Research Paper : Is Growth Good for Health ? 1999

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Is Growth Good For Health?

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1. Introduction

Economic development entails significant additional health hazards. Run-off of fertilizer and side products of industrial production contaminate streams used as source of drinking water, air pollution caused by industrial and household energy production impairs lung capacity, the typically more crowded living arrangements in urban areas ease the transmission of tubercolosis etcetera. At low levels of income per capita, one might thus expect a *Dickens effect*, a worsening of public health as economies transition from agriculture to industry. The effect can be quite dramatic: theWorld Bank (1999) estimates that in four Chines cities alone – Beijing, Chongqing, Shanghai and Shenyang -- , exposure to fine particles in the air will lead to 10,000 prematurely this year, while environmental factors, through diarehea and acute respiratory infections are a major contributor to the twenty percent under-five mortality rates in the poorest countries.

Yet the World Health Organization has called poverty the world's biggest killer: development and urbanization bring with it better provision of and access to health care, better nutrition and better housing conditions, improving public health. In addition, pollution levels for many pollutants appear to decrease after a threshold income level has been reached (captured by the pollution-growth Kuznets curve), be it because of decreased production of pollution intensive products or of increased abatement expenditures. The two offsetting processes potentially bring about a Kuznets curve for public health and income, with a worsening as countries transition from agriculture to early industrialization, followed by improvements as the transition progresses further.

Table 1: Major Environmental Causes of Death

Water contamination with human feces	7% of all deaths globally
Diarrhea	2.5 million deaths/year, mainly children under 5
Acute respiratory infections	4.0 million deaths/year, mainly children under 5

Source: Various, mostly WHO

Table 2: Basic Health Statistics

	GDP p.c.	*Child Mortality*	*Life Expectancy*
Sub-Saharan Africa	510	17.5%	52
India	360	12.7%	58
Middle East	1,720	11.1%	61
Latin America, Caribbean	2,190	6.0%	70
China	370	4.3%	69
Central/Eastern Europe	2,850	2.2%	72
Upper Income Economies	19,900	1.1%	76

Source: World Respources 98-99

While much is known about the links between individual pollutants and the incidence of associated diseases, with most studies focussing on specific sites, less is known about the overall link between development, environmental

pollution and public health. The present paper aims to address this question. Based on a new cross-country time-series dataset combining information about the levels of specific pollutants, the mortiality by age group of diseases associated with these pollutants, provision of health services, as well as general development indicators, we examine, both in cross-section and over time, the linkages between pollutants and mortality levels.

The analysis proceeds in two stages. We first employ decision tree analysis to identify sturdy linkages between pollutants and health effects. While decision tree analysis has been relatively rarely used in economics[2], the methodology is well suited to datasets likely to be characterizerd by threshold effects and context-dependent effects. Both are likely to play a role in the health-pollution nexus. Thus case studies strongly suggest that the link between pollution and health effects is often highly non-linear. For example, air pollution in excess of certain thresholds has been sturdily associated with sudden spikes in mortality.[3] Furthermore, though the evidence on this point is weaker, the effect of exposure to some pollutants appears to depend on the level of exposure to other polliutants. Lacking prior information about the trheshold levels and context dependence, neither effect is particualrly easy to capture in the standard log-linear regression framework. In contrast, classification trees readily capture both effects. Regression analysis can however be used on a more aggregate level. Based on the findings from the classification tree analysis, we hence next turn to a regression analysis of an overall measure of environmentatly related health effects to measures of pollution and the provision of public health services, as well as to exploring the presence of health-development Kuznets curve.

The main resulkts can be summarized briefly. …

We next turn to a description of the dataset, including a listing of the main diseases linked to environmental factors and the associated pollutants. Section three presents the classification tree analysis, section four discusses the results of the regression analysis and section five concludes.

2. Data

The paper is based on data on health, development and the environment collected from a wide variety of sources. The dataset covers more than 100 countries (though for some variables, data availability is much more restrictive). As pollution data are only available at infrequent intervals, data are collected for three five-year periods, covering 1981-85, 1986-90 and 1991-95. For all cases, data for the central years 1983, 1988 and 1993 were taken if available, else the

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[2] The only applications we are aware of are Johnson and Durlauf (19..) and Ghosh and Wolf (19..), both studying economic growth.

[3] This particular non-linearity is quite dramatic, so much so that it can trigger policy responses, such as the measures enacted after a 1953 spike in air pollution leading to some 4000 premature deaths to reduce coal heating in London.

closest datapoint within the five year period was selected. The quality of the underlying data unavoidably differs across sources, as well as across countries for given sources. To reduce sensitivity to extreme measurements, all data were plotted, and outliers removed (typically one or two per variable, with outliers often twenty to thirty times larger than the cluster of observations).

Pollution data are taken from the World Resources Institute database, the World Bank Development Indicators and the OECD Environmental database. Health data are primarily drawn from the World Health Organization Health-for-All database (covering up to 191 countries from 1983 to 1995) and the World Health Organization Mortality database. The health data are, with few exceptions, mortality ratios. In principle, morbidity data would have been more desirable, but availability of morbidity data is very limited. For the purpose of comparative studies across countries, as long as morbidity is proportional to mortality, the problem is of lesser concern. Development data are taken primarily from the World Bank World Development Indicators CD, augmented by some other sources. The appendix contains a complete listing of variables and sources. The selection of pollutants and diseases is based on the existing literature on public health in poorer economies. The following subsection provides capsule summaries of the major diseases that have been linked to the environment, as well as of the main pollutants.

2.1 Diseases

The definition of "environmental diseases" is subjective, as environmental factors are likely to exert at least some influence on morbidity and mortality rates of most diseases. Our classification is restricted to diseases for which environmental factors have been identified as a major cause. Environmental factors in turn divide into two broad groups, which might be labeled direct and incidental. The former group comprises pollutants released as side-effects of production, home heating, transportation etc. The set of diseases being attributable to specific pollutants includes acute respiratory infections, upper and lower respiratory infections (including bronchitis), trachea- bronchial and lung cancer, cardiovascular diseases, and chronic obstructive lung and respiratory diseases, including asthma. The second comprises lack of clean water and sanitation, crowded living conditions etc arising from urbanization, increasing the incidence of naturally occurring diseases, notably tuberculosis, diarrheal diseases, amebic dysentery, typhus, hepatitis B, trachoma, intestinal nematode infections and other infectious and parasitic diseases, including schistosomiasis. The following paragraphs provides very condensed capsule summaries for the major diseases of causes, effects and countermeasures.

- *Tuberculosis* (Communicable, global) Results in reduced lung capacity, general weakness. Individuals living in highly crowded conditions for prolonged periods are at highest risk, improvements in housing and nutrition are associated with reduced incidence of pulmonary tuberculosis.
- *Bacterial food/water contamination* (Communicable, global though more prevalent in warmer climates). Includes dysentery, cholera, and typhoid. Individuals with weak immune system, including children, are at highest risk. Fecal contamination of drinking water supplies, and unsound hygienic practices are major contributors. Reduced incidence associated with improved sanitation, access to clean water and changes in hygiene practices.
- *Hepatitis B.* (Communicable, global). Infection occurs via contact with various bodily fluids, including breast milk and blood. Use of safe water for irrigation and cooking, proper food preparation and sewage disposal are associated with reduced incidence.
- *Trachoma.* (Communicable, global but more prevalent in warmer climates) In severe cases leading to blindness, trachoma infestation has a worldwide incidence, with individuals living in crowded areas at greatest risk. Improvements in housing conditions, access to safe water, and improved nutrition are associated with reduced incidence of trachoma.
- *Intestinal nematode infections* (Communicable, global) Effects include loss of nutritional intake efficiency, and diarrhea. Morbidity is generally low, except in children. Individuals with already compromised immune systems, or

suffering from poor nutrition, are particularly susceptible. Transmission occurs via ingestion of food or plant and through cracked skin, particular through the feet . Improvement of sanitation has first order effects on prevalence.

- *Schistosomiasis* (Communicable, global but higher incidence in warm climates) One of the most prevalent parasitic infection, spread by skin exposure to infected water and contaminated drinking water. Mortality is low, but infection results in chronic debilitation. Improved housing conditions, access to clean drinking and cooking water, and improved sanitation have been linked to reduced incidence.
- *Acute Respiratory Infections* (Global) Includes lower (bronchitis) and upper (rhinitis) respiratory infections. Low mortality but frequently result in significant debilitation. While ARI has been traced to air quality, no consensus about the relative importance of different pathogens has been established. Tentative findings also suggests a positive correlation of incidence with nutritional deficiencies.
- *Malignant Neoplasms* (Global) Evidence points to link with environmental carcinogens. The strongest evidence links trachea, bronchial and lung cancers to airborne pollutants, notably from coals and other fossil fuels used for residential heating, as well as vehicular and industrial use.
- *Cardiovascular Diseases* (Global) Tentatively linked to exposure to both airborne (carbon-monoxide, sulfur dioxide and suspended particulate) and water born contaminants in addition to diet and sendentiary lifestyle. General reduction in physical capacity.
- *Chronic Respiratory Diseases* (Global). Both direct causal effects of air-born pollutants, mainly indoor air pollution caused by usage of traditional fuel for heating/cooking without adequate ventilation and aggrevation of existing conditions, notable asthma. General reduction in physical capacity.

2.2 Pollutants

As the effects of pollutants on health often arises in a non-linear fashion with substantial lags, the identification of specific contributors to specific diseases is diffiuclt. The following provides a brief listing of the properties and health effects for a set of pollutants for which the pre-ponderance of evidence suggests a significant link. More detailed discussions can be found in Elsom (1992), Hermamala et. al. (1995), Hettige et. al (1995), Moeller(1992), Wegman (1996) and the references cited therein.

- *Total Suspended Particles (TP) and Fine Particles (FP)* Fine liquid or solid particles such as dust, smoke mist, fumes or smog found in air emissions. Constituents vary over time and space, with a higher concentration of carbons and hydrocarbons in urban areas. Major man-made sources of TP/FP include industrial processes, combustion, transportation and forest burning forest burning. High levels of total, and in particular of fine particles are associated with greater morbidity and mortality from respiratory diseases. Spikes in TP are frequently associated with spikes in mortality notable in children and the elderly (London 1952, SE Asia 1997). TP/FP have also been implicated in heart disease.
- *Sulphur Dioxide* Resulting primarily from fossil fuel combustion, linked to morbidity and mortality from respiratory disease.
- *Nitrogen Oxides* A bi-product of fossil fuel combustion, may originate from gas heaters, boilers, cooking stoves. Inhalation of concentrated NO2 damages the respiratory tract, with effects ranging from mild reductions in pulmonary function to life threatening pulmonary edema.
- *Carbon Monoxide* Resulting from incomplete fossil fuel combustion, mostly from transportation (outdoors) and heating (indoors). Exposure to small concentrations reduces the blood's oxygen carrying capacity and may cause weakness, fatigue headaches and nausea. High doses can result in heart and brain damage, impaired perception and asphyxiation. Chronic exposure is associated with cardiovascular disease.

_ Volatile Organic Compounds (VOC)

_ VOC comprises a range of substances used mainly as solvents and fragrances, arising from combustion, transportation and industrial production. Exposure due to inhalation and to some extent contamination of drinking water, food, beverages has been linked to eye and lung irritation and inflammation as well as irritation in the mucous membranes.

_ *Water Pollutants*

_ Suspended Solids

_ Small particles of non-organic, non-toxic solids settling in sludge blankets can lead to a loss of micro-organisms prolonging lifespan of pathogens.

_ Toxic Chemicals

_ A number of chemicals in industrial emissions have been found to be poisonous to humans, either on immediate exposure or cumulatively. Adverse effects include damage to internal organs, neurological functions, reproductive problems, birth defects and cancer.

_ Coliform
_ Bacterial contamination arising from disposal of raw human sewage into water supply. Primary cause of diarrheal diseases.

_ *Soil Pollutants*

_ Bioaccumulative Metals

Bioaccumulated metals dissipate very slowly. Adverse effects, in particular on mental health, kidney damage and birth defects, have been argued to be present for mercury, lead, arsenic, chromium, nickel, copper, zinc and cadmium. Children are most susceptible. Main man-made sources are transportation, fuel combustion, lead smelting and refining, storage-battery

manufacturing.

Pesticides

Synthetic pesticides, while controlling pests and reducing some channels of disease transmission, have also been associated with cancer, neurologic, reproductive, immunologic disorders, in particular in the case of prolonged high dosage exposure.

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and and health measauresAggregation to an extent reduces these problems. The effects are however less likely

We then turn toFollowing up on this more detailed examinatiuo

of these spikes resulted in measures to eThus cateregression measures of the cross section from d levels in a large set of countries damage and public a feature well documented in Dicken's beginning leve Pollution per unit of production however tends to shrink as income per capita furthermore, the provision of public health services increase The These and other factefluents released by factories inoften entails both significant advances in the availability of public health care and significant additional health hazards, with opposite effects on public health. A large case-based literature has examined the effect of individual pollutants these lin, public health an dthe state of the environment are complexly inter-linked. In recent decades, components of this system have been studied in substantial depth both on the aggregate level, notably the issue of ozone depletion and its possible effect on global temperatures, and on the local level, primarily case studies examining individual pollutants and their health effects, often for individual locales. On the aggregate level, research to date has concentrated on the causal link between growth and the environment. Less light has been shed on the reverse linkage from environment to health to labor productivity and growth.

The most robust finding emerging to date from this research program is an apparent inverted U-curve link between GDP per capita and some, though not all pollutants.[4] The interpretation of this link remains the subject of research. Does the decline of pollution at higher income levels simply reflect the sectoral shift from high-pollution manufacturing to low-pollution services during the process of development? Is demand for environmental quality a superior good, motivating increasing abatement expenditures as countries grow, and thus decreasing pollution emissions per unit of manufacturing output? Does the linkage reflect the migration of polluting industries to poorer countries acting as ``pollution havens" for outsourced pollution-intensive production of richer countries? Or is indeed the causality chain reversed, with a better state of the environment enabling additional economic growth?

The largely unanswered empirical questions of the precise tradeoffs between environment
and economic growth once the two-sided causality is explicitly considered impede policy advice. If the direction of causality runs primarily from growth to the environment, the allocation of limited resources to pollution abatement at different levels of development will primarily reflect a tradeoff between public demand for a better environment relative to other objectives. Most prominent among the latter has been investment driven income growth. In this debate, it is not infrequently argued that at low income levels, the marginal benefit of additional income exceeds the marginal benefit of reduced pollution, or the marginal cost of increased pollution, leading to a postponement of anti-pollution spending until higher income levels have been reached.

This distinction --- better environment {\it or} faster growth --- however vanishes if causality is bi-directional, with greater pollution exerting a significant adverse effect on public health and consequently labor productivity and growth. In this case, anti-pollution expenditures may be optimal at low income levels even if the main policy objective is income growth. Ex ante, there is good reason to believe that pollution and inadequate supply of clean water and sanitation has significant health and output costs. A 1997 report on Asia [World Bank (1997)]

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[4] See Selden and Song (1994), ... Among the exceptions showing little evidence of a decline at higher income levels are carbon dioxide.

attributes 500.000 infant deaths a year in the Asia/Pacific region to dirty water and poor sanitation , while 50.000 premature deaths and 400.000 new cases of chronic bronchitis, with a cost estimated at twenty percent of urban income, are attributed to smoke and small particles in a sample of 11 large Chinese cities.

In this paper, we employ a comprehensive dataset covering indicators of development, health and the environment to shed some light on these issues. Section 2 contains a brief review of the major environmental health effects, and the implicated pollutants. Section 3 presents a set of stylized facts concerning ``typical'' levels of environmental degradation at various income levels, the incidence of the major environmental diseases at various income levels, as well as the unconditional relations between environmental degradation and environmental diseases and between diseases and growth.

Section 4 presents a stylized model of the relationship between health, pollution and growth providing the framework for the subsequent empirical analysis.

Sections 5 to 7 provide more detailed information about the individual linkages between growth and pollution (5), pollution and public health (6), and health and growth (7). Drawing on these results, section 8 explores the growth-health-pollution nexus in a simultaneous system framework.

The report is focused primarily on the local short to medium run connections between pollution, health and growth. We do not touch more than peripherally on either global or longer term impacts, specifically, we do not cover the global warming implications of pollution and deforestation, or the loss of biodiversity as a function of growth, deforestation and desertification. The restriction in focus is motivated by our desire to evaluate {\it comparative} environmental quality, and its immediate health effects, in countries on similar development levels. A full assessment of the growth-environment nexus will, however, also have to incorporate the costs of these longer term and global effects.

\section{Environmental Diseases and Pollutants: A Brief Overview}

The definition of ``environmental diseases'' is in the end subjective, as environmental factors are likely to exert at least some influence on susceptibility to most diseases, as well as on the severity of cases. Our classification is restricted to

diseases for which environmental factors have been identified as a major cause. Environmental factors in turn divide into two broad groups, which might be labeled direct and incidental. The former group comprises pollutants released as side-effects of production, home heating, transportation etc. The second comprises lack of clean water and sanitation, crowded living conditions etc, increasing the incidence of naturally occurring diseases, including bacterial (diarrheal diseases), viral (hepatitis and others) and parasitic (nematodes) illnesses.

\subsection{Diseases}

The set of diseases being attributable to specific pollutants includes acute respiratory infections, upper and lower respiratory infections (including bronchitis), trachea- bronchial and lung cancer, cardiovascular diseases, and chronic obstructive lung and respiratory diseases, including asthma.

Lack of safe water, crowding and lack of sanitation has been associated with tuberculosis, diarrheal diseases (including dysentery and cholera), amebic dysentery, typhus, hepatitis B, trachoma, intestinal nematode infections (including ascariasis, hookworm and trichuriasis), and other infectious and parasitic diseases, including schistosomiasis,

\begin{itemize}

\item {\bf Communicable Diseases}

\begin{itemize}

\item {\bf Tuberculosis} is a conglomerate of diseases resulting from several different bacteria, with a worldwide incidence. Individuals living in highly crowded conditions for prolonged periods are at highest risk. Improvements in housing and nutrition are associated with reduced incidence of pulmonary tuberculosis.

\item {\bf Responses to bacterial food/water contamination} include dysentery, cholera, and typhoid. Individuals with weak immune system, including children, are at highest risk. Fecal contamination of drinking water supplies, and unsound hygienic practices are major contributors. Improved sanitation, access to clean water and changes in hygiene practices are associated with reduced incidence.

\item {\bf Hepatitis B} infection occurs via contact with various bodily fluids, including breast milk and contact with infected

blood. Use of safe water for irrigation and cooking, proper food preparation and sewage disposal are associated with reduced incidence.

\item {\bf Trachoma}, in severe cases leading to blindness, has a worldwide incidence, with individuals living in crowded areas at greatest risk. Improvements in housing conditions, access to safe water, and improved nutrition are associated with reduced incidence of trachoma.

\item {\bf Intestinal nematode infections}, including ascariasis, hookworm, trichuriasis have worldwide incidence. Effects include loss of nutritional intake efficiency, and diarrhea. Morbidity is generally low, except in children. Individuals with already compromised immune systems, or suffering from poor nutrition, are particularly susceptible. Transmission occurs via ingestion of food or plant (Ascariasis, Trichuriasis) and through cracked skin, particular feet (hookworm). Improvement of sanitation has first order effects on prevalence.

\item {\bf Schistosomiasis} is one of the most prevalent parasitic infection, spread by skin exposure to infected water and contaminated drinking water. Geographic distribution is worldwide, though lower in moderate climates. Morbidity is low, but infection results in chronic debilitation. Improved housing conditions, access to clean drinking and cooking water, and improved sanitation have been linked to reduced incidence of infections.

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\item {\bf Non-Communicable Diseases}

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\item {\bf Acute Respiratory Infections}, including lower (bronchitis) and upper (rhinitis) respiratory infections, have fairly low mortality rates, but frequently result in significant debilitation. While ARI has been traced to air quality, no consensus about the relative importance of different pathogens has been established. Tentative findings also suggests a positive correlation of incidence with nutritional deficiencies.

\item {\bf Malignant Neoplasms} have been traced to environmental carcinogens. Specific, though tentative, findings include carcinogenic effects on the liver, bladder, skin, larynx, breast, typhoid, stomach, colon, ovary and nose. The strongest evidence links trachea, bronchial and lung cancers to airborne

pollutants, notably from coals and other fossil fuels used for residential heating, as well as vehicular and industrial use.

\item {\bf Cardiovascular} diseases have been tentatively linked to exposure to both airborne (carbon-monoxide, sulfur dioxide and suspended particulates) and water born contaminants.

\item {\bf Respiratory Diseases}. Some respiratory diseases, notably chronic obstructive lung disease have been directly linked to pollutants, mainly indoor air pollution caused by usage of traditional fuel for heating/cooking without adequate ventilation. Others, including asthma, do not trace to environmental conditions, but are aggravated by pollutants.

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\subsection{Pollutants}

This section provides a summary listing of the properties and health effects for a set of pollutants held accountable for some of the above diseases. More detailed discussions can be found in Elsom (1992), Hermamala et. al. (1995), Hettige et. al (1995), Moeller(1992), Wegman (1996) and the references cited therein.

\begin{itemize}

\item {\bf Air Pollutants}

\begin{itemize}

\item {\it Total Suspended Particles (TP) and Fine Particles (FP)}
Fine liquid or solid particles
such as dust, smoke mist, fumes or smog found in air emissions. FP are less than ten micron in diameter. The constituents vary over time and space, with a higher concentration of carbons and hydrocarbons in urban areas. Up to 20 percent of TP consist of sulfuric acids and other sulfur compounds. Major man-made sources of TP/FP include industrial processes (mainly outdoor), combustion (indoor and outdoor) and transportation (outdoor).

High levels of total, and in particular of fine particles are associated with greater morbidity and mortality from respiratory diseases. Spikes in TP are frequently associated with spikes in mortality notable in children and the elderly (London 1952, SE Asia

1997). TP/FP have also been implicated in heart disease.

\item {\bf Sulphur Dioxide (SO_2)}
A heavy, unguent, colorless, gas formed primarily by fossil fuel combustion. Sulphur dioxide has been linked to morbidity and mortality from respiratory disease, as well as being the primary source of acid rain and thus a threat to bio-diversity.

\item {\bf Nitrogen Oxides (NO_x)}
Thermal bi-product of fossil fuel combustion. Extremely high concentrations of nitrogen dioxide may originate from gas heaters, boilers, cooking stoves. Inhalation of concentrated NO2 damages the respiratory tract, resulting in mild reductions in pulmonary function to life threatening pulmonary edema.

\item {\bf Carbon Monoxide (CO)}
A colorless, odorless, and tasteless poisonous gas produced by incomplete fossil fuel combustion, mostly from transportation (outdoors) and heating (indoors). Exposure to small concentrations reduces the blood's oxygen carrying capacity and may cause weakness, fatigue headaches and nausea. High doses can result in heart and brain damage, impaired perception and asphyxiation. Chronic exposure is associated with cardiovascular disease.

\item {\bf Volatile Organic Compounds (VOC)}
VOC comprises a range of substances used mainly as solvents and fragrances, arising from combustion, transportation and industrial production. Exposure due to inhalation and to some extent contamination of drinking water, food, beverages has been linked to eye and lung irritation and inflammation as well as irritation in the mucous membranes. Mostly associated with an increase in morbidity, not mortality.

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\item {\bf Water Pollutants}

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\item {\bf Suspended Solids}

Small particles of non-organic, non-toxic solids settling in sludge blankets can lead to a loss of micro-organisms prolonging lifespan of pathogens.

\item {\bf Toxic Chemicals}

A number of chemicals in industrial emissions are poisonous to humans, either on immediate exposure or cumulatively. Adverse effects include damage to internal organs, neurological functions, reproductive problems, birth defects and cancer.

\end{itemize}

\item {\bf Soil Pollutants}

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\item {\bf Bioaccumulative Metals}

Bioaccumulated metals dissipate very slowly. Adverse effects, in particular on mental health, kidney damage and birth defects, have been argued to be present for mercury, lead, arsenic, chromium, nickel, copper, zinc and cadmium. Children are most susceptible. Main man-made sources are transportation, fuel combustion, lead smelting and refining, storage-battery manufacturing.

\item {\bf Pesticides}

Synthetic pesticides, while controlling pests and reducing some channels of disease transmission, have also been associated with cancer, neurologic, reproductive, immunologic disorders, in particular in the case of prolonged high dosage exposure.

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\section{Data and Stylized Facts}

We begin with a brief characterization of the data underlying the study. We then report basic statistics on the comparative level of various health and pollution indicators across countries on different income levels.

\subsection{Data}

The data underlying this paper have been collated from a wide variety of sources. Unavoidably, the quality of the underlying data differs substantially across sources, as well as across countries for given sources.

The measurement of pollutants in particular suffers from a host of problems. First, for some pollutants, no common standards for measurement, either in terms of unit or in terms of measurement technique, have yet emerged. Rarely are different techniques employed at the same time in the same location, hence it is generally not possible to make an assessment of the quality, or likely differences between, alternative measurement techniques for the same variable. Original sources at times differentiate between measurement techniques, but predominantly collate across techniques. In constructing the present dataset, we selected the most widely available measurement technique as a baseline, and complemented with data obtained by other techniques. To reduce sensitivity to extreme measurements, all data were plotted, and outliers removed (typically one or two per variable, with outliers often twenty to thirty times larger than the cluster of observations).

In terms of ex ante ``subjective quality", we would place least confidence in the fecal coliform series, as observations are crucially dependent on a host of factors, including rainfall in the period preceding the measurement and water temperature at the time of measurement. In consequence, subsequent observations for the same river often display significant jumps with no clear trend. Air pollutants in contrast are fairly stable over time and across similar countries, we would hence place higher subjective confidence in the series for air pollutants. Pollution data are taken from the World Resources Institute database (up to 37 countries, 1981-95), the World Bank Development Indicators, the OECD Environmental database (up to 22 countries, 1983 to 1995).

Health data are primarily drawn from the World Health Organization Health-for-All database (covering up to 191 countries from 1983 to 1995) and the World Health Organization Mortality database.\footnote{We are grateful to Dr. Odell Frank for making these data available.} The health data are, with few exceptions, mortality. In principle, morbidity data would have been more desirable, but availability is very limited. For the purpose of comparative studies, as long as morbidity is proportional to mortality, the problem is of lesser concern.

A range of development data were drawn from the World Bank Development Indicators (up to 211 countries, 1981-1996). Political freedom and civil liberties indices were taken from the

Heritage Foundation.

As pollution data are available at highly infrequent intervals, data are collected for three five-year periods, covering 1981-85, 1986-90 and 1991-95. For all cases, data for the central years 1983, 1988 and 1993 were taken if available, else the closest datapoint in the five year period was selected.

\subsection{Distribution}

Tables 3 to 10 report distribution statistics for the health and pollution variables, for all countries, as well as for countries with income per capita below 4000 US\$, between 4000 and 8000 US\$ and above 8000 US\$. For the full sample, the 1st,5th,10th,25th,50th,75th,90th,95th and 99th percentile is reported, for the subsamples, the 10th, 25th, 50th, 75th and 90th percentile is reported. If less than 15 observations were available, only the median is reported.

Tables 1 and 2 provide a summary view, reporting the medians for the full sample and the three subsamples. Not too surprising, access to safe water and sanitation improves strongly in income per capita. Among the pollution variables, various patterns are observable, from straight positive income elasticities (waste, fertilizer and pesticide use) to straight negative income elasticities (fecal coliform, SO2, suspended particulate matter) to inverted U-shape relationships (CO2, Methane).

On the health side, most diseases associated with access to safe water and sanitation decline in income as access improves. Death is of course unavoidable, the lower mortality rates from intestinal diseases in richer economies are matched by higher moralities from Neoplasms, pulmonary and some respiratory diseases.

Tables 3 to 10 present the data in substantially more detail. The grouping by income level permits a ready assessment of a countries pollution and health statistics relative to the peer group.

\section{A Simple Model}

Growth and the environment are inextricably intertwined through a range a channels. To provide a framework for the empirical work we next turn to a stylized endowment model incorporating the major links.

Pollution, P, measures the extent of man-made pollution, so

that an increase in P implies a worsening of the environmental quality. At any point in time, the extent of pollution depends upon two factors. First, the cumulative production of goods, $C(Y)=\int_{t0}^{t}Y(s)e^{\delta s}ds$ where $Y(t)$ denotes production at time t and δ denotes the natural decay of pollutants. Second, cumulative pollution reduction expenditures $C(E)=\int_{t0}^{t}E(s)e^{\gamma s}ds$, where $E(t)$ measures pollution reduction expenditures at time t, and $ \gamma $ measures the decaying efficiency of past expenditures.\footnote{Within the endowment model presented here, E is narrowly defined as actual expenditure. More generally, it may be thought of to include indirect expenditures such as increases in the cost of capital or consumption goods made necessary by environmental regulation, and foregone output due to environmental regulations and standards.} Overall, the state of the environment is thus given by:

\be P(t) = f(\int_{t0}^{t}Y(s)e^{\delta s}ds,
\int_{t0}^{t}E(s)e^{\gamma s}ds) \ee

Average health, H, is assumed to depend negatively on current and lagged pollution, allowing for both instantaneous and delayed adverse effects:

\be H(t)=g(L(P(t))) \ee

with $g'<0$. L denotes a lag operator. The economy obtains an endowment of $Z(t)$ ``inactive" units per time period, where $Z$ grows at a deterministic rate. In order to activate (``harvest") the units, labor input is required. Labor productivity, $LP(t)$ is a positive function of health:

\be L(t)=h(H(t)) \ee

with $h'>0$ and $0 \leq L(t) \leq 1$. Production Y is given by:

\be Y(t) = L(t)Z(t) \ee

Thus $0 \leq Y(t) \leq Z(t) $. Production in turn can be allocated to two uses, consumption C and pollution control expenditures E, with no storage:

\be Y(t) = C(t) + E(t) \ee

or, substituting:

\be h(g(L(f(\int_{t0}^{t}Y(s)e^{\delta s}ds,
\int_{t0}^{t}E(s)e^{\gamma s}ds)))) Z(t) = C(t) + E(t) \ee

The fairly simple setup yields rather rich dynamics. Current production depends on current health, which in turn is related to prior production and pollution control decisions. Allocation decisions are intrinsically dynamic. In particular, to the degree that pollution effects on health occur with lags rather than instantaneously, additional current pollution control expenditures reduce current consumption but increase future growth.

The model is closed by assuming that preferences encompass both material well-being, proxied by consumption, and health:

\be U(t) = u(C(t),H(t)) \ee

where health is broadly defined to include psychological benefits from a clean environment.\footnote{Cropper and Oates (19??) provide a survey of valuations placed on both direct health effects of pollution, and on enjoyment values.} The representative agent maximizes discounted utility:

\be Max U = \int_{t}^{t-max} U(t)e^{-\beta s}ds \ee

The simplified model assumes away many real world complications, in return for yielding a well defined optimal time path of pollution abatement expenditures reflecting both the ``enjoyment value'' of the environment, the health consequences of pollution, and the growth consequences of deteriorating health.

\section{Economic Growth And The Environment}

A large and growing literature examines the linkage between economic growth and the environment. While a subset of the theoretical literature allows for two-sided causality, the empirical work to date has predominantly focused on a causal link running from economic development to the state of the environment and the prevalence of pollution.

The key stylized fact emerging from the existing literature is the presence of an inverted U-shaped relation between income per capita and various measures of pollution. Figures xx to xx plot the pollution series described above against income per capita. Table \ref{quadrad} reports the results of regressions of log pollution levels on log income and the square of log income.

\begin{table}[htbp]
\begin{center}
\caption{\bf The Pollution-Income Link I}

```
\vspace{0.1cm}
\begin{tabular}{||l||r|r|r|r|r||}
\hline
        &  &    &    &  &   \\
Log(Pollutant)  &Cons-&Log Of  &Log of  &Obs.&$R^{2}$\\
        &tant &Income  &Income  &  &   \\
        &  &per cap.&per cap.&  &   \\
        &  &    &Squared &  &   \\
        &  &    &    &  &   \\
\hline
        &  &    &    &  &   \\
Particulates  &  &    &    &  &   \\
        &  &    &    &  &   \\
Sulfor Oxides  &  &    &    &  &   \\
        &  &    &    &  &   \\
$NO_x$      &  &    &    &  &   \\
        &  &    &    &  &   \\
Carbon Monoxide  &  &    &    &  &   \\
        &  &    &    &  &   \\
Carbon Dioxide  &  &    &    &  &   \\
        &  &    &    &  &   \\
Volatile Compounds&  &    &    &  &   \\
        &  &    &    &  &   \\
Fecal Coliform  &  &    &    &  &   \\
        &  &    &    &  &   \\
\hline
\end{tabular}
\end{center}
\end{table}
```

The interpretation of this correlation is far from obvious. The total new emission of a particular pollutant P in country q can be decomposed by sector:

```
\be P^q = \sum_{r=1}^{n} Y^{q}_{r}I^{q}_{r} \ee
```

where Y_{r} is the value of output in sector r and I^{q}_{r} denotes the emission of the pollutant per value unit in sector r. As income increases, emissions can decline because