality Rate	43.4	41.8	186.6	191	57.4	50.2	45.6	40.5
Life Expectancy	64.5	10.8	80.5	191	62.8	64.3	65.0	65.7

Tuberculosis and malaria incidence rates are per 100,000 population; Adult Survival and Infant Mortality rates are per 1,000 live births and individuals reaching fifteen years. Life expectancy is in years.

Examining all the health data available, as shown in table 3.2, it can be seen that while the broad measures of health have improved in each successive period, reported rates of tuberculosis and malaria have followed different paths. Malaria appears to have peaked between 1986 and 1990, while tuberculosis is still on the rise today, having reached its nadir during the same period.

3.3 Methodology

Following the literature this study will use an augmented Solow growth model taking into account both health and ability forms of human capital. Following equation (2.3) this is most simple estimated in log-log form as:

$$\Delta \ln y_{it} = \alpha + \beta \ln (SCH_{it}) + \sum_{j=1}^{J} \gamma_j \ln (HEA_{it})^j + \psi (POPG_{it}) + \sum_{k=1}^{K} \theta_k X_{it}^k + \eta_i + \varepsilon_{it} \quad (3.1)$$

where SCH_{it} is represented either Barro-Lee or Cohen-Soto years of schooling; HEA_{it} is represented by some combination of variables found in table 3.2; and X_{it} is a selection of potential TFP explanitors, such as those found in table 3.3.

This paper will test four functional forms of the error term, each of which has been considered in previous studies. The first approach is a pooled model, where η_i is assumed to be equal to zero; an approach which is often used as a base case. The second form is to assume that η_i exists but is independent of other regressors: a random-effects model, as used by Bhargava et al (2001) and Bloom, Canning and Sevilla (2002). The third is where η_i is believed to be related to the regressors in an unspecified manner as N-1 time-constant, country-specific effects: a fixed-effects model, such as is used by Islam (1995) and McDonald and Roberts (2002). The fourth method is used if η_i is thought to be a function of the

mean value of the regressors in each country: a correlated-effects model, such as that used by Islam (1995).[74]

Table 3.3

Descriptive Statistics by Sample Size

Source		All Data	WDI / BL 91	WDI / PWT / BL 78	WDI / PWT BL / CS 68
(191)	Tuberculosis	60.9	58.4	52.7	50.4
(207)	Malaria	1,555	1,704	1,829	1,471
*	Adult Survival Rates	768	782	788	794
*	Infant Mortality Rates	48.4	46.4	44.3	41.7
*	Life Expectancy	64.5	65.4	66.2	66.7
WDI	Growth in GDP	678	747	754	758
(119)	Initial GDP	6,263	7,943	7,567	7,738
	Gross Capital Formation	22.6	22.1	22.0	21.7
	Gov't Consumption Exp.	16.2	15.2	15.0	14.9
	Openness to Trade	76.9	70.9	64.6	60.4
	Population Growth	1.77	1.68	1.68	1.65
PWT	Growth in GDP	1,338		1,426	1,368
(103)	Initial GDP	16,426		18,537	19,085
	Investment	15.2		16.1	16.1
	Gov't Expenditure	20.0		18.5	17.6
	Openness to Trade	66.9		63.2	59.1
	Population Growth	1.81		1.70	1.67
BL (109)	Years of Schooling	5.51	5.65	5.73	5.81
CS (95)	Years of Schooling	5.94			6.64

The 'All Data' column contains averages for the largest available balanced panel, with the exception of variables marked (*) for whom all available data is included; for each variable or group of variables the number of countries used is provided in parentheses in the first column. The three subsequent data columns contain the number of countries indicated at their heads.

[74] A priori it might be expected that these last two are the most realistic given that technology levels, which appear to constitute a large part of the unobserved effect captured in the error term, are likely to dependent on national characteristics, but as shall be seen in section 4.1 it is possible to test each model for appropriateness.

Sample sizes for these regressions are determined largely by availability of balanced panels of data for the series to be used. As table 3.3 shows the broadest selections of data shown in tables 3.1 and 3.2 are rarely complete, and each source offers a different sample of countries. The core sample of 91 countries contains complete information for all periods from the Barro-Lee schooling dataset and from the WDI dataset, while the sample of 78 countries also covers the PWT dataset, and the 68 country sample ensures coverage of the Cohen-Soto schooling measures as well. As the sample size falls, almost all variables move in what is thought to be an improving direction at a steady rate. The two exceptions to this are tuberculosis and malaria. Early restrictions on malaria actually raise the average burden of disease above the average for the full sample, and the tuberculosis rate is far closer to the full sample average when the sample size is 91 than when it is 78 or 68 countries.

4. RESULTS

4.1 Initial findings

The first step in the process was to test for the strongest specification of the model. The basic model as set out in equation (3.1) was run using a MRW-style growth model augmented by Barro-Lee years of schooling and the tuberculosis incidence rate, both in log form. To take account of TFP variation variables for population growth, the openness of the economy to trade, and the level of government consumption expenditure are included, all in log form with the exception of population growth.[75]

Table 4.1 provides the results of running this equation as a pooled, a random-effects, a fixed-effects, and a correlated-effects model on the broadest possible sample. The Breusch-Pagan LM test in column two has as its null that the η_i section of the error term is equal to zero, which would indicate a pooled model is more appropriate than the random-effects model, however this hypothesis is strongly rejected. The fixed-effects model can be also be tested against the pooled regression, that is that the 90 η_i terms are jointly equal to zero, and again the use of the cross-sectional model is strongly rejected. It is also possible to test for the appropriateness of a random-effects model relative to that of a fixed-effects model, since in the case where it is unbiased the coefficients arrived at in

[75] Unless otherwise specified all regressions are run as fixed-effects models; the dependent variable is the difference in the log of GDP across four periods from 1981 to 2000; figures in parentheses are t-statistics; and significance is indicated by * at the 10%, ** at the 5%, and *** at the 1% level.

Table 4.1

Specification Regressions

Dependent Variable: Difference in log of GDP_{it} and GDP_{it-1} over five year periods (1981-2000)

	Pooled Regression (1.1)	Random-Effects GLS (1.2)	Fixed-Effects LSDV (1.3)	Correlated-Effects GLS (1.4)
Log of Initial GDP per capita	0.003 (0.49)	-0.003 (0.32)	-0.269 (7.53)***	-0.292 (7.58)***
Log of Gross Capital Formation	0.196 (9.21)***	0.195 (7.97)***	0.154 (4.81)***	0.153 (4.43)***
Log of Gov't Consumption Exp.	-0.056 (3.15)***	-0.077 (3.51)***	-0.126 (3.82)***	-0.118 (3.34)***
Log of Trade	0.009 (0.73)	0.024 (1.54)	0.160 (4.90)***	0.160 (4.55)***
Average Annual Pop. Growth	-0.009 (1.24)	-0.003 (0.39)	0.026 (2.21)**	0.025 (1.99)**
Log of Years of Schooling (BL)	0.011 (0.73)	0.029 (1.44)	0.105 (2.58)**	0.107 (2.43)**
Log of Average TB Incidence	-0.010 (1.52)	-0.013 (1.62)	-0.033 (2.31)**	-0.041 (2.72)***
Constant	-0.416 (4.76)***	-0.398 (3.60)***	1.264 (4.04)***	-0.436 (4.26)***
Observations	364	364	364	364
R-squared	0.29		0.37	
Number of Countries		91	91	91
Breusch-Pagan LM Test: X^2 (1)		18.78 (0.000)***		33.16 (0.000)***
Joint Test on Country Dummies: F (90,266)			3.50 (0.000)***	
Hausmann Specification Test: X^2 (7)		90.71 (0.000)***		0.00 (1.000)

Absolute value of t-statistics in parentheses, except for tests where probability that the test statistic is zero is provided. * significant at 10%; ** significant at 5%; *** significant at 1%

The fixed-effects model contains N-1, in this equation 90, country dummies; the correlated-effects model includes the country-specific mean values of the seven explanitory variables. In both cases these variables form part of the error term, and are not shown in this table.

the former should not be significantly different from those in the latter, and in this situation it is a more efficient estimator. This can be tested using a Hausmann specification test, with a null hypothesis of no difference between the two forms. In column 4 of the table however it is clear that the random-effects model is not appropriate.

Since the correlated-effects model is similar to the random-effects model in its use of a Generalised Least Squares formula, regression (1.4) can be tested against the fixed-effects model in a similar manner to regression (1.2). Strikingly, the inclusion of seven terms containing the mean of each country's explanitory variables gives coefficients for the seven exogenous regressors extremely close to those of the fixed-effects model, and it is not possible to reject the hypothesis that they are the same. On the other hand, the presumed benefit of using a correlated-effects model, that is gives more efficient results, does not appear to be significantly borne out by the two equations: as with the coefficients, the standard errors for regressions (1.3) and (1.4) are very similar indeed – although systematically slightly larger for the GLS regression. Since the fixed-effects model has been more widely used in the literature, and because it allows for a more nuanced interaction between the regressors and the error term, regression (1.3) will be used as the standard form for this study.

After considering the most appropriate form of the equation it is possible to turn to the coefficients themselves, focusing in particular on the latter two regressions. Following both theory and standard results these regressions suggest that conditional convergence is occurring among the 78 countries in this panel; that more investment, openness to trade, and schooling have a positive influence on growth; and that government consumption and poor health, here proxied by tuberculosis, have a negative impact on the economic condition of a nation. Population growth also enters this regression with a significantly positive coefficient but as shall be seen below this effect is the least robust of all the variables here. As Bloom, Canning and Sevilla (2001) discuss at length, the effect of high population growth differs significantly depending on the existing age structure of a given population, and this result will therefore be treated with caution.

Focusing on tuberculosis, regression (1.3) suggests that a 10 percent fall in the average incidence of the disease, as reported to the WHO, is correlated with a 0.33 percent rise in the rate at which GDP per capita grows. The mean figure for GDP growth in this sample is US$ 754 across a five-year period, and it covers 80 percent of the world's population. Even assuming that no other country is affected by tuberculosis, this effect amounts to some US$ 11.9 billion over a five year period, or US$ 2.4 billion per year. Regression (1.4) suggests that this figure is an underestimate, and that the effect is in fact more

than 0.4 percent per year. These figures are rather less than those presented by Gallup and Sachs (2001) for malaria, who estimate that a 10 percent reduction in malaria could lead to a 0.3 percent rise in growth each year.[76]

While this result is significant, there are various issues that should be borne in mind. First, it is very likely that the large effect seen here for tuberculosis is due at least in part to a high degree of correlation with other health variables not included in the regression, which would thus inflate its importance. Second, this effect may be a result of the particular variables and sample size used in this regression. Third, the effect may be more nuanced than this blanket regression suggests, varying according to the characteristics of particular countries. These three possibilities are explored in the following sections.

4.2 Results using broader measures of health

Since it is unusual for a country to be burdened only with one disease, it seems reasonable to explore the impact of including other measures of poor health in the regression alongside tuberculosis. Table 4.2 details regressions on the panel of 91 countries using tuberculosis, malaria and three broad measures of health.

[76] The difference is that Gallup & Sachs find that the fall in Malaria will raise growth by 0.3 percent, for example from 1.9 to 2.2 percent growth, while this paper finds that it will improve the growth **rate** by 0.33 percent, that is from 1.89 to 1.96 percent – the actual figures using the sample from equation (1.3).

As life expectancy is heavily linked to adult and infant survival rates they are not entered in the same regressions. The first point of note is that infant mortality is a very strong predictor/cause of poor economic growth. Since those under the age of one do not produce output over the five years after their birth it seems

Table 4.2

Health Data Regressions

	(2.1)	(2.2)	(2.3)	(2.4)	(2.5)	(2.6)
Log of Initial GDP per capita	-0.255	-0.275	-0.272	-0.343	-0.286	-0.350
	(7.21)***	(7.64)***	(7.59)***	(9.45)***	(8.04)***	(9.65)***
Log of Gross Capital Formation	0.153	0.161	0.152	0.164	0.140	0.155
	(4.68)***	(4.95)***	(4.75)***	(5.39)***	(4.39)***	(5.06)***
Log of Gov't Consumption Exp.	-0.114	-0.121	-0.122	-0.101	-0.128	-0.104
	(3.41)***	(3.63)***	(3.69)***	(3.21)***	(3.93)***	(3.33)***
Log of Trade	0.159	0.159	0.153	0.110	0.152	0.110
	(4.84)***	(4.89)***	(4.62)***	(3.43)***	(4.73)***	(3.43)***
Ave. Annual Pop. Growth	0.027	0.026	0.023	0.026	0.029	0.028
	(2.32)**	(2.21)**	(1.89)*	(2.34)**	(2.50)**	(2.52)**
Log of School Years (BL)	0.077	0.095	0.095	-0.012	0.079	-0.012
	(1.86)*	(2.27)**	(2.27)**	(0.27)	(1.82)*	(0.28)
Log of Average TB Incidence		-0.034	-0.027	-0.007	-0.031	-0.010
		(2.39)**	(1.81)*	(0.47)	(2.17)**	(0.66)
Log of Average malaria Incidence	-0.002	-0.003				
	(1.04)	(1.22)				
Log of Pop. Life Expectancy			0.148			
			(1.07)			
Log of Infant Mortality Rate				-0.165		-0.155
				(5.60)***		(5.10)***
Log of Adult Survival Rate					0.408	0.203
					(2.51)**	(1.26)
Constant	1.063	1.306	0.695	2.592	-1.208	1.305
	(3.54)***	(4.15)***	(1.12)	(6.83)***	(1.15)	(1.16)
Observations	364	364	364	364	363	363
Number of Countries	91	91	91	91	91	91
R-squared	0.36	0.38	0.38	0.44	0.39	0.45

reasonable to surmise that this effect reflects the link between better general health and good infant health. This may reflect good quality healthcare systems such as those outlined in section 2.7.

The two measures of adult and whole-life survival suggest that mortality during productive years is more closely linked to lower output than a longer life-span per se. This may be because the effect of longer life expectancy provides an indirect stimulus to growth, for example through higher returns to human capital investments, while more working age mortality directly reduces the stock of experienced workers, as well as having indirect effects on investment.

It is not clear why malaria, a disease which has previously been found to be significantly linked to lower growth, does not have a significant impact on growth, either alone or in conjunction with tuberculosis. The form in which it enters the regression and period of time studied differ from the work of McCarthy, Wolf and Wu (2000) and Gallup and Sachs (2001), and this may well have affected the results. Tuberculosis remains a significant factor in all regressions except those containing infant mortality rates – which also wash out adult mortality and schooling effects – and the magnitude of the effect seems reasonably stable, at between 0.27 and 0.34 percent per 10 percent lower incidence, across regressions (2.2), (2.3) and (2.5).

One crucial health factor which is not controlled for in these regressions, and which may have a significant effect on the results, is incidence of HIV/AIDS. The impact of this disease in Africa over the second half of the period under study was huge, and since tuberculosis is often a co-infection of HIV it is quite likely that the tuberculosis coefficient is picking up some of the effects of HIV. Unfortunately at this time consistent reports of HIV incidence are not available. Concern over this matter may be somewhat tempered by considering that in the absence of a cure for HIV, infection leads to death in a relatively short and fixed period of time after the appearance of symptoms, particularly in the most heavily affected nations. This unfortunate truth means that HIV infection rates should be highly correlated with changes in life expectancy, and as was seen in regression (2.3), the inclusion of life expectancy as an explanitory variable did not significantly alter the original findings of table 4.1.

4.3 Tests for robustness

In order to ascertain whether the results found in regression (1.3) and table 4.2 are a reflection of a robust and stable effect this section will test whether different data sources, different sample sizes, and different dynamics affect the results. In table 4.3 the first three regressions regress per person GDP from the WDI dataset over two reduced samples (regressions 3.1 and 3.2) and using Cohen-Soto instead

of Barro-Lee for years of schooling (regression 3.3). The other two equations regress per worker GDP on variables from the PWT, first using Barro-Lee years

Table 4.3

Regressions using other Datasets

	WDI (3.1)	WDI (3.2)	WDI (3.3)	PWT (3.4)	PWT (3.5)
Log of Initial GDP per capita	-0.289	-0.284	-0.295		
	(7.52)***	(7.08)***	(7.51)***		
Log of Gross Capital Formation	0.170	0.163	0.155		
	(4.84)***	(4.56)***	(4.44)***		
Log of Gov't Consumption Exp.	-0.099	-0.133	-0.121		
	(3.01)***	(3.92)***	(3.63)***		
Log of Trade	0.152	0.136	0.126		
	(4.36)***	(3.81)***	(3.61)***		
Ave. Annual Population Growth	0.026	-0.001	0.000		
	(2.23)**	(0.08)	(0.02)		
Log of Initial GDP per worker				-0.409	-0.419
				(8.59)***	(8.20)***
Log of Gross Capital Formation				0.102	0.164
				(2.93)***	(3.69)***
Log of Gov't Expenditure				-0.116	-0.093
				(3.41)***	(2.63)***
Log of Trade				0.105	0.072
				(2.77)***	(1.77)*
Average Annual Pop. Growth				0.003	-0.046
				(0.18)	(2.00)**
Log of School Years (BL)	0.114	0.157		0.069	
	(2.61)***	(3.11)***		(1.16)	
Log of School Years (CS)			0.202		0.117
			(4.29)***		(1.72)*
Log of TB Incidence	-0.036	-0.042	-0.048	-0.024	-0.027
	(2.50)**	(2.66)***	(3.06)***	(1.22)	(1.20)
Constant	1.347	1.510	1.544	3.500	3.513
	(4.10)***	(4.36)***	(4.55)***	(7.18)***	(6.51)***
Observations	312	272	272	312	272
Number of Countries	78	68	68	78	68
R-squared	0.39	0.43	0.45	0.32	0.36

The dependent variable is GDP per capita from the WDI in regressions (1-3) and GDP per worker from the PWT in regressions (4-5). See text or appendix 2 for details.

of schooling, then the Cohen-Soto figures. In terms of schooling, neither measure appears to make a significant difference to coefficients on other variables, although the estimates for Cohen-Soto suggest a higher return to schooling itself. It is also noticeable that as the samples get smaller, the coefficient on tuberculosis rises. The major point to note in this table is that while effects are broadly similar between the WDI and the PWT for series drawn from within them, the two sets differ in the effect they find for tuberculosis. Although the coefficients are lower for PWT regressions than for WDI ones, the differing results are also due to higher standard error terms using per worker figures. While the tuberculosis coefficients in regressions (3.4) and (3.5) are not significantly different from zero, the magnitudes of the effects found are not far from those seen in previous regressions.

The panel nature of this dataset allows for the study of the dynamic effects of tuberculosis on economic growth, which is useful given the previously noted existence of two-way causality between health and income levels. Table 4.3 presents regressions of per person GDP on current and lagged tuberculosis notification rates over three periods from 1986-2000. This should pick up effects running from better health to higher growth, but not those which run in the opposite direction. In order to keep the regressions somewhat comparable across measures of schooling, sample sizes of 78 and 68 countries are used. It is unfortunate that regression (4.1), using WDI economic data

and Barro-Lee schooling, does not produce a significant effect for tuberculosis over this fifteen year period, since this makes comparison with regression (4.2) less straightforward. For both Barro-Lee and Cohen-Soto data it can be seen however that using lagged values supports a hypothesis of tuberculosis having an effect of between 0.2 and 0.4 percent per 10 percent reduction in notified incidence, and that lagged values give more significant results for

Table 4.4

Regressions using Lagged Tuberculosis Data

	(1)	(2)	(3)	(4)
Log of Initial GDP per capita	-0.389 (8.27)***	-0.399 (8.59)***	-0.412 (8.06)***	-0.405 (8.13)***
Log of Gross Capital Formation	0.160 (3.99)***	0.160 (4.02)***	0.147 (3.55)***	0.151 (3.65)***
Log of Gov't Consumption Exp.	-0.090 (2.12)**	-0.094 (2.21)**	-0.104 (2.39)**	-0.099 (2.30)**
Log of Trade	0.195 (4.26)***	0.198 (4.35)***	0.145 (2.96)***	0.147 (3.03)***
Average Annual Pop. Growth	0.033 (2.93)***	0.032 (2.84)***	-0.009 (0.50)	-0.005 (0.26)
Log of School Years (BL)	0.100 (1.56)	0.110 (1.73)*		
Log of School Years (CS)			0.210 (2.55)**	0.227 (2.71)***
Log of Average TB Incidence	-0.005 (0.27)		-0.039 (1.66)*	
Lagged Log of Average TB Incidence		-0.023 (1.45)		-0.032 (1.84)*
Constant	1.885 (4.70)***	2.003 (5.13)***	2.343 (5.38)***	2.192 (5.44)***
Observations	234	234	204	204
Number of Countries	78	78	68	68
R-squared	0.49	0.50	0.50	0.51

These regressions cover three periods of five years from 1986 to 2000. See text for details.

tuberculosis than directly comparable regressions using contemporaneous data. Comparing regressions (3.1) and (4.2), and (3.3) and (4.4), which cover different time periods but include the same variables, it can be seen that in both cases the coefficient is reduced by around one-third when the lagged tuberculosis figure is used. This suggests that although some of the effect seen in earlier regressions is attributable to higher income lowering disease levels, the majority of the effect flows in the other direction.[77]

4.4 Results by sub-groups

Dividing the 91 countries into smaller sample, by geography (table 4.5), by income (table 4.6) or by health system performance (table 4.7), it is clear that these more limited samples make inference on the tuberculosis data difficult to conduct, since standard errors rise, and the significance of results diminish. Table 4.5 suggests that the strongest effect among the areas covered is for the richest group of nations, the OECD, and the weakest by some way for Africa. The former result may be an artefact of the variation found within what is relatively homogenous sample.[78] In Africa it may be that other health issues

[77] Cohen and Soto (2001) suggest that the effect of schooling on growth is a log-linear one and that schooling should therefore enter growth regressions in levels rather than in logs. Running regression (1.3) with levels of Barro-Lee schooling affects all the regressors' coefficients, but neither radically nor in a systematic manner. The absolute value of the coefficient on tuberculosis falls from 0.033 to 0.024, and the t-statistic falls from (2.31)** to (1.77)*.

[78] It should be noted that the maximum level of tuberculosis seen in the OECD over this twenty-year period was 234 cases per 100,000 population, a not inconsiderable figure, but the median

predominate, such as HIV/AIDS or malaria, or it may just be that investment in human capital, in both its education and health forms, is not consistently met with a positive return. Across the less developed world, regression (5.3) suggests that the effect of tuberculosis is not clearly significant, and that the coefficient is markedly smaller than that which has previously been found for the full sample.

Table 4.5

Regressions by Geographic Region

Region	All (5.1)	OECD (5.2)	Non-OECD (5.3)	Africa (5.4)	Americas[1] (5.5)
Log of Initial GDP	-0.269	-0.242	-0.299	-0.391	-0.352
	(7.53)***	(3.74)***	(6.95)***	(4.59)***	(3.15)***
Log of Gross Capital Formation	0.154	0.180	0.153	0.201	0.139
	(4.81)***	(2.88)***	(4.11)***	(3.11)***	(1.84)*
Log of Gov't Consumption Exp.	-0.126	-0.490	-0.117	-0.125	-0.096
	(3.82)***	(4.88)***	(3.12)***	(1.57)	(1.39)
Log of Trade	0.160	0.096	0.152	0.064	0.140
	(4.90)***	(1.76)*	(3.88)***	(0.86)	(1.66)
Ave. Annual Pop. Growth	0.026	-0.071	0.029	0.031	0.036
	(2.21)**	(2.44)**	(2.15)**	(1.74)*	(0.84)
Log of School Years (BL)	0.105	0.381	0.082	-0.017	0.301
	(2.58)**	(2.92)***	(1.73)*	(0.23)	(2.36)**
Log of Average TB Incidence	-0.033	-0.041	-0.015	0.003	-0.020
	(2.31)**	(1.39)	(0.89)	(0.08)	(0.47)
Constant	1.264	2.257	1.237	1.861	1.450
	(4.04)***	(3.14)***	(3.58)***	(2.88)***	(1.55)
Observations	364	104	260	88	84
Number of Countries	91	26	65	22	21
R-squared	0.37	0.49	0.40	0.47	0.47

[1] Available non-OECD nations in the Americas (not Mexico, Canada or the United States).

figure is just 16.8 cases. The only country with more than 100 cases per 100,000 in any period across the OECD was South Korea, prior to 1996.

Taking a slightly less crude approach than physical location, table 4.6 divides countries according to their income per capita at the end of the period of study, and table 4.7 divides them according to their ranking in the World Health Organization's (2001) health system performance index. Both tables suggest that the most significant effects of tuberculosis are felt among higher performing economies. In table 4.6 more tuberculosis incidence is associated with higher growth, although not significantly so, among the first two

Table 4.6

Regressions by World Bank Income Category

World Bank Income Category	Low (6.1)	Lower-Middle (6.2)	Upper-Middle (6.3)	High (6.4)
Log of Initial GDP	-0.385	-0.385	-0.360	-0.250
	(5.48)***	(5.27)***	(3.74)***	(4.47)***
Log of Gross Capital Formation	0.241	0.104	0.235	0.181
	(4.55)***	(1.64)	(2.54)**	(2.71)***
Log of Gov't Consumption Exp.	-0.107	-0.122	-0.178	-0.503
	(1.84)*	(2.27)**	(1.85)*	(5.75)***
Log of Trade	0.052	0.221	0.143	0.098
	(0.89)	(3.95)***	(1.48)	(1.77)*
Ave. Annual Pop. Growth	0.038	0.036	-0.067	-0.063
	(2.40)**	(1.35)	(0.99)	(2.80)***
Log of School Years (BL)	-0.048	0.186	0.185	0.559
	(0.75)	(2.15)**	(1.01)	(4.11)***
Log of Average TB Incidence	0.016	0.027	-0.149	-0.031
	(0.72)	(0.86)	(2.81)***	(1.19)
Constant	1.520	1.426	2.422	1.995
	(3.23)***	(2.49)**	(2.50)**	(3.17)***
Observations	100	104	52	108
Number of Countries	25	26	13	27
R-squared	0.55	0.48	0.57	0.48

income categories, and with lower growth among richer nations. This effect is most significant in upper-middle income nations, where the point estimate is four to five times what was seen in earlier regressions. In table 4.7 the coefficients in regressions (7.2) and (7.3) are close to those seen in the core regressions, while countries with poorly performing health systems show little response to tuberculosis, and those with high incomes show rather more.

Table 4.7

Regressions by Health System Performance Rating

WHO System Performance	Quartile 1 (7.1)	Quartile 2 (7.2)	Quartile 3 (7.3)	Quartile 4 (7.4)
Log of Initial GDP	-0.448 (4.45)***	-0.329 (4.51)***	-0.266 (4.13)***	-0.230 (3.68)***
Log of Gross Capital Formation	0.206 (2.57)**	0.147 (2.95)***	0.213 (2.80)***	0.234 (3.83)***
Log of Gov't Consumption Exp.	-0.103 (1.07)	-0.113 (2.01)*	-0.047 (0.80)	-0.363 (5.59)***
Log of Trade	0.067 (0.67)	0.146 (2.40)**	0.173 (3.00)***	0.115 (1.98)*
Ave. Annual Pop. Growth	0.032 (1.62)	0.051 (1.46)	0.012 (0.41)	-0.070 (2.79)***
Log of School Years (BL)	-0.009 (0.09)	0.151 (1.96)*	0.143 (1.38)	0.537 (3.87)***
Log of Average TB Incidence	0.008 (0.21)	0.046 (1.34)	0.037 (1.12)	-0.068 (2.79)***
Constant	2.032 (2.69)***	1.036 (1.85)*	0.409 (0.65)	1.282 (2.00)**
Observations	72	76	96	116
Number of Countries	18	19	24	29
R-squared	0.46	0.57	0.44	0.51

The results in this section make clear the fragility of the effects found for tuberculosis, although variability is also seen in the effects of other variables included in the regressions, especially among the poorest, and poorest performing, nations. Some of this variation may be the result of the more limited sample sizes, but it also acts as a reminder that caution should be exercised in conducting 'convergence club' analysis – it is often the differences between such clubs which allow causal relationships to be identified.

These regressions appear to show that the effect of tuberculosis is most strongly felt in middle to high performance nations. This might be because incidence of tuberculosis in these countries reflects specific weaknesses in the healthcare system, weaknesses which are not reflected in other data.

5. CONCLUSION

The literature on factors affecting economic growth has in recent years supported the result that better health leads to higher productivity, and thus at a macroeconomic level to higher levels of economic well-being. Previous empirical findings that disease burden has a significant impact on economic growth are supported in the paper in the case of tuberculosis. The magnitude of the effect appears to be in the range of a 0.2 to 0.4 percent increase in the rise of per capita income for a ten percent lower level of notified incidence of tuberculosis. This would amount to between US$ 1.8 and US$ 3.6 billion per annum in increased output, if this figure is representative of the whole world's population.[79]

This result should however be tempered by findings using other data sources such as the PWT, and using lagged values of tuberculosis, which suggest that these results are not very robust, although the result that higher notified incidence of tuberculosis reduces growth appears to remain while the sample is considered as a whole. When the sample is divided according to various criteria the effect of disease, and that of other variables, becomes rather harder to distinguish. More reliable data on tuberculosis incidence, and on co-infections, particularly HIV/AIDS, would allow stronger conclusions to be drawn.

[79] This is not an unreasonable assumption: the estimated average national tuberculosis incidence for the period 1996-2000 (World Health Organization, 2002) for the 91 countries included in regression (1.3) was 56.9 cases per 100,000 population. The figure for the 98 countries outside the sample the average was 53.9.

The link between tuberculosis and diminished growth suggests that there is a role for health programmes such as DOTS in improving not only the health of those living in the developing world, but also such individual's wealth. Any policy would need to take into account the problems involved in scaling-up treatments in countries with limited healthcare staff and health infrastructure, as outlined by Filmer, Hammer and Pritchett, and the Commission on Macroeconomics and Health. Both sets of guidelines appear to support a dedicated programme to treat tuberculosis using community based methods such as those of DOTS, which do not require long periods of hospitalisation or high-level infrastructure.

While it must be borne in mind that there are potential problems both with the theoretical grounding and empirical estimation of growth equations such as those undertaken in this study, particularly given the measurement error present in the notification data used as the core of this study, a tentative conclusion that further funding for tuberculosis treatment is worthwhile can be drawn. Given that the worldwide implementation of a DOTS style treatment programme is believed require only an additional US$ 300 million per annum, as outlined by Floyd et al (2002), the benefits appear to considerably outweigh the costs of such a programme by some considerable margin.

BIBLIOGRAPHY

'Processed' refers to documents not usually available through libraries.

Baer, Werner, Campino, A and Cavalcanti, T (2001) "Health in the development process: The case of Brazil" *Quarterly Review of Economics and Finance* Vol 41, p 405-25

Barro, Robert J (1991) "Economic Growth in a Cross Section of Countries" *Quarterly Journal of Economics* Vol 106 (2), p 407-43

Barro, Robert J and Lee, J (1993) "International Comparisons of Educational Attainment" *Journal of Monetary Economics* Vol 32, p 363-94

Barro, Robert J (1997) *Determinants of Economic Growth: an Cross-Country Empirical Study* Cambridge, Mass: MIT Press

Barro, Robert J and Lee, J (2001) "International Comparisons of Educational Attainment: Updates and Implications" *Oxford Economic Papers* Vol 3, p 541-63

Bhargava, Alok, Jamison, DT, Lau, J and Murray, CJL (2001) "Modeling the Effects of Health on Economic Growth" *Journal of Health Economics* Vol 20, p 423-40

Bloom, David E, Canning, D and Sevilla, J (May 2002) *Health, Worker Productivity, and Economic Growth* processed

Bloom, David E, Canning D and Sevilla, J (2001) *Economic Growth and the Demographic Transition* National Bureau of Economic Research Working Paper, No 8685

Bloom, David E and Mahal, AS (1997) "Does the AIDS Epidemic Threaten Economic Growth?" *Journal of Econometrics* Vol 77, p 105-124

Borgdorff, Martien W, Floyd, K and Broeckmans, JF (2001) *Interventions to Reduce Tuberculosis Mortality and Transmission in Low and Middle-Income Countries: Effectiveness, Cost-Effectiveness, and Constraints to Scaling Up* Commission on Macroeconomics and Health Working Papers, Working Group 5; Paper 8

Cohen, Daniel and Soto, M (2001) *Growth and Human Capital: Good Data, Good Results* OECD Development Centre Technical Papers, No 179

Commission on Macroeconomics and Health (2001) *Macroeconomics and Health: Investing in Health for Economic Development* Geneva: World Health Organization; Available from http://www.cmhealth.org

Corbett, Elizabeth L, Watt, CJ, Walker, N, Maher, D, Williams, BG, Raviglione, MC, and Dye C (2002) *The Growing Burden of Tuberculosis: Global Trends and Interactions with the HIV Epidemic* processed

Croft, RA and Croft, RP (1998) "Expenditure and Loss of Income incurred by Tuberculosis patients before reaching effective treatment in Bangladesh" *International Journal of Tuberculosis and Lung Disease* Vol 2 (3) p 252-4

Cuddington, John T (1993) "Modeling the Macroeconomic Effects of AIDS, with an Application to Tanzania" *World Bank Economic Review* Vol 7 (2), p 173-89

De Long, J Bradford (1988) "Productivity Growth, Convergence, and Welfare: Comment" *American Economic Review* Vol 78 (5) p 1138-54

Dye, Christopher, Scheele, S, Dolin, P, Pathania, V and Raviglione, MC (1999) "Global Burden of Tuberculosis: Estimated Incidence, Prevalence, and Mortality by Country" *Journal of the American Medical Association* Vol 282 (7) p 677-86

Dye, Christopher, Espinal, MA, Watt, CJ, Mbiaga, C and Williams, BG (2002) "Worldwide Incidence of Multidrug-Resistant Tuberculosis" *Journal of Infectious Diseases* Vol 185, p 1197-202

Filmer, Dean, Hammer JS and Pritchett, LH (2000) "Weak Links in the Chain: A Diagnosis of Health Policy in Poor Countries" *World Bank Research Observer* Vol 15 (2), p 199-224

Filmer, Dean, Hammer JS and Pritchett, LH (2002) "Weak Links in the Chain II: A Prescription for Health Policy in Poor Countries" *World Bank Research Observer* Vol 17 (1), p 47-66

Floyd, Katherine, Blanc, L, Raviglione, M and Lee, J-W (2002) "Resources Required for Global Tuberculosis Control" *Science* Vol 295 (March 15th), p 2040-1

Fogel, Robert W (1994) "Economic Growth, Population Theory, and Physiology: The Bearing of Long-Term Processes on the making of Economic Policy" *American Economic Review* Vol 84 (3), p 369-95

Gallup, John L and Sachs, JD (2001) *The Economic Burden of Malaria* Commission on Macroeconomics and Health Working Papers, Working Group 1; Paper 10

Gallup, John Luke and Sachs JD (1999) *Geography and Economic Development* Center for International Development at Harvard University, CID Working Paper, No 1

Heston, Alan, Summers, R and Aten, B (2002) *Penn World Table Version 6.1* Centre for International Comparisons at the University of Pennsylvania; Available from http://pwt.econ.upenn.edu

Islam, Nazrul (1995) "Growth Empirics: a panel data approach" *Quarterly Journal of Economics* Vol 110, p 1127-70

Kalemli-Ozcan, Sebnem, Ryder, HE, Weil, DN (2000) "Mortality Decline, Human Capital Investment, and Economic Growth" *Journal of Development Economics* Vol 62, p 1-23

Kamolratanakul, P, Sawert, H, Kongsin, S, Lermaharit, S, Sriwongsa, J, Na-Songkhla, S, Wangmanee, S, Jittimanee, S and Payanandana, V (1999) "Economic Impact of Tuberculosis at the Household Level" *International Journal of Tuberculosis and Lung Disease* Vol 3 (7) p 596-602

Lucas, Robert (1988) "On the Mechanics of Economic Development" *Journal of Monetary Economics* Vol 22, p 3-42

Mankiw, Gregory N, Romer, D and Weil, DN (1992) "A Contribution to the Empirics of Economic Growth" *Quarterly Journal of Economics* Vol 107, p 407-37

Mathers, CD, Stein, C, Fat, DM, Rao, C, Inoue, M, Tomijima, N, Bernard, C, Lopez, AD and Murray, CJL (2002) *Global Burden of Disease 2000: Version 2 Methods and Results* Global Programme on Evidence for Health Policy Discussion, Paper No. 50

McCarthy, F Desmond, Wolf, H and Wu, Yi (2000) *Malaria and Growth* World Bank Working Papers, No 2303

McDonald, Scott and Roberts, J (2002) "Growth and multiple forms of human capital in an augmented Solow model: a panel data investigation" *Economic Letters* Vol 74, p 271-6

Murray, Christopher JL, Styblo, K and Rouillon, A (1993) "Tuberculosis" in Jamison, JT, Mosley, WH, Measham, AR and Bobadila, JL (eds.) *Disease Control Priorities in Developing Countries* New York: Oxford University Press

Murray, Christopher JL and Lopez, AD (eds.) (1996) *The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020* Cambridge, Mass: Harvard University Press, 2 Volumes

Murray, Christopher JL, Lopez, AD, Mathers, CD and Stein, C (2001) *The Global Burden of Disease 2000 project: aims, methods and data sources* World Health

Organization: Global Programme on Evidence for Health Policy, Discussion Paper No. 36

Pritchett, Lant (2001) "Where has all the Education gone?" *World Bank Economic Review* Vol 15 (3), p 367-91

Pritchett, Lant and Summers, LH (1996) "Wealthier is Healthier" *Journal of Human Resources* Vol 31 (4), p 841-868

Romer, Paul M (1986) "Increasing Returns and Long-Run Growth" *Journal of Political Economy* Vol 94 (5), p 1002-37

Sawert, Holger, Kongsin, S, Payanandan, V, Akarasewi, P, Nunn, PP, and Raviglione, MC (1997) "Costs and Benefits of Improving Tuberculosis Control: the Case of Thailand" *Social Science and Medicine* Vol 44 (12) p 1805-16

Schultz, T Paul (1999) "Health and Schooling Investments in Africa" *Journal of Economic Perspectives* Vol 13 (3), p 67-88

Schultz, T Paul and Tansel, A (1997) "Wage and labor supply effects of illness in Côte D'Ivoire and Ghana: instrumental variable estimates for days disabled" *Journal of Development Economics* Vol 53, p 251-86

Solow, Robert M (1956) "A Contribution to the Theory of Economic Growth" *Quarterly Journal of Economics* Vol 70, p 65-94

Solow, Robert M (2001) "Applying Growth Theory across Countries" *World Bank Economic Review* Vol 15 (2) p 283-8

Soto, Marcelo (2002) *Rediscovering Education in Growth Regressions* OECD Development Centre Technical Papers, No 202

Strauss, John and Thomas, D (1998) "Health, Nutrition and Economic Development" *Journal of Economic Literature* Vol 36, p 766-817

Suárez, Pedro G, Floyd, K, Portocarrero, J, Alarcón, E, Rapti, E, Ramos, G, Bonilla, C, Sabogal, I, Aranda, I, Dye, C, Raviglione, M and Espinal, MA (2002) "Feasibility and cost-effectiveness of standardised second-line drug treatment for chronic tuberculosis patients: a national cohort study in Peru" *The Lancet* Vol 359 (June 8th) p 1980-9

Summers, Robert and Heston, A (1991) "The Penn World Table (Mark 5): An Expanded Set of International Comparisons, 1950-1988" *Quarterly Journal of Economics* Vol 106 (2), p 327-68

Temple, Jonathan (1999) "The New Growth Evidence" *Journal of Economic Literature* Vol 37, p 112-56

Weil, David (June 2001) *Accounting for the Effect of Health on Economic Growth* processed

Weekly Epidemiological Record (1999) *Malaria: 1982-1997* No 32, p 265-70

World Bank (2002) *World Development Indicators 2002* Washington, DC: World Bank

World Health Organization (1983) *World Health Statistics Annual* p 791-5 Geneva: World Health Organization

World Health Organization (2001) *The World Health Report 2000: Health systems: Improving performance* Geneva: World Health Organization

World Health Organization (2002) *Global Tuberculosis Control: Surveillance, Planning, Financing WHO Report 2002* WHO/CDS/TB/2002.295; Geneva: World Health Organization

World Health Organization (2003) *Global Tuberculosis Control: Surveillance, Planning, Financing WHO Report 2003* WHO/CDS/TB/2003.316; Geneva: World Health Organization

Wyss, Kaspar, Kilima, P and Lorenz, N (2001) "Costs of Tuberculosis for Households and Health Care Providers in Dar es Salaam, Tanzania" *Tropical Medicine and International Health* Vol 6 (1) p 60-8

APPENDICES

Appendix 1 Categorisation of countries

This table contains the 91 countries which are used in the broadest regressions. The eleven high-burden countries not in this sample are Afghanistan, Cambodia, the Democratic Republic of the Congo, Ethiopia, Myanmar, Nigeria, the Russian Federation, Thailand, Tanzania, Uganda, and Vietnam.

	Absent from Sample of 78	Absent from Sample of 68	OECD Member	World Bank Income Class	WHO Health System Class	DALE Quartile	High Burden Country	Ave. Reported TB cases '96-'00
Algeria	•	•		2	3	4		56.0
Australia			•	4	4	4		5.5
Austria			•	4	4	4		15.6
Bangladesh				1	3	2	•	53.6
Barbados		•		3	4	3		1.6
Belgium			•	4	4	4		12.0
Benin				1	2	2		40.6
Bolivia				2	2	2		126.2
Botswana	•	•		3	1	1		529.6
Brazil				3	2	3	•	51.2
Bulgaria	•	•		2	2	3		43.2
Cameroon				1	1	1		34.6
Canada			•	4	4	4		6.5
Central African Republic	•	•		1	1	1		126.5
Chile				3	4	4		24.8
China				2	2	3	•	36.0
Colombia				2	4	3		24.4
Costa Rica				3	4	4		19.6
Cyprus	•	•		4	4	4		4.8
Denmark			•	4	4	3		9.7
Dominican Republic				2	3	4		111.2
Ecuador				2	2	3		62.2
Egypt, Arab Rep.				2	3	4		19.0
El Salvador				2	2	4		27.0
Fiji	•	•		2	2	2		22.2
Finland			•	4	4	4		11.4
France			•	4	4	4		11.3
Germany	•	•	•	4	4	4		12.8
Ghana				1	2	1		58.2
Greece			•	4	4	4		8.5
Guatemala				2	3	2		27.4
Guinea-Bissau		•		1	1	1		111.8
Guyana	•	•		2	2	2		49.4
Haiti	•	•		1	2	2		116.6
Honduras				2	2	4		71.2
Hong Kong, China		•		4	n/a	n/a		102.8
Hungary			•	3	3	2		38.2
Iceland		•	•	4	4	4		4.6
India				1	2	2	•	120.4
Indonesia				1	3	3	•	21.6

	Absent from Sample of 78	Absent from Sample of 68	OECD Member	World Bank Income Class	WHO Health System Class	DALE Quartile	High Burden Country	Ave. Reported TB cases '96-'00
Iran, Islamic Rep.				2	3	3		18.4
Ireland			•	4	4	4		11.2
Israel		•		4	4	4		8.4
Italy			•	4	4	4		7.8
Jamaica				2	3	4		5.0
Japan			•	4	4	4		32.8
Jordan				2	3	2		8.4
Kenya				1	2	1	•	166.0
Korea, Rep.			•	4	3	2		70.2
Lesotho		•		1	1	1		385.2
Malawi				1	1	1		208.4
Malaysia				3	3	3		65.8
Mali				1	1	1		40.0
Mauritius				3	3	2		11.6
Mexico			•	3	3	3		16.4
Mozambique				1	1	1		113.4
Nepal				1	1	2		116.6
Netherlands			•	4	4	4		9.4
New Zealand				4	4	3		10.0
Nicaragua				1	3	3		55.8
Niger				1	1	1		36.8
Norway			•	4	4	4		5.2
Pakistan		•		1	2	3	•	23.3
Panama				3	2	3		48.8
Papua New Guinea	•	•		1	1	1		214.0
Paraguay				2	3	3		37.8
Peru				2	2	2	•	166.8
Philippines				2	3	2	•	220.4
Portugal			•	4	4	4		49.0
Romania				2	2	2		113.0
Rwanda		•		1	1	1		83.2
Senegal				1	3	2		21.6
Sierra Leone	•	•		1	1	1		50.4
Singapore	•	•		4	4	4		23.0
South Africa				2	1	1	•	21.4
Spain			•	4	4	4		36.8
Sri Lanka		•		2	3	3		7.8
Swaziland	•	•		2	1	1		425.4
Sweden			•	4	4	4		5.2
Switzerland			•	4	4	4		10.0
Syrian Arab Republic				2	2	3		34.0
Togo		•		1	1	1		34.0
Trinidad and Tobago				3	3	3		15.8
Tunisia				2	3	4		23.8
Turkey			•	2	3	4		34.4
United Kingdom			•	4	4	4		10.4
United States			•	4	4	3		20.4
Uruguay				3	3	3		6.6
Venezuela, RB				3	3	4		26.6
Zambia				1	1	1		449.7
Zimbabwe				1	1	1	•	374.8

Appendix 2 Sources and manipulations of variables

Raw data from the World Development Indicators (2002):

(W1)	Gross Domestic Product per capita (Constant 1995 US$)	NY.GDP.PCAP.KD
(W2)	Gross Capital Formation (Percent of GDP)	NE.GDI.TOTL.ZS
(W3)	General Government Final Consumption Expenditure (Percent of GDP)	NE.CON.GOVT.ZS
(W4)	Trade (Percent of GDP)	NE.TRD.GNFS.ZS
(W5)	Population Growth (Annual percentage)	SP.POP.GROW
(W5a)	Population	SP.POP.TOTL
(W6)	Total Life Expectancy at Birth	SP.DYN.LE00.IN
(W7)	Infant Mortality Rate (per thousand live births)	SP.DYN.IMRT.IN

Raw data from the Penn World Table (Version 6.1):

(P1)	Real Gross Domestic Product per worker (Constant 1996 US$)	RGDPWOK
(P2)	Investment (Percent of GDP)	KI
(P3)	Government Expenditure (Percent of GDP)	KG
(P4)	Openness (Percent of GDP in constant prices)	OPENK
(P5)	Population	POP

Raw data from other sources:

(BL)	Average Schooling of all those over fifteen years of age	TYR 15
(CS)	Average Schooling of all those over fifteen years of age	TY 15

(TB)	Tuberculosis reported incidence:	1980-2000	World Health Organization (2002)
(Mal)	Malaria reported incidence:	1962-1982	Weekly Epidemiological Report (1999)
		1983-1997	World Health Statistics Annual (1983)

Data series used in this paper:

Series	Description	Source
GDP_{it}	GDP at the end of each period	(W1) or (P1) for the years 1985, 1990, 1995 and 2000
GDP_{it-1}	GDP at the beginning of each period	(W1) or (P1) for the years 1980, 1985, 1990 and 2000
ΔGDP_{it}	Difference in logs of GDP	$\ln(GDP_{it}) - \ln(GDP_{it-1})$
INV_{it}	Investment as a percentage of GDP	(W2) or (P2) averaged across each period (1981-5, 1986-90, 1991-5, 1996-2000)
BL_{it}	Average years of Schooling among adults	(BL) for the years 1980, 1985, 1990 and 2000
CS_{it}	Average years of Schooling among adults	(CS) for the years 1980 and 1990 (CS) average of available figures for the years 1985 and 1995 [80]
$GOVT_{it}$	Government Expenditure as a percentage of GDP	(W3) or (P3) averaged across each period
TRA_{it}	Trade (Imports plus Exports) as a percentage of GDP	(W4) or (P4) averaged across each period
$POPG_{it}$	Average annual Population growth	(W5) averaged across each period or (P6) differenced for each years, averaged across each period
LE_{it}	Life Expectancy at Birth	(W6)
IMR_{it}	Infant Mortality Rate per 1000 live births	(W7)
ASR_{it}	Adult Survival Rate (to age 60) per 1000 individuals reaching the age of 15	Provided by Alok Bhargava as used in Bhargava et al (2001)
MAL_{it}	Reported cases of malaria per 100,000 population	(Mal) divided by the population level (W5a) in each year, multiplied by 100,000, averaged over each period[81]
TB_{it}	Reported cases of tuberculosis per 100,000 population	(TB) averaged over each period

[80] For example, the figure for 1995 is the average of the 1990 and 2000 figures.
[81] In the final period of study this variable is an average of the only two years for which malaria data is available: 1996 and 1997.

Appendix 3 Glossary of acronyms

AIDS	Acquired ImmunoDeficiency Syndrome
ASR	Adult Survival Rate
BCG	Bacillus of Calmette and Guérin (Tuberculosis vaccine)
BL	Barro-Lee Schooling dataset
CMH	Commission on Macroeconomics and Health
CS	Cohen-Soto Schooling dataset
DALY	Disability Adjusted Life Years
DALE	Disability Adjusted Life Expectancy
DOTS	Directly Observed Treatment, Short course (Tuberculosis treatment)
GDP	Gross Domestic Product
HIV	Human Immunodeficiency Virus
HIV-TB	HIV and Tuberculosis co-infection
MDR-TB	Multi-Drug Resistant Tuberculosis
MRW	Mankiw, Romer and Weil (1992)
OECD	Organization for Economic Cooperation and Development
PHC	Primary Health Care
PWT	Penn World Table
TB	Tuberculosis
TFP	Total Factor Productivity
WDI	World Development Indicators
WHO	World Health Organization

A	Technology
K	Capital
L	Labour
Y	Income