INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.
The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author’s permission.

L’auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

L’auteur conserve la propriété du droit d’auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.
Abstract


Andrew Thomas Pricesmith. Department of Political Science, University of Toronto.

This thesis investigates the influence of infectious disease upon state capacity, stability and prosperity. It argues that the increasing global proliferation of infectious human pathogens will have a significant negative effect upon the ability of the state to govern itself effectively, and to maximize its economic power. First, this thesis defines the scope of the problem of emerging and re-emerging human pathogens and explores the pathways of disease emergence. Second, it presents a methodology that includes quantifiable measures of State Capacity, and proxy measures for the global burden of disease. Third, it presents the findings of the correlations run on the independent and dependent variables, and concludes that there is in fact a strong and significant negative association between infectious disease prevalence and state capacity. Fourth, this thesis argues that infectious disease will contribute to increasing poverty, and have a significant negative impact on the formation and consolidation of human capital. Given that infectious disease may generate increasing poverty and deprivation of the population, while simultaneously reducing the state’s ability to govern effectively, this may lead to increasing levels of intra-state violence and political instability.
Acknowledgements

This study owes a profound debt to the many teachers, colleagues, and friends that provided valued support, comments and inspiration to me during the writing of this dissertation. My thesis advisors David Welch and Janice Gross Stein, provided me with the freedom to pursue an intellectual journey into uncharted waters, while providing a reliable compass to steer a clear course. Their critical insights, support, and encouragement over the years were invaluable over my years at the University of Toronto, and this work is that much stronger for their precision and prodding.

I would also like to thank Robert O. Matthews, whose presence on the advisory team encouraged me to think beyond the limits of mainstream approaches and try to integrate findings from the ‘international relations’ and ‘development’ sub-fields into a greater whole. Additionally, I would like to thank the two departmental readers, Louis Pauly and Ronald Deibert, and my external examiner Mark Zacher, who all gave insightful comments and critiques. I am fortunate to have had such a strong cadre examine my work at this nascent phase of my career. I would also like to thank Dr’s Pauly, Stein, and Welch for investing in the unusual idea of creating the Program on Health and Global Affairs in the Centre for International Studies at the University of Toronto. This gave me the intellectual space, incentive, and funding to pursue this novel stream of research.

Many others deserve considerable credit for their help along the way in the form of thoughtful discussions, insights, and comments on my work. I am particularly indebted to Stephen S. Morse of the Columbia School of Public Health, Marc Levy of CIESIN/Columbia University, Mark Zacher at the University of British Columbia, Col. Patrick Kelley of the USDOD, and Thomas Homer-Dixon at the University of Toronto for his early encouragement during the project. I also owe a considerable debt to my colleague and friend Peter Zoutis for his guidance and insight. Others who deserve considerable credit for their insight and support include; Catherine Chalin, David Charters, Martha Cottam, Geoffrey Dabelko, Marion Danis, Ronald Deibert, Dan Deudney, David Fidler, Laurie Garrett, Arthur Gilbert, Nils Petter-Gleditsch, Jack Goldstone, Franklyn Griffiths, Barry Hughes, Jay Keystone, Ann-Marie Kimball, John Kiyaga-Nsubuga, John Last, Dennis Pirages, Wesley Wark, Jim Whitman, and David Zakus. I would also like to thank my former advisor, Salim Mansur, for guiding me through the MA thesis at the University of Western Ontario, and Catherine Conaghan and Colin Leys of Queen’s University for inspiring me to pursue a career in political science in the first place. Additionally, I would like to thank all the staff at the Centre for International Studies and the Department of Political Science and Michelle Rizzoli in Peace and Conflict Studies.

Generous financial support during my doctoral studies was provided by the Environmental Change and Security Project of the Woodrow Wilson Center, a doctoral research fellowship from the Canadian Institute for Advanced Research, the Alexander Brady-MacGregor Dawson Doctoral Fellowship in political science and an Open Fellowship from the University of Toronto, the Sir Val Duncan travel award, a doctoral fellowship and office space from the Centre for International Studies, a University of
Toronto Associates Travel Grant, an award from the Connaught Foundation for a conference on the issue of Health Security, and finally a doctoral research fellowship at the Boston University School of Public Health.

I want to express my deep gratitude to my parents Cynthia Smith-McLeod and Jack McLeod for their unflagging support through all my years of graduate studies. I particularly want to thank my wife and dearest friend Lisa Fairclough, who endured the attendant academic madness that all doctoral candidates put their partners through. I would also like to thank my trusted friends Shaun Curtis, Peter Gizewski, Allen Chong, Don Operario, and Peter Zoutis. A debt of gratitude is owed to the physicians who repeatedly patched me up such that I could actually write this, thanks to Dr. Gordon Greenberg in particular.

I am also grateful to my grandparents Marjorie and Harry Price-Smith (dec.) for their generosity. Finally, I wish to thank my dear grandparents Margaret (Arla) McKay Thomas (dec.) and Douglas Cameron Thomas, for their unflagging support and guidance over an occasionally turbulent youth. Their kindness, generosity, wisdom and commitment has always been the greatest source of inspiration to me.

I dedicate this thesis to them.
# Table of Contents

Abstract ii

Acknowledgements iii

List of Tables and Figures viii

Chapter One: Introduction 1

Scope of the Problem 3

Trends in Disease Emergence 7

The State of Knowledge on Health Security 14

*Historical Overview* 16

Redefining Security 19

Effects of Infectious Disease on the State/System 30

*Disease and Economic Productivity* 30

*Disease and Governance* 31

*Possible Systems Level Effects* 32

Thesis Design 37

Chapter Two: Methodological Considerations 40

The Dependent Variable 46

The Independent Variable 52

Theories of Causation 64

Intervening Variables 67

*Ecological Disruption* 68

*Migration* 68
List of Tables and Figures

<table>
<thead>
<tr>
<th>Table/Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 2.1</td>
<td>Probabilistic Relations between Variables</td>
<td>43</td>
</tr>
<tr>
<td>Table 2.1</td>
<td>Indicators of State Capacity</td>
<td>47</td>
</tr>
<tr>
<td>Table 2.2</td>
<td>Pathogenic Agents</td>
<td>53</td>
</tr>
<tr>
<td>Table 2.3</td>
<td>Disease Data Availability, Per Pathogen, Per Country</td>
<td>56</td>
</tr>
<tr>
<td>Table 3.1</td>
<td>Infant Mortality vs. Gross National Product</td>
<td>84</td>
</tr>
<tr>
<td>Table 3.2</td>
<td>Infant Mortality vs. Government Expenditure</td>
<td>86</td>
</tr>
<tr>
<td>Table 3.3</td>
<td>Infant Mortality vs. Military Expenditure</td>
<td>87</td>
</tr>
<tr>
<td>Table 3.4</td>
<td>Infant Mortality vs. Secondary School Enrollment</td>
<td>89</td>
</tr>
<tr>
<td>Table 3.5</td>
<td>Infant Mortality vs. Net Long-Term Capital Inflow</td>
<td>90</td>
</tr>
<tr>
<td>Table 3.6</td>
<td>Life Expectancy vs. Gross National Product</td>
<td>92</td>
</tr>
<tr>
<td>Table 3.7</td>
<td>Life Expectancy vs. Government Expenditure</td>
<td>93</td>
</tr>
<tr>
<td>Table 3.8</td>
<td>Life Expectancy vs. Military Expenditure</td>
<td>94</td>
</tr>
<tr>
<td>Table 3.9</td>
<td>Life Expectancy vs. Secondary School Enrollment</td>
<td>96</td>
</tr>
<tr>
<td>Table 3.10</td>
<td>Life Expectancy vs. Net Long-Term Capital Inflow</td>
<td>97</td>
</tr>
<tr>
<td>Table 3.11</td>
<td>ERID Proxies vs. National State Capacity</td>
<td>99</td>
</tr>
<tr>
<td>Table 3.12</td>
<td>ERID Proxies vs. SC Indicators: Americas</td>
<td>101</td>
</tr>
<tr>
<td>Table 3.13</td>
<td>ERID Proxies vs. SC Indicators: Europe</td>
<td>102</td>
</tr>
<tr>
<td>Table 3.14</td>
<td>ERID Proxies vs. SC Indicators: Asia</td>
<td>102</td>
</tr>
<tr>
<td>Table 3.15</td>
<td>ERID Proxies vs. SC Indicators: Africa</td>
<td>103</td>
</tr>
</tbody>
</table>
Table 3.16: Global IM vs. Global SC p. 107
Table 3.17: Global LX vs. Global SC p. 108
Table 3.18: Global SC vs. Global IM p. 109
Table 3.19: Global SC vs. Global LX p. 109
Table 3.20: Tuberculosis vs. SC Indicators (U.S.) p. 112
Table 3.21: U.S. State Capacity vs. Tuberculosis Incidence/Prevalence p. 114
Chapter One

Introduction

Ingenuity, knowledge, and organization alter but cannot cancel humanity's vulnerability to invasion by parasitic forms of life. Infectious disease which antedated the emergence of humankind will last as long as humanity itself, and will surely remain, as it has been hitherto, one of the fundamental parameters and determinants of human history.


In the post-Cold War era policy-making communities are increasingly confronted with significant new challenges to the security and prosperity of the citizens over which they preside. Policy-makers must now address diffuse threats to state interests, particularly renewable resource scarcities, environmental degradation, and international migration. Indeed the rise of ‘low politics’ to the national security agenda of the modern state requires that international relations theorists design new ‘tools of analysis’: models that explain current developments (such as chronic state failure in sub-Saharan Africa) and that foreshadow dangers in order to guide policy. This thesis tests the hypothesis that increasing levels of emerging and re-emerging infectious diseases (ERIDs) act as

---


2 This project examines the effects of the most destructive emerging and re-emerging infectious diseases. The disease agents central to this project are HIV/AIDS, Malaria, Tuberculosis, Hepatitis, Cholera, Dengue, diarrhoeal rota and adenoviruses, plague, the BSE prion, and syncytial respiratory viruses. Different societies will be at risk from different pathogens, varying along differences in climate, wealth, and geographical position.
stressors on state capacity, undermining national prosperity and governance and possibly state survival.

Arguably, the primary raison d'etre of International Relations theory is to construct models that will assist in averting the premature loss of human life and productivity as a result of war. Indeed, as Thomas Hobbes claimed, it is the central function of the state to guarantee the physical safety of its citizens from both internal and external forms of predation. However, traditional concepts of security have ignored the greatest source of human misery and mortality: the microbial penumbra that surrounds our species. I argue here that it is time to consider the additional form of ecological predation wherein the physical security and prosperity of a state's populace is directly threatened by the global phenomena of emerging and re-emerging infectious disease.

Throughout this study, I shall use the following definition:

Emerging infectious diseases are those whose incidence in humans has increased during the last two decades or which threatens to increase in the near future. The term also applies to newly-appearing infectious diseases, or diseases that are spreading to new geographical areas - such as cholera in South America and yellow fever in Kenya. (Re-emerging infections are) diseases that were easily controlled by chemotherapy and antibiotics, but which have developed anti-microbial resistance.

---

Scope of the Problem

Throughout recorded history infectious disease has consistently accounted for the greatest proportion of human morbidity and mortality, surpassing war as the foremost threat to human life and prosperity. Even in the era of modern medicine, states annually suffer much greater mortality and morbidity from infectious disease than from casualties incurred during inter and intra-state military conflict. According to the World Bank, of the global total deaths recorded in 1990 (49,971,000), infectious disease claimed 16,690,000 lives (34.4 per cent of deaths), while war7 killed 322,000 (0.64 per cent of total deaths). These statistics demonstrate the relative destruction wrought by disease when compared to deaths from military actions, and in terms of a ratio the deaths resulting from infectious disease compared to war are a significant 52:1 for this year. Infectious disease also accounts for greater global morbidity and mortality than any other single cause.

According to the World Health Organization, of the 51,000,000 global deaths in 1993, infectious disease (ERIDs) caused 16,445,000 deaths (32.24 per cent of the total). By comparison, ‘motor and other-road vehicle accidents’ accounted for 885,000 deaths (or 1.7 per cent of global mortality), and ‘homicide and violence’ contributed to 303,000 deaths (0.6 per cent of global mortality).9

---

7 This incorporates statistics from both inter and intrastate warfare.
8 For purposes of clarification, ‘mortality’ represents deaths that result from infectious disease; ‘morbidity’ denotes serious physical debilitation of an individual as a result of prior or current exposure to infectious pathogens. These statistics on the causes of global deaths in 1990 are derived from World Bank, World Development Report 1993: Investing in Health, (New York: Oxford University Press, 1993), pp. 224-5.
A recent report by the CDC warns that “the spectrum of infectious diseases is expanding, and many infectious diseases once thought to be controlled are increasing.”

During the past two decades at least thirty-three new pathogens have emerged to compromise the health of the human species. For the majority of these new diseases there is no vaccine, therapy, or cure and the ability to anticipate, prevent or control them is extremely limited. The best known examples of emerging pathogens are HIV, Ebola, and the BSE prion; however, old scourges such as tuberculosis, cholera and malaria are becoming increasingly resistant to our anti-microbial armamentarium due to rapid microbial evolution, and are spreading across the globe.

It must be understood that infectious disease is one of humanity’s oldest and direst enemies. Various diseases have wracked societies from time immemorial, resulting in panic, debilitation, and death. As such we must recognize that we are dealing with a very old adversary here, and it is rather our growing understanding of how pathogens interact with the economic, political, and social factors that results in the sense of novelty in regards to the claim that infectious disease represents a threat to human development and security. Indeed, our ancient predecessors (Thucydides, Gibbon, Hippocrates) duly recognized the enormous negative social and economic impact of infectious diseases on their respective societies. If infectious disease is seen as a ‘new’ threat to political and

---

economic stability it is largely because our species tends to exhibit the affliction of short
generational memory.

Before the coming of the ‘golden age’ of antibiotics and vaccines in the early
1900s, diseases such as polio, smallpox, and tuberculosis were rampant on a global scale.
These lethal and crippling diseases combined with cholera, malaria and plague to kill and
disfigure millions. With the discovery of the cholera vibrio by the Prussian scientist
Robert Koch in 1883, humanity began its scientific examination and war against
infectious disease. Advances in public health have led to the general eradication of polio
in the developed world,\textsuperscript{13} the near-eradication of leprosy, and the selective incarceration
of the various strains of varoiila (smallpox) within U.S. and Russian military facilities.\textsuperscript{14}

Throughout the Twentieth Century the human species has witnessed successive
triumphs over the microbial world, to the extent that prominent experts in medicine
frequently spoke about the eradication of infectious disease and the subsequent need to
close public health programs and training facilities during the mid-1970s. This hubris led
in turn to complacency, and funding for research to control the spread of malaria (and
other vector-borne pathogens)\textsuperscript{15} was cut drastically from the mid-1970s on. Firms and
governments subsequently curtailed funding for improved anti-microbial agents and the
development of vaccines. In the minds of many, the enemy was vanquished and the
medical world turned to focus its wealth of resources on other health scourges such as
cancer, heart disease and genetically-transmitted infirmities.

The rapid emergence of HIV/AIDS in the early 1980's served as a wake up call to populations in Europe, North America, and Africa. Here was an example of a 'new' zoonosis rapidly expanding outward from its animal reservoir in Africa to infect millions on a global scale by the end of the century. Similarly, many other viruses began to emerge from the mid-1970s onward; pathogens causing haemorrhagic fevers such as the Ebola viruses (subtypes Zaire, Machupo, and Junin), and respiratory viruses such as Hanta throughout the Americas. New strains of bacteria such as legionella, e-coli O157, and cholera (El Tor) also began to conquer new territory, and adding to the threat was the new phenomenon of drug resistance in malaria, tuberculosis, enterococci (VRE), and staphylococcus aureas (MRSA). At the end of the millennium the human species finds itself again facing the resurgent specter of (ERID), and burdened with new zoonoses such as HIV that mutate rapidly, such that vaccine development has failed repeatedly and incredibly complex and expensive therapies are required to extend the life-span of individuals with AIDS in the developed world.

Critics of 'health security' argue that microbes and humanity have co-existed for millennia, and besides the collapse of a few empires and the deaths of billions, the human species has managed to survive. All of this is true, yet the human species finds itself in a very different world now; a world where individuals can travel around the globe via airplane rapidly, where global overpopulation and the growth of megacities create

---

15 Vector-borne pathogens are disease agents that are transmitted to humans through an intermediary animal or insect host. The Malaria plasmodium is a classic example of a vector-borne pathogen as it uses the mosquito to jump from one human host to another.

16 A zoonosis is a pathogenic agent that has crossed over from its original animal or insect host into human populations. Recent crossover pathogens tend to exhibit exceptional lethality in human populations (e.g. HIV, and falciparum malaria), but this lethality tends to decline over millennia as the pathogen and host adjust to each other.
entirely new ‘disease pools’ that will allow new pathogens to emerge and flourish within these greater population densities. This brave new world is also witnessing human-induced global environmental destruction that results in the release of pathogens from their ancient reservoirs in the core of rain forests, and where virulent new microbes result in the widespread destruction of aquatic life. Rapid global changes may accelerate the diffusion, lethality and resistance of the plethora of species within the microbial world, of which we have identified very few. While old scourges acquire resistance and conquer or reclaim territory within the human ecology, it is also likely that the natural processes of zoonotic transfer will persist and that new human pathogens will continue to emerge.

In the spring of 1996 the World Health Organization declared a global health emergency, as the global spread of ERID reached crisis proportions, and now exceeds the organization’s capacity to monitor, let alone contain, the various pandemics currently spreading across the globe.17 A synopsis of the spread of the most important known pathogens is appropriate, to give the reader an idea of the scale of the problem.

Trends in Disease Emergence

It is important to remain as objective as possible in this type of inquiry as much hyperbole exists on the subject of health security courtesy of Hollywood, some journalists, and fiction writers. We must ask pertinent questions: (1) Is the prevalence of a particular infectious pathogen rising within a given state’s population and at the global level as well? (2) Is this pathogen moving into new geographic regions or reclaiming lost
territory, and is it affecting new demographics within given societies? (3) What regions of the globe (if any) are particularly vulnerable to this resurgence in infectious disease, and where are the greatest increases in prevalence taking place? (4) At what rates are these pathogens expanding their territories, both demographic and geographic?

The global proliferation of HIV/AIDS over the past two decades has resulted in a staggering amount of human suffering, death, debilitation and fear. From the earliest genetic traces of HIV proto-DNA, culled from the tissues of a Zairean male who died in 1954, the pathogen has spread relentlessly outward from its Central African epicentre to the Americas, Western Europe and is now spreading rapidly through populations in South and East Asia, and Eastern Europe.

According to statistics gathered by the Harvard-based Global AIDS Policy Coalition, approximately 22 million people were infected with HIV/AIDS, and 4.7 million new infections occurred globally during 1995. Of these new infections, 2.5 million occurred in Southeast Asia and 1.9 million in sub-Saharan Africa while the industrialized world accounted for approximately 170 000 new HIV cases. The pace of the HIV/AIDS pandemic is accelerating, with a total of 33.4 million people now

---


18 On occasion, emergent illnesses may shift their effect on a given population distribution, from one region of the distribution to another. The classic case is that of Spanish Influenza which killed otherwise healthy adults in the prime of their lives. Normally influenza is only fatal to the extremely young and very old, who possess weakened immune systems.


21 An epidemic is an outbreak of disease that exhibits both geographical and temporal boundaries. One example is the brief outbreak of Plague in Surat, India during the fall of 1994. A pandemic is a outbreak of disease that is not geographically restricted (e.g. HIV), nor is it necessarily confined within a set time frame (e.g. malaria, tuberculosis).
infected, 5.8 million new HIV infections annually, and 2.5 million HIV-induced deaths in 1998.22 Thus the global pace of infection has increased by 24 per cent over 1995 levels. The HIV pandemic now rivals (in terms of the absolute magnitude of mortality) the greatest plagues of history including the Black Death of Middle Ages Europe and the Influenza Pandemic of 1918, both of which killed over 20 million people. To date the HIV pandemic has resulted in the infection of 47 million and resulted in the death of 14 million people, and the contagion is spreading rapidly throughout South and South-East Asia, Eastern Europe, and Latin America.23

The heart of the HIV pandemic lies in Sub-Saharan Africa where many states are now reporting HIV seroprevalence24 levels in excess of 10 per cent. Indeed, South Africa, Kenya, Uganda, Zambia, Namibia, Swaziland, Botswana and Zimbabwe all have seroprevalence levels ranging from 10 to 25 per cent of the population.25 Botswana, for example, has seen national HIV seroprevalence rates rise from 10 per cent in 1992 to 25.1 per cent in 1997, an increase of 250 per cent over five years.26 South Africa has seen total HIV infection levels rise from 1.4 million in 1995 to over 3 million in 1998.27 This represents an increase of HIV seroprevalence in the South African population of over 200 per cent in 3 years time. Some regions within these states have even higher infection

23 Ibid, p. 3.
24 Seroprevalence is an estimation of the rate of pathogen-infected individuals in a given population set. For example, if country X shows 20 per cent HIV seroprevalence, then 20 per cent of that country’s population is HIV positive.
25 Namibia and Swaziland currently report HIV seroprevalence levels in excess of 20 per cent, and Zimbabwe, Botswana have the dubious distinction of having infection levels of over 25 per cent of the total population. See the individual country annual seroprevalence statistics available at http://www.unaids.org/highband .
levels: HIV prevalence in KwaZulu-Natal (South Africa) has now reached the level of 30 per cent, and Francistown in Botswana reports that 43 per cent of its citizens are infected. Certain towns along the South African – Zimbabwe border boast astonishing rates of approximately 70 per cent HIV seroprevalence.

The pandemic is expanding into Eastern Europe at an ever-increasing pace. Former Russian Minister of Health Tatyana Dmitriyeva has predicted that over 1 million Russians will be infected with HIV by the year 2000. Ukraine has also seen HIV incidence soar from a modest 44 cases in 1994 to an astonishing 110 000 cases as of mid-1998. India is also seeing HIV spread throughout its vast population at a rapacious pace. Five years ago, AIDS was practically unheard of in India; now almost 1 per cent of all pregnant women tested throughout the country are HIV positive. Disturbingly, by 1997 the epidemic was already firmly entrenched in regions of India such as Nagaland along the Burmese border (7.8% HIV seroprevalence), and nearby Manipur (over 10% HIV seroprevalence). Indeed, with the exception of the developed world, and certain states such as Uganda and Thailand (which have seen some reduction in the rate of new infections), the HIV pandemic continues to expand at a rapid pace.

Tuberculosis (TB) has been making a steady comeback as a global scourge, and WHO declared the TB pandemic a global crisis in 1993. WHO estimates that “8.9 million

people developed tuberculosis in 1995, bringing the global total of sufferers to about 22 million, of whom about 3 million will have died in the same space of time.\(^{35}\)

Furthermore, in the absence of increased effectiveness and availability of tuberculosis control measures, over 30 million tuberculosis deaths and more than 90 million new TB infections are forecast to occur by the turn of the century.\(^{36}\) Tuberculosis is making inroads into the industrialized nations, particularly Canada and the United States, where it infects disadvantaged urban and incarcerated populations and then spreads throughout society. The incidence of tuberculosis in the United States is climbing rapidly. For example, in the U.S., reported cases of TB had declined from 84 300 in 1953 to 22 200 in 1984, a drop of approximately 4 per cent per annum. However, from 1985 to 1993, the number of cases increased by a cumulative 14%, and the pace of increase continues to accelerate.\(^{37}\) Similarly, Zimbabwe has reported massive increases in TB incidence, from 5000 cases in 1986 to 35 000 cases in 1997.\(^{38}\) Feschbach notes that the incidence of tuberculosis in Russia is increasing rapidly, and based on estimates provided by the Russian Ministry of the Interior, he predicts that tuberculosis will result in the deaths of 1.75 million Russians per year by 2000.\(^{39}\)

---

36 Ibid, p. 27.
37 Ibid, p. 28.
38 Ibid; p. 27.
40 Feschbach, p. 27.
Malaria continues its relentless expansion into former regions of endemicity. For example, in 1989 malaria claimed 100 lives in Zimbabwe while debilitating many thousands; by 1997 malaria was responsible for the deaths of 2800 in that country, an astonishing rate of increase for a disease that was once thought to be controlled. Indeed, the best available estimates project that malaria currently claims 5000 lives every day in Africa, approximately 1.8 million deaths a year. Global estimates put the total annual death rate from malaria at upwards of 2.7 million and note that malaria debilitates as many as 500 million people every year. Shell claims that global incidence of malaria has increased by approximately 400 per cent over the 1992-97 period, and notes that the disease has re-emerged in North America from urban centres in California to Michigan, to New York City and Toronto.

Other pathogens are also re-emerging on a global scale throughout the developing world and are increasingly penetrating the porous borders of the industrialized states. For example, a new strain of cholera (designated 0139 El Tor) appeared in south-eastern India in 1992 and is now endemic throughout South and South East Asia, Africa, South and Central America and is spreading rapidly through Oceania. Moreover, mosquito-borne dengue fever has re-established itself in Central America and Mexico and is currently

---

40 Endemic diseases are those that are geographically but not temporally bounded. In other words, malaria has always been present in north western Brazil (endemic), but it has not always been present in Patagonia (non-endemic). Human actions had pushed malaria out of certain regions where the disease had been endemic prior to the 1960s, but vector-resistance to pesticides and new strains of malaria have seen the pathogen re-conquering its former territory.
42 Ellen Ruppel Shell, “Resurgence of a Deadly Disease” Atlantic Monthly, August 1997, p. 47.
44 Ibid, p. 45. Malaria’s re-emergence as an endemically transmitted pathogen in Toronto, Canada has been verified by Kevin Kain of the Tropical Disease Unit, Toronto General Hospital. Comments made to the author 30 October 1998.
making inroads into the southern United States, particularly in Florida, Louisiana, and Texas.

Meanwhile familiar pathogens continue to exact their toll on humanity with relentless vigor. For example, acute lower respiratory infections slay nearly 4 million children annually, while diarrhoeal diseases such as adnovirus and rotavirus kill nearly 3 million infants every year. Viral hepatitis is another global scourge as a minimum of 350 million people are chronic carriers of the hepatitis B virus, and an additional 100 million harbor the hepatitis C virus. According to WHO projections at least 25 per cent of these carriers will expire due to related liver disease. To make matters worse, many of the ten million new cases of cancer diagnosed in 1995 were caused by viruses, bacteria and parasites. WHO calculates that 15 per cent of all new cancer cases (1.5 million) are the result of exposure to infectious agents, and this percentage of ERID-induced cancer mortality is estimated to increase as our knowledge of both infectious disease and cancer advances. New evidence is linking many other supposedly chronic or genetic diseases such as heart disease and Multiple Sclerosis to common infectious agents (chlamydia and herpes, respectively) which promote long-term disease processes within human hosts. If certain conditions such as cancer, heart disease, and MS, are in fact pathogen-induced, then the global burden of infectious disease may be far greater than we previously thought.

While it is relatively easy to see that ERID is a central agent of misery throughout the developing world, it is not often apparent that infection-induced mortality has been on

---

the rise in the developed world as well. For example, the United States, which is arguably the only superpower and has enormous levels of State Capacity,\(^47\) has seen steadily increasing mortality from infectious disease over the last two decades, rising from 15,360 deaths in 1979 to 77,128 ERID-induced deaths in 1995,\(^48\) a significant increase of 502% over that time period.

The State of Knowledge on Health Security

Without exception, the literature on health security is particularly thin, likely due to the novelty of the concept. The few works that do exist have succeeded in raising interest in the issue area, and have spurred deeper analysis of the hypothesis that pathogens present a threat to national security and development. One example is Laurie Garrett’s book *The Coming Plague*,\(^49\) which enjoyed considerable attention within the United States’ foreign policy and security establishment. It contains a decent overview of the various conditions that have led to the global resurgence of ERID. In the final chapter, Garrett claims that the global proliferation of disease poses a threat to U.S. National Security. Garrett reiterates this point in her *Foreign Policy* article, “The Return of Infectious Disease,”\(^50\) wherein she attempts to clarify the particular threat that ERID poses to American global interests and national security. The reader is left with the


\(^{47}\) I provide a detailed technical definition of State Capacity in Chapter Two, and I am capitalizing the term here to indicate a specific technical meaning.

\(^{48}\) See [http://cdc.gov/nchs-www/fastats](http://cdc.gov/nchs-www/fastats) for this data.


\(^{50}\) Laurie Garrett, “The Return of Infectious Disease” *Foreign Policy*, Jan/Feb 1996, pp. 66-79.
impression that Garrett’s claims have some inherent common sense, and she provides us
with numerous examples of disease emergence on a global scale. Garrett’s works are
notable in that they first brought the issue of health security to the attention of the policy
community, and raised the possibility that the return of infectious disease might
consistute a significant threat to American interests.

Similarly, Dennis Pirages expounds upon the power of infectious disease as a
threat to state security and foreign policy interests. His work provides an important initial
blueprint for further investigation in the realm of health security, and he provides
interesting anecdotal evidence that the global resurgence of infectious disease is directly
related to human-induced changes in the biosphere. Given that this pioneering work had
little to build upon it stands as a reasonable first attempt to clarify the issues, and suggests
many avenues for further research.

Infectious diseases are potentially the largest threat to human security
lurking in the post-Cold war world. Emerging from the Cold-war era, it is
understandably difficult to reprogram security thinking to take account of
non-military threats. But a new focus that included microsecurity issues
could lead to interesting cost-benefit thinking. Winning the war against
new and reemerging infectious diseases requires both long-term and
immediate changes. Educating people to think about this struggle with
microbes in an evolutionary way is the ultimate solution. In the short term,
policymakers need to understand the potential seriousness of the problem
and reallocate resources accordingly.51

While Pirages’ and Garrett’s theories are both intuitively appealing, they fail to address
the global impact of ERID, namely whether the resurgence of disease will have a
differential impact upon different societies. Does ERID constitute a direct or indirect
threat to states and/or societies, and is it a greater threat to some regions of the world than
others? Recent advances have also been made by historians who have traced the impact of various pathogens on societies and warfare across the centuries. While the evidence is largely (if not entirely) anecdotal, the historical community does a good job of examining the diverse effects of infectious disease on the societies in question over time.

**Historical Overview**

As William McNeill proposed in *Plagues and Peoples*, microbes have been relentless adversaries of humanity and of human societies since time immemorial.

Current anthropological evidence suggests that the expansion and collapse of various societies throughout history may have resulted in part from the transmission of lethal and/or debilitating pathogens. Thucydides' account of the eventual fall of Athens during the Peloponessian Wars pays particular attention to the devastating effect that 'the plague' had on Athenian governance, and by extension the Athenian war effort.

> The bodies of the dying were heaped one on top of the other, and half-dead creatures could be seen staggering about in the streets or flocking around the fountains in their desire for water. For the catastrophe was so overwhelming that men, not knowing what would happen next to them, became indifferent to every rule of religion or law. Athens owed to the plague the beginnings of a state of unprecedented lawlessness. Seeing how quick and abrupt were the changes of fortune...people now began openly to venture on acts of self-indulgence which before then they used to keep in the dark. As for what is called honor, no one showed himself willing to abide by its laws, so doubtful was it whether one would survive to enjoy the name for it. No fear of god or law of man had a restraining influence. As for the gods, it seemed to be the same thing whether one worshipped them or not, when one saw the good and the bad dying indiscriminately. As for offences against human law, no one expected to live long enough to be brought to trial and punished.\(^2\)

---


McNeill argued that the collapse of the Byzantine Roman empire in the sixth century A.D. resulted from the 'plague of Justinian' which was a consequence of the merging of two previously isolated disease 'pools' via Asian trade routes (the Silk Road). Gibbon recounts the devastation wrought by the plague.

I only find that, during three months, five and at length ten thousand persons died each day at Constantinople; and many cities of the East were left vacant, and that in several districts of Italy the harvest and the vintage withered on the ground. The triple scourges of war, pestilence and famine afflicted the subjects of Justinian; and his reign is disgraced by a visible decrease of the human species which has never been regained in some of the fairest countries of the globe.

The destruction of feudalism may have also resulted in large part from the recurrent waves of bubonic and pneumonic plague (i.e. the Black Death) that repeatedly swept Europe throughout the 14th and 15th centuries. [In a sense then, 'bubonic plague' (the Black Death) is a progenitor of the entire system of states as we know it.] Alfred Crosby and William Denevan have constructed detailed accounts of how the merging of the American and European disease pools permitted the rapid and absolute conquest of the Americas by relatively modest European military forces. This demographic catastrophe derived from the importation of 'civilized' diseases (smallpox, etc.) to an immunologically naive population, and resulted in the collapse of the Aztec and Incan empires and centuries of subjugation of the Amerindian peoples. McNeill puts the Amerindian population at the beginning of the conquest at approximately 100 million.

---

53 Concerning the gradual collapse of the Roman Empire and the Han Dynasty as well see McNeill, Plagues and Peoples, pp. 101-106.
55 Ibid, pp. 132-175.
56 The conquest of the American peoples by smaller European forces through infectious disease is described in W.M. Denevan. The Native Population of the Americas in 1492, (Madison, WI: University of
Starting from such levels, population decay was catastrophic. By 1568, less than fifty years from the time Cortez inaugurated epidemiological as well as other exchanges between Amerindian and European populations, the population of Mexico had shrunk to about three million, [in other words] to about one tenth of what had been there when Cortez landed. Decay continued, though at a reduced rate, for another fifty years. Population reached a low point of about 1.6 million by 1620. [Such a disaster] carries with it drastic psychological and cultural consequences. Faith in established institutions and beliefs cannot easily withstand such disaster; skills and knowledge disappear. Labour shortage and economic regression was another obvious concomitant.  

Infectious disease continued to play a role in the evolution of political entities, and may have had a significant impact on the outcome of the American Revolutionary War of 1776, as smallpox helped to prevent the armies of the United States from capturing Canada.

During the Revolutionary War, the American colonial government sent an army to wrest Canada away from the English. Having captured Montreal, the colonial army, superior in number, marched on to engage in the conquest of Quebec City. But smallpox entered their ranks. The decimated American army, soon after burying their dead in mass graves, retreated in disorder from Quebec.  

Oldstone notes that of the 10,000 US troops originally involved in the campaign, 5,500 developed smallpox and died, effectively nullifying the American offensive and allowing Britain to maintain its stronghold in British North America. ERIDs also caused significant governance problems on occasion for the fledgling United States as evident during the Yellow Fever Epidemic of Philadelphia in 1793.


Philadelphia had suffered a previous yellow fever plague in 1762, when a hundred had died, but now thousands were dying. Thomas Jefferson wrote from Philadelphia to James Madison in Virginia, telling about the fever, how everyone who could was fleeing and how one of every three stricken had died. Alexander Hamilton, the secretary of the Treasury, came down with the fever. He left town, but when he was refused entry to New York City, he turned to upstate New York... There he and his wife were obliged to stay under armed guard until their clothing and baggage had been burned, their servants and carriage disinfected. Clerks in the departments of the federal government could not be kept at their desks. In the Treasury Department, six clerks got yellow fever and five others fled to New York; three sickened in the Post Office and seven officers in the Customs Service. Government papers were locked up in closed houses when the clerks left. By September, the American government came to a standstill.\textsuperscript{60}

This abbreviated overview of the possible historical impact of infectious disease on the currents of history is merely intended to demonstrate to the reader the profound relationship between forces of the natural world, such as pathogens, and the evolution of human societies. This is not to imply that diseases have been the major force in defining the outcomes of all human history;\textsuperscript{61} to argue such brings us to the shores of biological determinism, a conceptual model unlikely to take us very far. Yet, it is fascinating to note that biological forces may, in fact, have had a significant effect on the broader outlines of human history, and will likely continue to do so as disease continues to proliferate on a global scale.

Redefining Security

\textit{Swords and lances, arrows, machine guns, and even high explosives have had far less power over the fates of the nations than the typhus louse, the plague flea, and the yellow-fever mosquito. Civilizations have retreated}

\textsuperscript{60} Oldstone, pp. 47-8.
\textsuperscript{61} McNeill makes this argument, occasionally verging on biological determinism, in \textit{Plagues and Peoples}.
from the plasmodium of malaria, and armies have crumbled into rabbles under the onslaught of cholera spirilla, or of dysentery and typhoid bacilli. War and conquest and that herd existence which is an accompaniment of what we call civilization have merely set the stage for these more powerful agents of human tragedy.

- Hans Zinsser

The skeptic will ask, why should international relations theorists be concerned with what essentially amounts to a public health problem? Why should infectious disease concern us now in the modern era near the end of the Twentieth Century? The world has changed significantly since the medical ‘golden era’ of the mid-1900s. Humanity currently finds itself in a state of profound ecological disequilibrium, where factors such as the rapid destruction of the biosphere, changes in the speed and availability of transport technologies, rapidly increasing global population density and migration, economic development, and the overuse and misuse of antibiotics, have all contributed to the emergence of new pathogens and the re-emergence of diseases previously thought to have been under control.

The inclusion of disease in the security agenda poses a challenge to orthodox theories of international relations, particularly to Neo-Realism and Realism. This type of inter-disciplinary research agenda requires a fundamental reconceptualization of standard definitions of national interest and security. Constrictive definitions that focus exclusively on the relative military capability of states are increasingly sterile in the face of the many global challenges of the post-Cold War world. Threats to human welfare such as global environmental degradation, resource scarcity, population growth, and infectious disease present policy makers with difficult policy dilemmas in the form of collective action.
problems. Richard Ullman argued that “defining national security merely in military terms conveys a profoundly false image of reality...it causes states to concentrate on military threats and to ignore other and perhaps more harmful dangers.”\textsuperscript{63} The same can be said of Realist concepts of international security, which focus exclusively on military threats and the relative power of states within the context of an eternal security dilemma.\textsuperscript{64}

Of particular interest is the recent work of Kalevi Holsti on the changing nature of conflict in the post-1945 era. Holsti has found that the incidence of interstate war has declined precipitously since the 19\textsuperscript{th} century, and that the current principal foci of violence are at the intra-state level.\textsuperscript{65} If Holsti’s calculations are correct, then political scientists concerned with the study of conflict must shift their focus to concentrate on the growing incidence of intra-state violence, and develop theoretical models that explain the recent collapse of states like Zaire, Rwanda and Haiti. Holsti emphasizes the need for this conceptual shift in security studies.

Overall... strategic studies continue to be seriously divorced from the practices of war.... Most fundamentally, the assumption that the problem of war is primarily a problem of the relations between states has to be seriously questioned. The argument ...is that security between states in the Third World, among some of the former republics of the Soviet Union, and elsewhere has become increasingly dependent upon security within those states. The trend is clear: the threat of war between countries is

\begin{quotation}
\textsuperscript{62} Zinsser, pp. 9-10.
\textsuperscript{65} Holsti argues that “...there have been on average only 0.005 interstate wars and armed interventions per state per year since the end of World War II. This figure contrasts to the 0.019 wars per state annually in the European states system of the eighteenth century, 0.014 in the nineteenth century, and 0.036 in the 1919-39 period.” See Kalevi J. Holsti. \textit{The State, War, and the State of War}, (Cambridge: Cambridge University Press, 1996), p. 23. Also see Edward Rice. \textit{Wars of the Third Kind: Conflict in Underdeveloped Countries}. (Berkeley: University of California Press, 1988).
\end{quotation}
receding, while the incidence of violence within states is on an upward curve.\textsuperscript{66}

Given this shift in the loci of conflict, from the inter-state to the intra-state level, Ayoob notes that the majority of these recent conflicts now occur in the developing world.\textsuperscript{67} The existing literature in the field has developed two principal hypotheses that link environmental and demographic change to intra-state violence: the relative deprivation and state weakness hypotheses.

The deprivation hypothesis argues that certain processes, such as environmental scarcity and demographic growth, may have a significant negative long-term effect on individual living standards and quality of life within a given society, inducing either relative or absolute deprivation.\textsuperscript{68} Increasing levels of deprivation eventually translate into increasing frustration, that in turn generates increasing aggression by disaffected individuals and collectivities. Thus, greater deprivation may generate increasing social violence and political chaos.\textsuperscript{69} As Eckstein and Kahl have noted, the deprivation hypothesis is incapable of explaining political violence in and of itself.\textsuperscript{70} Specifically, it generates an excess number of false-positives in its prediction of sub-state violence. If the

\textsuperscript{66} Holsti, pp. 15-6.
hypothesis were true, then most poor nations of the earth would be caught up in a perpetual maelstrom of political chaos and violence, and this is simply not the case. In all likelihood, this lack of rebellion derives from the fact that the poor frequently lack the organizational capacity, economic resources, and opportunity to rebel. Thus, not every state that suffers from poverty is exceptionally prone to chronic internal violence, indeed conflict seems to occur more frequently when mounting deprivation is combined with declining state capacity, which generates increasing probability that the desire to redress perceived or absolute inequalities between classes, ethnicities and elites, may provoke collective violence.\(^1\)

The state weakness hypothesis suggests that intra-state organized violence tends to occur when stressor variables (poverty, environmental scarcity, etc) create both opportunities and incentives for citizens to engage in collective violent action against the status quo. These stressors create increasing competition between groups within the general polity for increasingly scarce resources, thus creating additional incentive for violent action against competitor groups or against the state itself. Additionally, stressor variables may both increase demands upon the state to provide services, while simultaneously reducing state capacity, increasing institutional fragility, and undermining the cohesion and legitimacy of the state.\(^2\) Thus the prospects for the success of rebellious action improve – providing greater incentive for collective violence.


As I demonstrate in chapters 3 and 4, the proliferation of infectious disease may significantly reduce individual and societal prosperity, producing absolute and relative deprivation in seriously affected populations. Rapid negative change in the health status of a given population and pathogen-induced demographic collapse may therefore play a role in the destabilization of states. This study demonstrates that pathogen-induced declines in population health generate increasing poverty at both the individual and macro level in a given society, and widen economic disparities between social classes within a given society. Thus, this dissertation builds upon and compliments the arguments of deprivation and state weakness theory.

Building upon the state weakness hypothesis, I argue that rapid and significant declines in population health may give citizens greater incentives to engage in violent action, given that the opportunity to gain from collective violence increases as state capacity declines, reducing the states capability to govern effectively and maintain order. Thus, the effect of increasing poverty and human misery (deprivation) combines with the erosion of state capacity (governance) to generate the preconditions for widespread social destabilization. ERID is then simultaneously a generator of absolute deprivation and a stressor on state capacity. Therefore, the increasing prevalence of infectious diseases in a given society results in declining public health, which generates deprivation and then increases the probability of organized collective violence. Therefore, while ERID may be a significant contributor to the preconditions for sub-state violence and state failure, it is unlikely to generate these effects in and of itself. It is important to
recognize that ERID may combine with environmental degradation and scarcity to magnify both relative and absolute deprivation, and hasten the erosion of state capacity in seriously affected societies. Thus, ERID may in fact contribute to societal destabilization, chronic low-intensity intra-state violence, and in extreme cases, accelerate the processes that lead to state failure.

Microbial threats to human health do not respect international borders, and are extremely difficult to monitor and contain. Infectious disease constitutes a truly global challenge, and as a global challenge it must be met with international cooperation. The implication here is that Realist policy prescriptions will not protect states from the negative consequences of ERID resurgence. Liberal theory, which emphasizes cooperation between states, holds an optimistic view of human nature, sees international organizations as significant actors, and argues that states seek prosperity and stability in addition to survival and power, is likely to provide a better theoretical foundation than Realism for tackling the problems posed by the resurgence of infectious disease and other global issues. Unilateral efforts that focus on limiting the spread of ERID within one’s own state are bound to fail over the long term, as ERID is a transboundary phenomenon.\(^4\) Liberal solutions to ERID would take the form of a global multilateral health regime that would seek to prevent, monitor and control the spread of ERID. In fact, regimes already


\(^{74}\) The construction of a global ERID surveillance and containment regime requires significant investments to be made, and maintained, in the health infrastructure of all states to permit the diagnosis and treatment of disease on a global scale. This is no mean feat when one considers that the only facility in the world with a 100 per cent rate of pathogen diagnosis is the CDC. The central diagnostic laboratories in other developed countries like Canada, France, and Britain only succeeded in correctly identifying 50 per cent of the pathogens sent to them in a recent test by the CDC. See Garret, *The Coming Plague*, p. 606.
exist to deal with the spread of pathogens between sovereign states, and a nascent global surveillance system (ProMED) has proved highly effective in monitoring pathogen outbreak events on a global scale. Jessica Tuchman Mathews writes, “On the political front, the need... for new institutions and regulatory regimes to cope with the world’s growing environmental interdependence is...compelling. Put bluntly, our accepted definition of the limits of national sovereignty as coinciding with national borders is obsolete.”

Despite the enormous technological and economic power of the North, it is extremely unlikely that the developed world will be able to remain an island of health in a global sea of disease. Global interdependence applies to the microbial threat just as it applies to the pervasive degradation of the biosphere, and unilateral, isolationist policies will only compromise the prosperity and well-being of all peoples over time. American policymakers are increasingly cognizant of the threat that the resurgence of infectious disease may pose to U.S. interests. Within the Clinton administration’s national security strategy of ‘engagement and enlargement,’ infectious disease (as a function of global environmental degradation) is noted as a novel threat to American foreign policy interests, particularly economic growth and democratic stability in the developing world.

New diseases, such as AIDS, and other epidemics which can be spread through environmental degradation, threaten to overwhelm the health facilities of developing countries, disrupt societies and stop economic growth. Developing countries must address these realities with national sustainable development programs that offer viable alternatives. U.S. leadership is of the

---


essence to facilitate that progress. If such alternatives are not developed, the consequences for the planet's future will be grave indeed.\textsuperscript{77}

President Clinton appointed the National Science Council on Emerging and Re-Emerging Infectious Diseases (NSTC) to determine the direct and indirect threat of infectious disease to U.S. security and prosperity and to further evaluate the potential impact of ERIDs on American foreign policy interests at the global level. The report of the NSTC states that, "the improvement of international health is a valuable component of the U.S. effort to promote worldwide political stability through sustainable economic development. Thus, the effort to build a global (ERID) surveillance and response system is in accord with the national security and foreign policy goals of the United States."\textsuperscript{78}

Former Undersecretary of State for Global Affairs Timothy Wirth was also keenly aware of the threat that HIV/AIDS poses to state stability and prosperity. He argued,

It is...evident that as the pandemic spreads, HIV/AIDS has potentially devastating impacts on whole sectors of societies. In the most vulnerable nations, these trends could have devastating consequences for sustainable development and contribute to conflict and instability. [W]e must understand the pandemic for its ability to affect the social, economic, and political fabric of many nations and, thus, its implications for U.S. foreign policy, American leadership, and global cooperation. Viewed in the context of national security interests, many countries are today waging (and losing) a war with this infectious disease.\textsuperscript{79}

At this point in time, the United States under the Clinton administration is in the vanguard in terms of recognizing and dealing with the growing threat of infectious disease. Other countries (such as Canada and the United Kingdom) have pledged limited


funding towards the development of a global disease surveillance system, but the vast majority of the world has yet to recognize the severity of the ERID problem, let alone participate in the construction and consolidation of nascent global ERID surveillance and control regimes. It should be noted that cooperative international legal agreements on the containment of infectious disease outbreaks have existed since the Black Death swept through the city states of Italy in the 14th century. Mark Zacher and David Fidler argue that the aged practice of regarding infectious disease as an international threat, and the subsequent emergence of norms and codified international law to deal with these issues, constitute some of the first international regimes to govern transborder problems.\footnote{Wirth, Timothy E. “Foreward” in Kimberly A. Hamilton, \textit{Global HIV/AIDS: A Strategy for U.S. Leadership.} (Washington, D.C.: Center for Strategic and International Studies, 1994), p. vii.} Despite this legacy of international law dealing with stemming the tides of infection, international surveillance regimes are still at a very early stage, with initiatives coming from both civil society on the one hand, and state-centric militarized sources on the other. The ProMED surveillance network is a global civilian-based epidemiological surveillance network based on individual physicians reporting disease outbreaks via the internet to a central facility, whereupon significant epidemiological events are then transmitted via e-mail to all users on the system.\footnote{Mark W. Zacher, “Epidemiological Surveillance: International Cooperation to Monitor Infectious Diseases.” In Inge Kaul, Marc Stern, and Isabelle Grunberg, eds., \textit{Global Public Goods}, (Oxford: Oxford University Press, 1999), pp. 268-285; and Fidler, “Microbialpolitik: Infectious Diseases and International Relations,” pp. 1-53.} A militarized epidemiological surveillance system is the USDOD’s Global Epidemiological Information System (GEIS), which uses a number of
military bases around the globe to gather and analyze information on pathogen prevalence and emerging disease outbreaks.82

The presence of ERIDs in foreign military theatres has resulted in the past, and continues to result in the exposure of troops to previously unknown pathogens. Troop movements frequently serve as 'vectors' (modes of transmission) for diseases to move rapidly around the globe to areas where the new host population has little natural immunity to the new pathogen that is introduced into the local ecology. During World War I malaria was transmitted as far north as Archangel because of the troop vectors involved in the war. Subsequently, the influenza and typhus pandemics of 1918 claimed almost 40 million lives as they circled the globe along with the moving armies.83 The Second World War saw enormous morbidity of Allied troops in the Pacific theater due to malaria, and during the Korean War, U.S. troops were exposed to the Seoul Hantaan virus that subsequently traveled via troop supply ships to the United States, where it is now endemic.84 War can act as a disease 'amplifier', creating physical conditions (poverty, famine, and large population movements) that are conducive to the spread and mutation of ERIDs.

The presence of ERIDs in military populations is consequential as it jeopardizes military readiness, international cooperation, national security, and the ability of a state to preserve its territorial integrity. At the intra-state level, ERIDs deplete force strength through the loss of skilled military personnel, reduce the supply of able draftees or

---

82 Author's numerous conversations with the architect of GEIS, Lt. Col. Patrick Kelley of USDOD, Army. Walter Reed Army Institute of Medicine, Alexandria, VA, 1998-99.
recruits and impose costs that constrain military budgets, all of which impair the state’s capacity to defend itself against a potential aggressor and limits the state’s ability to project power abroad.

Effects of Infectious Disease on the State

*Disease and Economic Productivity*

The negative effects of ERID in the domain of economic productivity include reductions in national GDP and Government expenditure (per capita), decreases in worker productivity, labour shortages and increased absenteeism, higher costs imposed on household units (particularly on the poor), reductions in per capita income, reduced savings, and increases in income inequalities within a society that may in turn generate increased governance problems. ERID also generates disincentives to invest in child education, impedes the settlement of marginal regions and the development of natural resources, negatively affects tourism, and results in the embargoing of infected goods. The significant negative association between increasing ERID levels and the economic prosperity of affected societies may lead to mounting absolute and relative economic deprivation in affected states. All of these effects, taken together, demonstrate how the global resurgence of infectious disease is likely to produce negative outcomes for the prosperity of states.
Disease and Governance

The effects of a succession of epidemics upon a state are not measurable in mortalities alone. Whenever pestilences have attained particularly terrifying proportions, their secondary consequences have been much more far-reaching and disorganizing than anything that could have resulted from the mere numerical reduction of the population. Panic bred social and moral disorganization: farms were abandoned, and there was shortage of food; famine led to displacement of populations, to revolution, to civil war, and, in some instances, to fanatical religious movements which contributed to profound spiritual and political transformations.

- Hans Zinsser

At the unit level, in the domain of governance, high levels of ERID incidence undermine the capacity of political leaders and their respective bureaucracies to govern effectively as the infection of government personnel results in the debilitation and death of skilled administrators that oversee the day to day operations of governance. The destructive impact of ERID-induced mortality in human-capital intensive institutions generates institutional fragility that will undermine the stability of nascent democratic societies. In Zimbabwe an estimated 25 per cent of urban adults in the 19 to 45 age group are HIV positive, and at least three government ministers have succumbed to AIDS in recent years. Hugette Labelle of CIDA estimates that circa 50% of the armed forces and police forces in Zambia are HIV positive as of 1999. When these individuals perish, there will be enormous negative repercussions for governance in afflicted societies, with a likely corresponding rise in crime, civil unrest and low-intensity violence.

__References__

Zinsser, pp. 128-9.


Hugette Labelle, CIDA. Speech to the International Health Program at the University of Toronto. Friday, 26 February, 1999, Toronto.
Possible Systems Level Effects

ERIDs negative effect on state capacity at the unit level may produce related 
pernicious outcomes at the systems level. Within the domain of economics, as ERID 
produces a significant drag on the economies of affected countries, we may see chronic 
underdevelopment, which may in turn exert a net drag on global trade and impair global 
prosperity. In all likelihood, due to the nature of spiral dynamics inherent in the 
relationship between ERID and SC, countries with low initial levels of SC will suffer 
greater losses over time from increasing prevalence of infectious disease within their 
populations. Due to this negative spiral effect, ERID’s negative influence on the 
economic development of states may exacerbate the economic divide between North and 
South. Furthermore, the negative effects of infectious disease are not confined to the 
developing world. At the systems level, trade goods from ERID-affected regions may be 
subject to international embargo (e.g. Bovine Spongiform Encephalopathy\textsuperscript{38} in British 
beef, and influenza-infected chickens in Hong Kong). As infectious agents continue to 
emerge and re-emerge, and as agricultural crops and animal stocks become increasingly 
infested, we should expect that presumably infected trade goods from affected states will 
be embargoed, tourism to affected regions may decline, and economic damage to affected 
states will likely intensify.

This study will demonstrate that increasing levels of ERID correlate with a 
decline in the State Capacity. As State Capacity declines, coupled with an increase in

\textsuperscript{38} Bovine Spongiform Encephalopathy is a prion-induced condition in cattle that results in massive 
deterioration of the brain, ultimately causing death. These prions (infective proteins) may be transmitted 
via contact with infected meat to humans, causing Creutzfeld-Jacob disease in the human host which 
usually results in death.
pathogen-induced deprivation, and increasing demands upon the state, we may observe an attendant increase in the incidence of chronic sub-state violence and state failure. State failure frequently produces chaos in affected regions, as neighboring states seal their borders to prevent the massive influx of ERID-infected refugee populations. Adjacent states may also seek to fill the power vacuum, and seize valued territory from the collapsing state, prompting other proximate states to do the same, exacerbating regional security dilemmas. An example of this is the widening conflict in Central Africa, where the collapse of governance in Zaire (and continuing insurgency in the successor state, the Democratic Republic of the Congo) is generating a wider conflict wherein the mercenary armies of Uganda and Rwanda seek to topple the fragile government of Laurent Kabila in Kinshasa. Conversely, military forces from Angola, Namibia, and Zimbabwe, Sudan and Chad have recently been deployed to the Democratic Republic of the Congo to crush the aspirations of the rebels and their masters in Kigali and Kampala.

As ERID incidence and lethality increase, deprivation will mount and SC will decline, generating increasing levels of stress and demands upon government structures. Thus, as ERID prevalence increases and the geographical range of pathogens expands, the number of failing states may rise, necessitating increased humanitarian intervention by U.N. security forces to maintain order in affected regions. As we have seen from recent experiences in Sub-Saharan Africa, the U.N. is unlikely to have a lasting effect in restoring order to areas where ERID incidence and lethality remain high.

---

It is necessary to differentiate between what I will call outbreak events and attrition processes (author’s terms) as these two phenomena may have dissimilar but significant effects at both the unit and systems level. Examples of classic outbreak scenarios are the bubonic plague in Surat, India (Fall 1994) and the ebola epidemic in Zaire (spring/summer 1995). These outbreaks generated fear and panic on a global scale, mass out-migration, military quarantine to contain the exodus of infected persons, and economic damage. Attrition epidemics (HIV, tuberculosis, malaria) do not generate similar levels of fear and out-migration as ‘outbreak’ events, but usually result in greater actual human morbidity and mortality, and significant long-term economic and social erosion. The distinction between these two types of phenomena is important because outbreak events and attrition processes result in somewhat different outcomes depending on the level of fear generated by the pathogen in question.

One preliminary conclusion we can draw from the emergence of V-CJD, ebola, HIV, and plague is that people are extremely risk-averse when it comes to the emergence of new pathogens, and that emergence tends to generate paranoia, hysteria, and xenophobia that may manifest itself in the foreign policy of a state, impairing rational decision-making. The recent epidemic of pneumonic plague (*yersina pestis*) in western India during the fall of 1994 gives an idea of how the psychological effects of infectious disease (in the form of outbreak events) may affect both State Capacity and an afflicted state’s relations with its neighbours. The very rumor of plague in Surat prompted the frenetic exodus of over 300,000 refugees from the city who then carried the pestilence
with them to Bombay, Calcutta, and as far as New Delhi.\textsuperscript{90} Out of fear, Pakistan, Bangladesh, Nepal and China rapidly closed their borders to both trade and travel from India, with some going so far as to restrict mail from the affected state: India had become an instant international pariah. As the plague spread, concern mounted and international travel to, and trade with, India became increasingly restricted. On September 22, 1994 the Bombay stock exchange plunged and soon thereafter many countries began to restrict imports from India, placing impounded goods in quarantine or turning them back altogether at the border.\textsuperscript{91}

As the crisis deepened, the Indian army was called in to enforce a quarantine on the affected area in western India, and doctors who had fled Surat were forced back to work under threat of legal prosecution by the government. In the aftermath of the epidemic that killed 56 people, the Indian government was notified by the Centers for Disease Control in Atlanta that the \textit{yersina pestis} bacillus was an unknown and presumably new strain. This information was interpreted by Indian authorities as ‘unusual’, and they promptly accused rebel militants (Ultras) of procuring the bacillus from a pathogen-manufacturing facility in Almaty, Kazakhstan with the object of manufacturing an epidemic in India. This paranoia on the part of Indian officials resulted in the transfer of the inquest of the epidemic from public health authorities to the Department of Defense.\textsuperscript{92} Beyond the acrimony that the plague fostered between India and its Islamic neighbors, the economic toll of the plague has been estimated at a

\textsuperscript{90} "The Old Enemy," \textit{Economist}, 1 October 1994, p. 40.
minimum of $1.8 billion in lost revenue from exports and tourism. While the loss of $1.8 billion may seem trivial, to a developing state like India it represents a serious blow to the economy with negative repercussions throughout numerous sectors. As we can see in the Surat event and the continuing BSE scare in Europe, infectious disease and the irrational behavior that it generates may worsen relationships between states and/or cultures. For example, the recent panic in Britain over BSE or ‘Mad Cow Disease’ has resulted in the embargo of many beef-derived British products, and dictated the cull of a significant proportion of the U.K’s beef stocks. The BSE scare has frightened the British population as scientists talk about the possibility that thousands of Britons are infected with a new variant of Creutzfeld-Jacob disease (human BSE), and the U.K’s European partners have summarily banned the import of British beef in violation of EU trade law.

Disease emergence must be understood not as a singular isolated ‘event’, but rather as part of biological evolutionary processes taking place at the macro level. The concept of emergence as a process is important because ‘outbreak events’ like the plague in Surat, and ebola in Zaire, are really just ERID manifestations that rise above the lower threshold of our perceptions just long enough to alarm us momentarily. It is best to think of these ‘outbreaks’ as being akin to upward spikes on a stock market graph. While the spikes penetrate the threshold of our perception and then retreat, the process of ERID

---

evolution and emergence continues to grow inexorably. Eventually, ERID emergence, prevalence and lethality may cross a crisis threshold and we will be forced to take serious action to reduce the microbial threat. The only question is, will we still have the ingenuity needed to deal with the problem when we realize the significance and magnitude of the task? At the moment, we have the global wealth and social/technical ingenuity to check the spread of ERID and limit the destruction and misery that most infectious disease causes. Yet dealing with the global proliferation of so many diverse pathogenic agents will require enormous amounts of political will, international cooperation, continued regime consolidation, and a significant redistribution of resources from the developed to the developing world.

Thesis Design

The following is a brief synopsis of the subsequent chapters in the order that they appear. Chapter Two presents a summary of the concepts surrounding the emergence and re-emergence of infectious disease, details those pathogens that are currently making inroads against our best anti-microbial defenses, discusses theories of microbial evolution that are relevant to the study at hand, sets out the research method employed in the study and details the various data sources and collection techniques used in the study. Additionally, this chapter examines the important role that facilitating variables (such as war, famine, ecological destruction, poverty, international migration, and misuse of medical technologies) play as ‘disease amplifiers’ in altering the flow of viral traffic.
Chapter Two lays out the model that I propose to test, to determine whether infectious disease has a measurable effect on state capacity over time.

Chapter Three presents the empirical findings of the study, based on a quantitative analysis of the relationship between ERID and State Capacity at the national, regional, and global levels. This chapter also examines the global correlations between the various individual indicators of State Capacity and ERID, in order to note the strength of the correlations and scale of the effects. Finally, and most importantly, this chapter demonstrates the empirical existence of a feedback loop between population health and state capacity, with population health exhibiting a greater downstream effect on SC than the reverse.

Chapter Four is an analysis of the effects of infectious disease on economic productivity at the state level. This chapter combines empirical epidemiological data with economic indicators, and using basic techniques of statistical analysis, notes the effect of disease on economic productivity. The profound negative effect of ERID on societal prosperity at the individual and macro levels will result in increasing relative and/or absolute deprivation in severely affected countries. The chapter employs process-tracing techniques to track the likely relations between health and development at the microeconomic, sectoral and macroeconomic levels of a given state's economy. This chapter also demonstrates how the proliferation of ERID may compromise the economic development and productivity of the state, generate absolute economic deprivation at the micro level, and increase the economic gap between the wealthy and the poor in severely affected countries.
The conclusion, Chapter Five, examines and analyzes the evidence gathered for this project and presents the findings of the research. It also develops policy recommendations based on these findings, for dissemination to the foreign policy and international development communities. Finally, this chapter delineates pathways for further scientific inquiry into the associations between the biological and political realms.
Chapter Two

Methodological Considerations

A balanced perspective cannot be acquired by studying disciplines in pieces; the consilience among them must be pursued. Such unification will be difficult to achieve. But I think it is inevitable. Intellectually it rings true, and it gratifies impulses that arise from the admirable side of human nature. To the extent that the gaps between the great branches of learning can be narrowed, diversity and depth of knowledge will increase. They will do so because of, not despite, the underlying cohesion achieved. The enterprise is important for yet another reason: It gives purpose to intellect. It promises that order, not chaos, lies beyond the horizon. Inevitably, I think, we will accept the adventure, go there, and find what we need to know.

- E.O. Wilson

Over the centuries, science has been the cornerstone of almost every advance in human well-being, from Hippocrates’ initial inquiries into the nature of contagion and human health, to Darwin’s hypotheses on the evolution of species, to Pasteur’s development of anti-microbial vaccines, and finally to Robert Fogel’s nobel-prize winning thesis that reductions in morbidity and mortality of the British population impelled the industrialization of the United Kingdom.²

---

Recent research has focused on the study of complex relationships between political systems and parameters of state capacity such as environmental change, resource scarcity, population and migration. This chapter discusses methodological principles for the study of complex systems which include health (and specifically infectious disease) as another, heretofore unexamined, principal determinant of state capacity. This study tests the hypothesis that increasing levels of infectious disease (variable X) exert a negative effect on state capacity (variable Y), such that increases in the value of X result in correspondingly diminishing values of Y. Thus, we seek to both understand the causal role that X plays in determining the value of Y, and determine the causal relations between the two variables. We also seek the answer to several questions: (1) Can infectious disease negatively affect state capacity - generating political, economic and social instability? (2) If so, how does infectious disease contribute to political instability and underdevelopment? (3) Is this contribution useful to the discipline?

Edward O. Wilson concedes that the narrow compartmentalization of science over the past two centuries has indeed provided many benefits to society, but he bemoans the modern lack of consilience as detrimental to the greater pursuit of scientific knowledge in the years to come. Consilience is defined as the “jumping together of knowledge as a result of the linking of facts and fact-based theory across disciplines to create a common

---

groundwork of explanation.""¹ Francis Bacon, who took all knowledge to be his province, also recognized the need for the practitioners of divergent scientific disciplines to communicate their findings across the artificial boundaries between the branches of human knowledge.⁵ As the Enlightenment thinkers of Seventeenth and Eighteenth century Europe understood, there is a profound need to seek scientific insight in the form of consilience at the nexus points where the disciplines meet. If we reject consilience we risk continuing the fragmentation of knowledge and the creation of a scientific ‘Tower of Babel’ wherein we are incapable of communication across the disciplines.

This dissertation tests the hypothesis that increasing values for Emerging and Re-emerging Infectious Disease (the independent variable, ERID) impairs State Capacity (the dependent variable, SC) and thus diminishes prospects for state prosperity and stability.

---

¹ Wilson, “Back from Chaos,” p. 41. The term ‘consilience’ was originally coined by William Whewell in his 1840 book The Philosophy of the Inductive Sciences. He wrote “The Consilience of Inductions takes place when an Induction, obtained from one class of facts, coincides with an Induction, obtained from another different class. This Consilience is a test of the truth of the Theory in which it occurs.”

⁵ See Sir Francis Bacon, The Advancement of Learning (1605), Novum Organum (1620), and The Sylva Sylvanum (1626) for his recognition of the need for consilience between the disciplines of scientific thought, and the deficiencies wrought by the specialization and fragmentation of knowledge.
Figure 1: Probabilistic Relations Between Variables
Displays the complex system of linkages between disease pathogens, facilitating variables, and state capacity.

Pathogens existing in Physical Environment

<table>
<thead>
<tr>
<th>Exogenous Inputs</th>
</tr>
</thead>
<tbody>
<tr>
<td>(+)</td>
</tr>
<tr>
<td>(-)</td>
</tr>
</tbody>
</table>

State Adaptation

Intervening Variables (+) ERIDs (-) State Capacity

An explanation of the causal relations detailed in the above chart is appropriate at this point. Pathogenic microbes exist independently throughout the earth's biosphere with the vast majority of them present in the zoonotic pool and outside of the human ecology. In a very real way these pathogens are independent variables and are exogenous to the state as they are truly global phenomena (existing at the system level). These pathogens may cross over from the zoonotic reservoir into the human ecology at any time with emergence being largely governed by the principles of chaos.

After pathogenic agents enter the human ecology (and become endogenized within human societies) their effects are augmented by disease amplifiers (DAs). These DAs generate changes in viral traffic that result in emerging and re-emerging infectious diseases (ERIDs). Thus ERIDs are a product of the synergy between the independent variable (pathogens) and the intervening variables. These ERIDs may in turn have a
pervasive negative impact on state capacity (the dependent variable) which ranges from poverty to social and political instability.

States and societies may at this point use adaptive resources to mitigate the effects of ERIDs on state capacity. The state’s ability to adapt is limited by several factors. First, the initial level of state capacity will determine the scale of adaptive resources that may be mobilized to deal with the ERID problem. States with higher initial capacity will therefore have greater technical, financial and social resources to adapt to crises. Furthermore, state adaptation will also be affected by exogenous inputs of capital, and social and technical ingenuity by actors such as IOs and NGOs. Finally, state adaptation may be compromised by certain outcomes generated by facilitating variables, such as war, famine and ecological destruction.

Exogenous inputs (EIs) take the form of inputs of capital, technology and ingenuity into the state from external sources such as IOs, Foreign Aid, etc. Without exogenous inputs we cannot account for different state adaptation techniques and technologies. EIs such as capital infusions (from the IBRD, etc) also directly affect the resources available to the state when responding to crises, and therefore augment the efficacy of adaptation responses.

There is a positive association between SC and SA because greater initial capacity means that there are more human, economic, and technical resources endogenized within the state that can then be mobilized to deal with various crises. The lower the initial value of SC, the lower the amount of resources that can be mobilized to offset the crisis. This relationship operates in a reciprocal spiral such that greater initial capacity leads to greater adaptive ability, which should in turn reduce the ERID-induced loss to SC. Thus
states that have lower SC when ERIDs afflict them generally suffer much greater SC losses than states with high initial SC. The only means by which states with lower SC can ameliorate the effects of ERID is through exogenous inputs which give low SC states both greater resources to mobilize and advanced tactical knowledge to deal with the crisis.

Although other relations between variables in the model are also important, in-depth examination of these associations is beyond the scope of this dissertation. The relationship between pathogens in the state of nature and intervening variables is not examined for several reasons; (a) pathogens in the state of nature are generally assumed to be static in population size, (b) the potential lethality and transmissibility of these pathogens is unknown until they are affected by intervening variables and become ERIDs, (c) the manner in which exogenous pathogens become endogenized within the human ecology has been extensively documented by epidemiologists. Research on these causal relationships is best left to microbiologists, epidemiologists and public health scientists, and there is no need to deal with it in this limited space.

This dissertation does not test other certain relationships outlined in the causal diagram. Specifically, I do not analyze the relationship between the intervening variables and the dependent variable, outside of the context of infectious disease. Variables such as war, disasters and environmental degradation undoubtedly have an independent and

---

logically negative impact on SC, but these questions fall outside of the purview of this thesis. Similarly, the effects of war, environmental degradation, disasters and migration will undoubtedly have negative effects on SA, but these questions also fall beyond the boundary of the work at hand. Such questions are excellent avenues for further research following the completion of the dissertation. Finally, this dissertation does not test the relationship between exogenous inputs and state capacity. This area is very much in need of elaboration, although the researchers at the State Capacity Project of Peace and Conflict Studies at the University of Toronto have begun to untangle the problem. This is best left to future study in the field.

**The Dependent Variable**

Thomas Homer-Dixon has developed a coherent and comprehensive definition of state capacity that I adopt with minor revisions. He defines the state as “the government, including the center, provincial, and local levels.” Capacity generally refers to power and/or capability. Thus state capacity refers to the ‘capability of government’. I define state capacity (SC) as one country’s ability to maximize its prosperity and stability, to

---

7 There is still much work to be done on each of these issues. Although significant amounts of academic print have been devoted to the question of the effects of environmental scarcity and degradation on state stability and development, there is still a question as to whether this relationship can be demonstrated empirically. Therefore, I shall leave this to those scholars who specialize in the environmental subfield.

exert *de facto* and *de jure* control over its territory, to protect its population from predation, and to adapt to diverse crises.

This definition of state capacity roughly corresponds to Homer-Dixon's multi-dimensional definition of SC which consists of two sets of variables; the first set measures the state's intrinsic characteristics, and a second set measures state-societal relations. These variables are laid out in the table below.

**Table 2.1: Indicators of State Capacity**

This table is taken from the Project on Environmental Scarcities, State Capacity and Civil Violence at the University of Toronto.9

<table>
<thead>
<tr>
<th>Indicators of the State's (or its Components') Intrinsic Characteristics:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Human Capital</strong></td>
<td>The technical and managerial skill level of individuals within the state and its component parts.</td>
</tr>
<tr>
<td><strong>Instrumental Rationality</strong></td>
<td>The ability of state’s components to gather and evaluate information relevant to their interests and to make reasoned decisions maximizing their utility. (Note that “utility” may be locally defined; i.e., it may reflect the narrow interests of the component and not the broader interests of the state or society.)</td>
</tr>
<tr>
<td><strong>Coherence</strong></td>
<td>The degree to which the state’s components agree and act on shared ideological bases, objectives, and methods; also, the ability of these components to communicate and constructively debate ideas, information, and policies among themselves.</td>
</tr>
<tr>
<td><strong>Resilience</strong></td>
<td>The state’s capacity to absorb sudden shocks, to adapt to longer-term changes in socio-economic conditions, and to sustainably resolve societal disputes without catastrophic breakdown. The opposite of “brittleness.”</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indicators of the Relations between the State (or its Components) and Society:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autonomy</strong></td>
<td>The extent to which the state can act independently of external forces, both domestic and international, and coopt those that would alter or constrain its actions.</td>
</tr>
<tr>
<td><strong>Fiscal Resources</strong></td>
<td>The financial capacity of the state or of a given component of the state. This capacity is a function of both current and reasonably feasible revenue streams as well as demands on that revenue.</td>
</tr>
<tr>
<td><strong>Reach and Responsiveness</strong></td>
<td>The degree to which the state is successful in extending its ideology, socio-political structures, and administrative apparatus throughout society (both geographically, and into the socio-economic structures of civil society); the responsiveness of these structures and apparatus to the local needs of the society.</td>
</tr>
<tr>
<td><strong>Legitimacy</strong></td>
<td>The strength of the state's moral authority — the extent to which the populace obeys its commands out of a sense of allegiance and duty, rather than as a result of coercion or economic initiative.</td>
</tr>
</tbody>
</table>

State capacity includes the concept that states are entities that evolve over time.10

This evolution occurs because of the changing factors (parameters) that affect state power.
such as land, resources, population, health, technology, human capital, and prosperity. Thus when we quantify SC we should attempt to measure changes in value on a continuous scale.

For the purposes of this dissertation I have ranked these attributes of SC in order of relative importance: (1) Fiscal Resources (2) Human Capital (3) Reach and Responsiveness (4) Resilience (5) Legitimacy (6) Autonomy (7) Coherence and (8) Instrumental Rationality. This ranking emphasizes the primacy of fungible economic power, and the importance of human capital and adaptive ability in dealing with the problematic transboundary and internal issues of the post-Cold War era, such as global environmental degradation, crime, weapons proliferation, ethnic violence, and pathogen proliferation. While the factors listed above are all captured by the definition of state capacity, I plan to operationalize SC by using a limited set of empirical indicators.

State capacity is the capability of government, and the level of SC determines the state’s ability to satisfy its most important needs: (1) survival; (2) the protection of its citizens from physical harm as a result of internal and external predation; (3) economic prosperity and stability; (4) effective governance; (5) territorial integrity; (6) power projection, and (7) ideological projection. Aside from this normative definition which articulates the dynamic nature and needs of the state, the notion of state capacity should be quantifiable, such that (a) we can determine diachronic variance in the value of SC; (b) we can determine the relative SC of states vis a vis one another; and (c) we can measure

---

10 The author was rapporteur on the ‘Dynamics of Human Well-Being Project’ of the Canadian Institute for Advanced Research. During this project individuals such as Thomas F. Homer-Dixon, Jack Goldstone and I stressed the need to move away from static concepts of state capacity.
11 This ranking reflects my ordering of state needs from survival (most important) to ideological projection.
correlations between quantifiable indicators of SC against other parameters (population, resource scarcity, health, technology, and environmental degradation) which may affect the value of SC.

How can we then quantify SC in a meaningful way? One answer is to develop a core set of cross-national statistical indicators of SC that may then be correlated against diverse independent variables (parameters). To that end this study employs the following set of five statistical indicators for SC and correlates them against two proxy indicators for ERID to determine the empirical associations between SC and infectious disease. All indicators of SC are logically valid measures of the performance of government functions.

(1) Net Long-Term Capital Inflow: (Standardized currency, per capita): measures the influx of economic capital into the state from exogenous sources over time. It is reasonable to assume that rational investors will seek to put their capital into politically stable and economically productive societies, and thus this variable indicates a measure of state stability and prosperity. This indicator also gives an idea as to external perceptions of state stability. This indicator is a logically valid measure of SC, because countries with low SC cannot guarantee a stable investment climate and a decent rate of return. This indicator measures dimensions of state capacity such as fiscal resources, resilience, reach and responsiveness, autonomy, and legitimacy.

(2) Gross National Product, per capita (Current prices, US 1980 $): measures the total value of goods and services produced by the state on an annual basis. The sum is divided into a per capita measure and standardized for current prices. This is a
logically valid measure of SC because high values of this variable require an effective regulatory apparatus. This variable measures dimensions of state capacity such as fiscal resources, autonomy, reach and responsiveness, resilience, human capital, and legitimacy.

(3) Government Expenditure (Standardized currency, per capita): measures the total fiscal outlay of the state on the provision of services (e.g. education, healthcare) to its population on an annual basis. This is a logically valid measure of SC because the more a state spends, the more it is able to generate a revenue stream and it is able to fund a greater number of programs. This variable measures dimensions of state capacity such as reach and responsiveness, legitimacy, resilience, and human capital.

(4) School Enrollment Ratio, secondary (%): measures the percentage of the total population of possible secondary school attendees actually receiving secondary education on an annual basis. This is a logically valid measure of SC because education is a core state function and it is expensive. This variable measures dimensions of state capacity such as human capital, legitimacy, resilience, reach and responsiveness, fiscal strength, and autonomy.

(5) Military Spending per soldier, per capita (Standardized currency): measures the government’s annual fiscal outlay for defense. The aggregate amount is then divided by the number of soldiers in the defense forces, and then the value is adjusted so that it reflects a per capita ratio. The per capita, per soldier ratio allows a relative ranking of the amount spent on the training of soldiers and expenditures on weapons systems. High spending per soldier per capita is an indication of high-tech, capital-intensive, and training-intensive armed forces that can only be created and maintained by states
that possess high levels of state capacity. This is a logically valid measure of SC because only states with high values of SC can afford to fund an efficient high-quality defense.

Taken together this group of SC indicators provides us with a large dataset with values ranging from the early 1950s up until 1991. This allows us to analyze the diachronic associations between variables in order to generate conclusions about the evolutionary path of individual states. It also provides us with enough data so that we can run diachronic correlations to examine the significance of the association between variables. These five SC indicators, the data from 1950 to 1991 and the (20) countries in the sample provide us with a rich set of data points that increase both the significance and certainty of our correlations and the inferences we draw from them. Ultimately, we will be able to run multi-variate regressions controlling for the independent variable (e.g. IM), and an intervening variable (e.g. agricultural production) against an aggregate indicator of SC which is comprised of the five indicators noted above.

Political scientists who advocate interpretivist or post-modern analyses may reject the utility of any attempt to quantify SC, particularly using this set of economic, demographic, and social indicators. However, Jack Goldstone argues that empirical social indicators are important in revealing the nature of societal instability, and that economic and demographic pressures have been the core sources of rebellion and revolution over the centuries.12 This study adopts the perspectives of Goldstone and Homer-Dixon in their attempts to measure the associations between empirical indicators and SC, and beyond that to employ qualitative means to determine the causal linkages between variables. That
said, some dimensions of Homer-Dixon’s model of SC remain difficult to quantify, particularly (1) Instrumental Rationality and (2) Coherence. It may be possible to explore such non-quantifiable dimensions of SC during the case studies that will eventually follow. Perhaps social scientists will need to develop new types of quantitative indicators in order to explore the mathematical associations between these aspects of SC and the various parameters that drive these dependent variables.

The Independent Variable

For the purposes of this study, I provide a specific definition of ERID. Emerging and Re-emerging Infectious Diseases are pathogen-induced human illnesses, that have increased in incidence, lethality, transmissibility, and/or expanded their geographical range since 1973. Specifically, this includes previously unknown pathogenic agents such as Human Immunodeficiency Virus, eschericia coli - 0157 H7, ebola, hantavirus, prions, hepatitis (A through C), and antibiotic resistant pathogens such as vancomycin-resistant enterococci (VRE) and methycillin-resistant staphylococcus aureas (MRSA). Re-emerging diseases are those pathogen-induced human illnesses that were previously controlled or declining in range and/or incidence, but are now expanding in range, incidence, drug-resistance, and increasing transmissibility and/or lethality. Examples of

---

12 This argument is made in Goldstone, Revolution and Rebellion in the Early Modern World.
13 1973 may be viewed as a turning point in the ‘health transition’. Up until the early 1970s, advances in public health had contributed to the dramatic fall in infectious-disease induced morbidity and mortality on a global scale. Thus the prevalence of infectious disease had reached its nadir circa 1973. This year also saw the recognition of a new pathogen ‘rotavirus’, the first of many new pathogenic agents to emerge in the coming decades. Essentially, 1973 is the turning point in the health transition where the curve of infectious disease incidence stops declining and begins its ascension. See Report of the NSTC Committee on International Science, Engineering, and Technology (CISET) Working Group on Emerging and Re-Emerging Infectious Diseases: Global Microbial Threats in the 1990s, (Washington, DC: White House, September 1995).
re-emerging ERIDs are tuberculosis, malaria, cholera, dengue fever, yellow fever, schistosomiasis, rotavirus, adnovirus, and amoebic dysentery. Pathogens are defined as viral, bacterial, parasitic or proteinic organisms or agents that live in a parasitic and debilitating relationship with their human host.

It is important to think of pathogens as exogenous variables, as natural agents that for the most part exist independently of humanity and for all intents and purposes have one central goal, survival. As zoonoses, microbes have historically crossed over from disease pools that exist in animal reservoirs. Human activity frequently alters flows of viral traffic and these novel pathogens may subsequently take root within the human ecology. The following major human pathogens qualify as ERIDs under this particular definition:

Table 2.2: Pathogenic Agents

<table>
<thead>
<tr>
<th>No.</th>
<th>Pathogenic Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Human Immunodeficiency Virus (AIDS)</td>
</tr>
<tr>
<td>2</td>
<td>Mycobacterium tuberculosis (Tuberculosis)</td>
</tr>
<tr>
<td>3</td>
<td>Plasmodium malariae, falciparum, vivax, and ovale (Malaria)</td>
</tr>
<tr>
<td>4</td>
<td>Hepatitis A,B,C viruses</td>
</tr>
<tr>
<td>5</td>
<td>Vibrio cholerae (various subtypes – notably El Tor)</td>
</tr>
<tr>
<td>6</td>
<td>Flavivirus DEN-1,2,3,4 (Dengue fever)</td>
</tr>
<tr>
<td>7</td>
<td>Filoviruses (e.g. Ebola virus)</td>
</tr>
<tr>
<td>8</td>
<td>E-coli 0157:H7</td>
</tr>
<tr>
<td>9</td>
<td>Flaviviridae viruses (Yellow fever)</td>
</tr>
<tr>
<td>10</td>
<td>Phlebovirus bunyaviridae (Rift Valley Fever)</td>
</tr>
<tr>
<td>11</td>
<td>Schistosoma mansoni, haematobium, jarpiculum (Schistosomiasis)</td>
</tr>
<tr>
<td>12</td>
<td>Onchocerca volvulus (River blindness)</td>
</tr>
<tr>
<td>13</td>
<td>Mycobacterium leprae (Leprosy)</td>
</tr>
<tr>
<td>14</td>
<td>Dracunculus medinensis (Guinea Worm)</td>
</tr>
<tr>
<td>15</td>
<td>Hantaviruses (Hantavirus Pulmonary Syndrome)</td>
</tr>
</tbody>
</table>

Zoonoses are microbial or parasitic agents that cross over from the vast reservoir of disease agents that exist in animal reservoirs in a natural state. As these agents cross over from this exogenous reservoir (the zoonotic pool) and enter the human ecology, they may become pathogenic to humanity.
<table>
<thead>
<tr>
<th>No.</th>
<th>Pathogenic Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>Leishmania chagasi (Leishmaniasis)</td>
</tr>
<tr>
<td>17</td>
<td>Shigella dysenteriae, flexneri, boydii, and sonnei (Shigella)</td>
</tr>
<tr>
<td>18</td>
<td>Corynebacterium diphtheriae (Diptheria)</td>
</tr>
<tr>
<td>19</td>
<td>Rotavirus (Severe Diarrhoea)</td>
</tr>
<tr>
<td>20</td>
<td>Respiratory syncytial virus and parainfluenza virus type 3 (ARV)</td>
</tr>
<tr>
<td>21</td>
<td>Adnovirus (Severe Diarrhoea)</td>
</tr>
<tr>
<td>22</td>
<td>Legionella pneumophila (Legionnaires Disease)</td>
</tr>
<tr>
<td>23</td>
<td>Cryptosporidium parvum (Cryptosporidiosis)</td>
</tr>
<tr>
<td>24</td>
<td>Human T-lymphotropic virus (1 and 2)</td>
</tr>
<tr>
<td>25</td>
<td>Toxin producing strains of staphyococcus aureas (i.e. ‘flesh-eating disease’)</td>
</tr>
<tr>
<td>26</td>
<td>Borrelia burgdorferi (Lyme disease)</td>
</tr>
<tr>
<td>27</td>
<td>Prion proteins (Creutzfeld-Jacob disease in humans)</td>
</tr>
<tr>
<td>28</td>
<td>Helicobacter pylori</td>
</tr>
<tr>
<td>29</td>
<td>Enterocytozoon bieneusi</td>
</tr>
<tr>
<td>30</td>
<td>Cyclospora cayetanensis (Cyclosporosis)</td>
</tr>
<tr>
<td>31</td>
<td>Herpesvirus simplex 1 &amp; 2 (Herpes)</td>
</tr>
<tr>
<td>32</td>
<td>Treponema pallidum (Syphilis)</td>
</tr>
<tr>
<td>33</td>
<td>Haemophilus influenzae type B (Meningitis)</td>
</tr>
<tr>
<td>34</td>
<td>Ehrlichia chaffeensis (Ehrlichiosis)</td>
</tr>
<tr>
<td>35</td>
<td>Encephalitozoon hellem</td>
</tr>
<tr>
<td>36</td>
<td>Bacillus anthracis (Anthrax)</td>
</tr>
<tr>
<td>37</td>
<td>Trypanosoma cruzi (Chagas’ disease)</td>
</tr>
<tr>
<td>38</td>
<td>Bartonella henselae</td>
</tr>
<tr>
<td>39</td>
<td>Encephalitozoon cuniculi</td>
</tr>
<tr>
<td>40</td>
<td>Neisseria gonorrhoeae (Gonorhea)</td>
</tr>
<tr>
<td>41</td>
<td>Influenza virus A,B,C (Flu)</td>
</tr>
<tr>
<td>42</td>
<td>Bordetella pertussis (Pertussis)</td>
</tr>
<tr>
<td>43</td>
<td>Borrelia burgdorferi (Lyme disease)</td>
</tr>
<tr>
<td>44</td>
<td>Salmonella typhi (Typhoid)</td>
</tr>
<tr>
<td>45</td>
<td>Yersinia pestis (Plague)</td>
</tr>
<tr>
<td>46</td>
<td>Chlamydia trachomatis (Chlamydia)</td>
</tr>
<tr>
<td>47</td>
<td>Trypanosoma brucei rhodesiense/gambiense (African Sleeping Sickness)</td>
</tr>
<tr>
<td>48</td>
<td>Arenavirae; sub-types Tacaribe, Junin, Machupo, Lassa, Guananito, Sabia (Viral Haemorrhagic Fevers)</td>
</tr>
<tr>
<td>49</td>
<td>Clostridium botulinum (Botulism)</td>
</tr>
<tr>
<td>50</td>
<td>Campylobacter jejuni</td>
</tr>
<tr>
<td>51</td>
<td>Giardia lamblia</td>
</tr>
<tr>
<td>52</td>
<td>Entamoeba histolytica (Amoebic dysentery)</td>
</tr>
<tr>
<td>53</td>
<td>Filarial nematodes (Filarisis)</td>
</tr>
</tbody>
</table>
These various microbes and parasites constitute the majority of human pathogens that generate significant morbidity and/or mortality in human beings. Since I intend to test the effect of these diseases on state capacity I would want to employ detailed state-specific information on national prevalence rates. In an optimal scenario, standardized prevalence rates for each major disease would be available for each and every country, from 1950 to the present day. Unfortunately, such data are unavailable on a global basis due to the many measurement problems detailed below. Since very few states collect comprehensive and composite national (let alone global) pathogen-specific disease prevalence indicators, we must employ proxy indicators that measure the overall burden of disease upon selected states in the sample.¹⁵

This problem is best illustrated by the lack of mortality and morbidity data available in many regions of the developing world. For example, certain countries do not collect malaria prevalence on an annual basis, in fact the WHO has abandoned the collection of plasmodium prevalence statistics for all of Sub-Saharan Africa as rates have gone off the scale, and would therefore skew any statistical correlations using such data. Furthermore, many countries do not report the occurrence of certain diseases such as HIV, due to a lack of political transparency (e.g. China, Burma). To complicate matters further, many diseases carry a social stigma, and physicians are often pressured into

¹⁵ Note that Murray and Lopez's study measures the burden of disease for the year 1990 on an aggregate basis for eight demographic regions of the world. Murray and Lopez employ the concept of disability-adjusted life years (DALYs) to quantify the burden of morbidity and mortality on given demographic regions. Unfortunately, while this study is very useful on many levels it does not provide us with national prevalence data per specific diseases. See Christopher Murray and Alan Lopez, eds., *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: Harvard University Press, 1996). The GBD study follows on Preston's seminal work on global causes of mortality. See, for example, S.H. Preston, N. Keyfitz, and R. Schoen, *Causes of Death: Life Tables for National Populations*, (New York: Seminar Press, 1972).
falsifying diagnoses in order to ‘preserve face’ for the afflicted. The greatest difficulty arises out of the fact that in some countries reports of disease incidence are often sporadic, and often only cover a few of the many diseases that compromise the health of that state’s given population. All told, these problems make it extremely difficult for the analyst to conduct a statistical analysis of the aggregate impact of infectious diseases on the stability and productivity of that given society, (i.e. its state capacity – SC). However, limited incidence and prevalence rates are available for selected diseases in certain industrialized countries. These data sources and their availability are described in the table below.

Table 2.3: Disease Data Availability, Per Pathogen, Per Country
Displays disease incidence by country, years available and data sources.

<table>
<thead>
<tr>
<th>Country</th>
<th>Disease</th>
<th>Data availability</th>
</tr>
</thead>
</table>


Statistics for these diseases are available for the US in Morbidity and Mortality Weekly Report. Stats are given in weekly, and four week totals, according to state and overall national incidence. National prevalence levels are not similarly available for these years. See http://www.cdc.gov/epo/mmwr/mmwr.html and http://www.cste.org.
Disease
HIV/AIDS, Malaria, Hepatitis B,C, Tuberculosis, Cholera, Diptheria, Dengue, Typhoid, Syphilis

Diarrhoeal diseases
Influenza
Measles
HIV

National HIV seroprevalence levels for 1997, 98.

Data availability
Annual national prevalence data available since 1985.18
1990-96
1984-96
1985-95
1989-95
Available in graph format.19
Incidence by province/month 1995-9820
Global for 199721, 98

Given these measurement problems the social scientist needs to employ indicators that serve as comprehensive proxies to measure the burden of disease-induced morbidity and mortality on societies. Such an indicator must be highly sensitive to the societal burden of disease, it must be standardized across states, it must be available for most countries over a broad span of time, and it must be comprehensive (such that it reflects mortality associated with the most prevalent pathogens in a given society). Given these requirements, the most valuable comprehensive empirical indicators are (1) Infant

---

19 See http://www.b3c.jussieu.fr/sentiweb. Note that exact data values are not given.
21 Current estimates of overall HIV prevalence at the national level for 1997 are regarded as being reasonably accurate, and therefore useful to this type of analysis. See http://www.unaids.org/highband/document/epidemio/june1998/fact_sheets/pdfs/botswana.pdf.
Mortality rates per 1000 children, and (2) Life Expectancy at birth (years). Data for these
two proxy indicators are available for the vast majority of countries over the 1950-1991
time period.

Infant Mortality (IM) is arguably the best indicator for measuring the aggregate
burden of disease on a population, as it incorporates mortality from every disease
pathogen, including malaria, tuberculosis, measles, syncytial respiratory viruses and
diarrhoeal rota and adnoviruses. Infant Mortality measures the impact of infectious
disease on the first tail of the demographic distribution of a given population, children
from 0 to 5 years of age. With the notable exception of HIV/AIDS, IM is the best
indicator for measuring the burden of disease across divergent societies because the
majority of disease-induced mortality on a global level shows up in the 0-5 year age
sector of the demographic curve. In layman’s terms this means that the vast majority of
human beings killed by infectious diseases are children under six years of age. Indeed,
Murray and Lopez demonstrate that over 70 per cent of global infant mortality is
attributable to microbial and/or parasitic infection. Therefore, fluctuations in IM over
time typically result from the changing prevalence and lethality of infectious diseases
within specified populations.

Nowhere in the published literature will one locate a statement that specifically
states that IM is a good proxy measure for a comprehensive snapshot of the burden of

\[\text{22 There is a significant differential in IM between developed countries and the rest of the world, as}
\text{industrialized states have far lower infant mortality rates than the global average. Nonetheless, the greatest}
\text{proportion of mortality is present within this cohort of the demographic distribution and thus IM is}
\text{extremely sensitive to changing prevalence and lethality of the spectrum of infectious diseases within the}
\text{human ecology. See Murray and Lopez, Global Burden of Disease, pp. 1128-33. The notion of IM as a}
\text{good proxy measure for ERID was reinforced through the author’s numerous conversations with Stephen}
\text{S. Morse.}\]
infectious diseases on societies. The reason for this is that medical scientists (a) usually study a single disease pathogen, and (b) have never thought about the relationship between disease and state capacity. In other words, they have never had to argue that IM is a good comprehensive indicator, although most medical scientists I have spoken with agree that this is an empirically valid claim.\(^{23}\)

Murray and Lopez quantify the aggregate burden of disease by including both long-term and sporadic morbidity, and disease-induced mortality. They use the measurement tool of the disability-adjusted life year (DALY) in order to look at the true impact of different diseases, injuries, and risk factors upon affected populations.

DALYs provide a common metric to aid meaningful comparison of the burden of risk factors, diseases, and injuries. ... the primary indicator used to summarize the burden of premature mortality and disability (including temporary disability) is the disability-adjusted life year (DALY). DALYs are the sum of life years lost due to premature mortality and years lived with disability adjusted for severity.\(^{24}\)

Murray and Lopez demonstrate that the top two contributors to the (global) burden of disease are communicable diseases affecting children, namely lower respiratory infections (LRIs) and diarrhoeal diseases primarily caused by the adno and rotaviruses and amoe bic agents. Tuberculosis, measles, malaria, and pertussis also came in as the 7\(^{th}\), 8\(^{th}\), 11\(^{th}\), and 23\(^{rd}\) greatest single contributors to global death and disability, and all of these illnesses are found at relatively higher levels in the youngest tail of the population curve, namely the 0-5 year age group. Murray and Lopez note that infectious disease constitutes the single greatest burden on human populations relative to all other causes of

---

\(^{23}\) Thanks in particular to Prof. Stephen S. Morse of the Columbia School of Public health for his support on this issue.

death: "...the three leading contributors to the burden of disease are lower respiratory infections, diarrhoeal diseases, and perinatal disorders. Together with measles, the eighth largest cause of burden, these childhood diseases account for 25% of the whole burden of premature mortality and disability."25

Optimally, at some future date disease-specific DALY measurements may be available for all states over a significant period of time. Such data would be of immense value to this type of project. However, until this information is available we must use proxy indicators that are extremely sensitive to the burden of disease. Thus IM’s sensitivity to the comprehensive societal burden of disease makes it the best available indicator for measuring the effects of ERID on SC over broad stretches of time, and across diverse cultures and societies.26

However, IM will not include the burden of certain pathogens (such as HIV) that predominantly affect the central part of the demographic distribution curve, namely those adults in the 15-45 year age range. Life Expectancy (LX) measures the total burden of disease on a specified population, covering the complete demographic curve including both tails of the population distribution. Unfortunately, the mortality shown under LX does not replicate IM’s extreme sensitivity to infectious disease, as it includes mortality resulting from accidents, suicides, and violence. However, rapid increases in the

---

25 Note these infections account for 25 per cent of the total global DALYs lost for all populations including all age groups. Ibid, p. 1441.
26 Additionally, IM data are useful because they are usually unavailable during years of warfare or extreme conflict. Thus, IM data are generally resistant to statistical distortion that may be induced by the skewing effects of war or intense internal conflict. During years of intense conflict within a given state, the data for that dyadic statistical year will either be unavailable – or will be adjusted to mirror a standard using statistical models based on data from previous years. Life expectancy data are frequently available during years of conflict and thus logically more sensitive to the skewing effect of war. See Althea Hill. "Trends in
prevalence of HIV within a given society will show up only in the 15-45 year portion of the demographic distribution. Thus, while the effects of the HIV pandemic on national productivity and stability are unlikely to show up in IM, they may be observed through the use of LX.

From independence in 1980 and for nearly a decade thereafter, Zimbabwe made stunning health advances. But AIDS has already erased all the life expectancy gains made since then. Further, if the worst projections come to pass, by about 2010 life expectancy will return virtually to where it stood the day I was born, in what was then Southern Rhodesia, half a century ago.27

Preston’s detailed international statistical analyses of the major causes of mortality decline are valuable in determining the major causes of death over time. Preston expanded on previous work done with Keyfitz and Schoen28 to examine the relative importance of various causes of death using data for 165 populations from different countries and across various time periods.29 Preston found that at least 60 per cent of global mortality over the 20th century was in fact attributable to infectious disease. Vallin notes that Preston’s estimates of the influence of disease on mortality were on the low side, as the remaining 40 per cent of mortality was attributed to ill-defined causes, and did not include pathogen-induced cancers. Vallin et al, also attribute considerable weight in diachronic measures of global mortality to infectious diseases, and provide evidence that reinforces Preston’s conclusions.30 Of course, variation will occur in the causes of

---

29 See Preston, Mortality Patterns in National Population.
death between different populations and over time, but diachronic and randomized statistical studies such as this should minimize those possible skewing effects. LX is therefore required as a supplementary indicator to give us an accurate picture of the effects of the HIV pandemic.

Recent medical advances have shown the central role that some pathogens play in inducing many forms of human cancer. "Up to 84% of cases of some cancers are attributable to viruses, parasites or bacteria. WHO estimates that over 1.5 million (15% of the new cases occurring each year) could be avoided by preventing the infectious disease associated with them. About 1.2 million of cancer cases (20%) in developing countries and 363,000 (9%) in developed countries are attributable to infectious agents." These cancers include stomach cancers (Helicobacter pylori), cervical cancers (human papilloma virus), liver cancers (hepatitis B and C), AIDS-related cancers (numerous pathogens), Burkitt’s lymphoma (Epstein-Barr virus), Hodgkin’s disease (Epstein-Barr virus), and bladder cancer (schistosomiasis). Ergo, LX compliments IM because it measures mortality resulting from both the global HIV pandemic and pathogen-induced cancers.

Life Expectancy displays an inverse statistical association with Infant Mortality, such that there is a significant negative correlation (of -.935) between IM and LX for the twenty country sample over the 1950 to 1991 period of the analysis. Thus, we can state with assurance that IM and LX are generally ‘mirror proxies’ which both measure the

---


burden of disease on populations but have an inverse relation to one another. This is useful because these indicators allow us to analyze the burden of disease on the complete demographic distribution of the population within a given state. IM and LX also provide us with a comprehensive snapshot of the burden of disease over a relatively broad span of time and reflect the decline over time in morbidity and mortality as measured since the early 1950s, which can then be measured against changes in state capacity over the same time period.

One caveat to using IM and LX as proxy indicators for ERID is that while they give us an excellent idea of ERID-induced mortality over the decades, they only give us indirect knowledge of the morbidity associated with ERID over the same time period. For example, malaria-induced mortality will show up in IM, but we can only guess as to the ratio of individuals killed as to the proportion of the population that is debilitated. This also varies according to the lethality of the disease, as malaria generally debilitates far more people than it kills (low-moderate lethality), while HIV generally debilitates and kills those who it affects (high lethality). Regrettably, the majority of the global population of states do not have the ability to accurately track disease-induced morbidity within their populations. Therefore, I cannot employ the 20 country sample to ascertain the impact of specific disease-induced morbidity on SC. At this point in time, only a very small sub-set of industrialized nations (Canada, US, UK, Australia) keep limited statistics on productivity lost to diseases such as HIV. Obviously, studies such as the Global Burden of Disease which measure morbidity and mortality-generated DALY’s will be of enormous value if differentiation of pathogen weight by country per year is included in future editions. Notably, the WHO has begun to publish reasonably accurate statistics on
HIV seroprevalence rates within national populations for most countries of the world for the year 1997.\textsuperscript{33}

Theories of Causation

There is... a good deal of evidence that bacteria became capable of producing infections millions of years ago, and there is no reason to doubt that man from the very beginning suffered from infectious disease; and at the time when mankind had reached the period of the earliest historical records, infectious diseases of many varieties already existed...\textsuperscript{34}

Microbial pathogens evolved from the primordial soup of life millions of years ago, along with other single-celled creatures, and thus have existed far longer than human societies (much less human societies), preying on all manner of flora and fauna over the eons.\textsuperscript{35} Thus, pathogens predate humanity, tend to exist independently of humanity in nature, and will continue to exist whether the human species endures or not. Therefore, pathogens should be correctly seen as independent variables that can be affected by human actions (facilitating variables) that may alter microbial transmissibility and lethality.

There has been some debate regarding the lines of causation concerning ERIDs and their effects on state capacity. The principal objection voiced is that infectious disease is in fact \textit{endogenous} and therefore caused by pre-existing human-induced

\textsuperscript{33} These data are available or 1997 at \url{http://www.unaids.org} under the epidemiological country fact sheet listings. The annual number of HIV diagnoses are generally available per country per year, however, WHO advises that these incidence rankings are fraught with significant error. The best available data are the national seroprevalence levels for 1997 which are based upon new surveillance mechanisms and methodologies.

\textsuperscript{34} \textit{Zinsser, Rats, Lice, and History}, pp. 106.

\textsuperscript{35} Ibid, p. 105.
conditions such as poverty, war, famine, environmental degradation, etc. The fact of the matter is that these social conditions are actually *intervening variables* that (depending on their individual nature) may increase both the transmission capacity and lethality of pathogenic agents within affected regions. However, the argument that these conditions actually *create* the pathogens in question is incorrect. There is significant archeo-epidemiological evidence that infectious pathogens antedated the arrival of humans (and multi-cellular life in general) and their rapid and unpredictable evolution is guided to a large degree by chaos and occasionally accelerated by human actions.

The concept of pathogen emergence is critical to this project as new disease agents tend to exhibit the greatest virulence when first introduced to immunologically naive populations. To paraphrase Morse and Schluederberg 'emerging' pathogens refer to disease agents that either have recently appeared in the population or are rapidly expanding their range. Morse argues that known disease agents "...are only a fraction of the total number that exist in nature." Furthermore, 'newly evolved' disease agents are most often the descendants of parent strains that already exist, this is a function of Darwinian evolution through processes of natural selection. "Given these constraints of organic evolution, then, there are fundamentally three sources (which are not necessarily mutually exclusive): (1) evolution of a virus *de novo* (usually the evolution of a new viral

---

36 I refer here to comments made by PRIO researchers during a seminar I gave in November 1996 at the International Peace Research Institute, Oslo.
37 The dynamics of chaos seem to govern the likelihood of pathogenic agents swapping genetic information through a process called 'antigenic shift', which frequently alters the genetic code of the receiving microorganism and may change its lethality and/or transmissibility. For an excellent discourse on the discipline of chaos see James Gleick, *Chaos: Making a New Science.* (New York: Penguin Books, 1988).
39 Ibid. p. 16.
variant); (2) introduction of an existing virus from another species; (3) dissemination of a virus from a smaller population in which the virus might have arisen or originally been introduced."$^{40}$ Similar processes also hold for bacteria, parasites and possibly infectious proteins (prions).

However, according to Morse, pathogen evolution is not the most significant driver behind the emergence of 'new' infectious diseases. "(O)ver the period of recorded history... 'emerging viruses' have usually not been newly evolved viruses. Rather, they are existing viruses conquering new territory. The overwhelming majority are viruses already existing in nature that simply gain access to new host populations."$^{41}$ These pathogens exist in nature in disease 'reservoirs' and may jump the species barrier to humanity from the "zoonotic pool" (i.e. the vast plethora of diseases that pervade all niches of life in the biosphere). While the chance that any one particular 'zoonosis' is pathogenic to humans is relatively low, the sheer magnitude of infectious agents that exist in the zoonotic pool makes the 'emergence' of human pathogens more likely. Morse coined the term viral traffic to demonstrate how infectious agents move between different species and individuals, and he argues that the majority of outbreaks of 'new' diseases tend to be the result of changes in patterns of viral traffic. Viral traffic is altered by changes in the ecological, economic, and social environment, which I refer to as facilitating variables, in that they may exacerbate both the lethality and transmission of ERIDs, and thereby intensify the negative effects of ERID on state capacity.

$^{40}$ Ibid, p. 12.
$^{41}$ Ibid, p. 15.
Intervening Variables

The spread of [leishmaniasis] is accelerated by development programs such as road building, dam construction, mining and forest exploitation that bring increasing numbers of people into contact with the disease vectors. Another factor enhancing spread is the haphazard growth of major urban centers which creates conditions that increase transmission risks. A third factor is the movement between countries or regions of migrant workers who themselves act as vehicles for the disease.\(^{42}\)

It is important to keep in mind that the effects of ERID on SC are distinctly non-linear in nature, as ERIDs are subject to intervening variables, such as ecological disruption, increased human mobility, poverty, technology, war, and famine. These factors frequently alter the flow of viral traffic and thus produce, and affect the course of, epidemics and pandemics. In this way these intervening variables act as disease amplifiers. Pathogenic virulence and transmissability are augmented by these disease amplifiers thereby generating epidemic and/or pandemic disease. These facilitating variables generally magnify and exacerbate the ERID threat, but it is important to understand the dynamics between ERID and these facilitating variables as they frequently influence each other in a complex web of mutual and non-linear interactions.\(^{43}\) These interactions require the fulfillment of necessary conditions which taken together are jointly sufficient to produce ERID. These facilitating variables are briefly listed in order of relative importance.

Ecological Disruption

Environmental destruction releases new pathogens into the human ecology as the disruption of habitat brings humans into contact with new microbial agents from the zoonotic pool. Additionally, overpopulation, urbanization and climate change all aid in pathogen transmission and make the human population increasingly vulnerable. Climate change increases the range of pathogen vectors and environmental degradation fuels the rapid evolution and dissemination of microorganisms.44 Large-scale water development projects, particularly irrigation systems that augment agricultural productivity, have contributed to the diffusion of schistosomiasis (blood flukes) to formerly unaffected regions.45 Furthermore, ozone depletion compromises the immune systems of animals (including the human species) making it far easier for microorganisms to colonize new hosts, while increasing radiation levels may accelerate the rate of pathogenic evolution.

Migration

International and intra-state migration is playing a significant role in the global diffusion of pathogens as travelers to and from previously isolated regions may distribute previously contained microorganisms into the global population, many of whom will be immunologically naïve to the emerging infectious agent. Furthermore, travelers from the developed world bring pathogens from their sojourns abroad back into their home countries where these agents may eventually take hold within that new population. Rapid

44 Lederberg, Shope and Oaks, Jr., eds., Emerging Infections: Microbial Threats to Health in the United States, p. 43.
45 "The construction of dams in three African river deltas - the Nile, the Senegal and the Volta - has led in recent years to schistosomiasis infection in up to 75% of local villagers, and even among people as far as 500 kilometers upstream of the dams." World Health Report 1996, p. 39.
advances in transportation technologies (the ship, railway, car, airplane) have accelerated this process of global pathogen diffusion, and the profusion of international travelers for both recreational and business purposes is bound to exacerbate the problem of ERID dissemination in the coming decades.\textsuperscript{46}

\textit{Trade}

Throughout history, trade has been implicated in the diffusion of pathogens on a global scale. For example, both flavidirae viruses (e.g. Yellow Fever) and their principal vectors (\textit{Aedes aegypti} mosquitoes) were transmitted to the Americas from Africa courtesy of the slave trade. The mosquito vectors fed on the blood of infected slaves during the transit and then spread the contagion throughout the New World.\textsuperscript{47}

Additionally, the Pan American Health Organization believes that the recent transmission of El Tor cholera to South America was facilitated by a Chinese freighter which jettisoned its contaminated bilge water into a Peruvian harbor, after which the disease spread through seafood products and tainted regional water supplies.\textsuperscript{48} Additionally, infected foodstuffs and livestock transported across borders have resulted in the dissemination of the BSE prion into beef cattle in France, Germany, and Switzerland. Infected berries (cyclospora) from Guatemala were also recently implicated in a large outbreak of diarrhoeal disease throughout North America during the summer of 1996.

\textsuperscript{47} Ibid. p. 19.
**Human Ecology**

The actions of individuals within a society, and societal habits at large can also influence the course of viral traffic and lead to the emergence and re-emergence of infectious disease, both regionally and globally. For example, the annual pilgrimage to Mecca by Islamic peoples is generally associated with the proliferation of cholera throughout the population of travelers, who then bring the bacilli back home to their own countries. Other modes of behavior, particularly sexual activity/promiscuity and the use of illicit narcotics, assist in the diffusion of many disease agents. Furthermore, the burgeoning magnitude, density, and distribution of human populations also facilitates the dissemination of pathogens, particularly since once population levels reach a new threshold then ‘disease pools’ within those populations become large enough to sustain new infections.49

**Misuse of Antimicrobial Drugs**

The consistent misuse of antimicrobial drugs within both the developed and developing world has resulted in the emergence of drug-resistant strains of parasites, bacteria, and viruses. For example, the Thai-Burmese border region is practically uninhabitable due to the recent spread of drug-resistant strains of malaria throughout the region. Meanwhile in the developed world bacterial strains such as Vancomycin-resistant enterococci (VRE) and Methycillin-resistant staphylococcus aureus (MRSA) are plaguing our hospitals, and multi-drug resistant tuberculosis (MDRTB) is spreading

---

through the marginalized portion of the population. The problem stems from the fact that organisms develop drug resistance through evolutionary pressures when the pathogens in question are exposed to antimicrobial drugs. These antimicrobial agents kill susceptible bacteria which in turn generates evolutionary pressures on those members of the species that possess a gene that provides resistance to that particular drug. These resistant microbes then expand their population to fill the ecological niches of other pathogens that were eradicated by the same antimicrobial agents. While physicians only use the current drug of last resort (vancomycin) extremely sparingly, tremendous amounts of similar drugs are distributed through domestic animal feed, which in turn results in the spread of resistant bacteria throughout the animal world. These resistant pathogens may then cross the species barrier to cause zoonoses in human populations.

Disasters

In addition to the above facilitating variables, both natural and human-induced disasters (e.g. earthquake, flood, war, famine) may also affect viral traffic in a manner that leads to disease amplification through increased transmission and/or lethality of the infectious agents, and which may result in epidemics and even pandemics. The single greatest case of this occurred during 1918-19 when the global movements of armies served as vectors for the distribution of influenza and typhus. The resulting 'Spanish Flu' pandemic claimed an estimated 20 million lives, while the typhus pandemic also resulted

\[^{50}\textit{World Health Report 1996}, \text{p. 18.}\]
in almost 20 million deaths, dwarfing the mortality caused by military action during the war itself.\textsuperscript{51}

Similarly, the breakdown of food distribution within a region which results in famine will also deplete the health of a population, such that infectious agents may have an easier time colonizing their hosts and may cause greater morbidity and mortality in the weakened population, as it takes the weakened host a longer time to mount an effective immune response to the invading pathogens. The greatest historical example of this synergy between famine and disease is the Great Hunger which struck Ireland (and the rest of Europe) in 1845. This catastrophe was generated by a fungus (\textit{phytophthora infestans}) which destroyed the potato crops of Europe, caused massive starvation and governance problems in Ireland, and led to terrible outbreaks of typhus and cholera in affected regions, which were subsequently carried overseas to North America and Australia via infected immigrants who fled the devastation in the Old World.\textsuperscript{52}

Data

The data I use in this analysis is taken from a random global sample of 20 countries. The countries are Botswana, Brazil, Colombia, Ethiopia, Haiti, Iceland, India, Italy, Japan, Kenya, Malawi, Netherlands, Norway, Peru, Rwanda, Saudi Arabia, South Africa, Tanzania, Thailand, and Uganda. The country data used for the statistical analysis is drawn from the World Bank Statistical Tables dataset, the WHO’s World Health Reports, and the UNAIDS statistical country fact sheets. Primary and secondary source

\textsuperscript{51} Morse, “Origins of Emerging Viruses”, p. 10.
epidemiological and microbiological data was obtained from the Population and International Health, and Countway Libraries at the Harvard School of Public Health. I have also used the ProMed global disease surveillance system, the US Bureau of the Census HIV Surveillance sentinel site database, the World Health Reports, Morbidity and Mortality Weekly Reports, journals such as Science, Nature, the Economist, the Journal of the American Medical Association, New England Journal of Medicine, Lancet, Emerging Infectious Diseases and numerous health-related internet sites as core data sources.

For the purposes of the quantitative analysis I employ standardized diachronic global indicators of SC such as GNP per capita, school enrollment, etc. These measures run from circa 1960 to 1991 and are all obtained from standardized World Bank data. IM and LX data is generally available for the full sample from 1952 to 1991.

The data I use allow for the variation of both the dependent and independent variables while allowing for some control over potentially confounding variables. In order to avoid selection bias in the analysis I have randomly selected cases hoping for significant variance in the independent variable between the countries in the sample. Thus, I compare indicators from highly developed temperate states such as Iceland (low values of ERID intensity) with those of tropical countries like Rwanda (high values for ERID intensity). This eliminates the possible bias that would result from selecting only developing countries with low state capacity.

52 Karlen, pp. 118-121.
53 See (1) http://www.cdc.gov (2) http://who.org (3) and Emerging Infectious Diseases at http://cdc.gov/ncidod/eid.
Randomization of the selection process is key to bias reduction. Before randomization, certain states had to be selected out of the population for inclusion in the sample because they could not satisfy the minimum data requirements. Countries such as Sierra Leone and Liberia that lacked a minimum standard of data for the selected indicators were excluded from the sample as their inclusion would have been of low utility. The sample was drawn randomly from the remainder of countries that met the minimum data requirements for the relevant indicators, even if there were occasionally significant gaps in the annual data. This randomization was generally successful, as the countries in the sample represent all climatic regions of the world, all levels of development, and most continents. This random selection of the sample should suffice to reduce the probability of bias to a reasonable minimum in this study. Furthermore, the sample size (N of 20) is also sufficient to do a good job in terms of obtaining a ‘snapshot’ of the correlations between the variables on a comparative cross-national basis.

Despite the advances of modern ERID surveillance technologies there remain data problems that other scientists must consider and circumvent, if possible. First, a lack of transparency often hinders the collection of accurate field data and the dissemination of accurate statistics within the country in question. As well, political barriers may rise in order to hide the true state of affairs (i.e. the massive debilitation of the population by a stigmatized disease such as HIV). This was commonplace with HIV prevalence in Sub-Saharan Africa in the 1980s, and throughout South and East Asia in the 1990s. The intrastate dissemination of accurate statistics is also problematic because of the lack of technological diagnostic and communication infrastructure, and manpower, in many of the rural hospitals. Furthermore, regional authorities may have interest in exaggerating
the infectious disease situation in order to receive greater amounts of aid, or conversely
downplaying the gravity of the situation to avoid unfavorable reviews from their
superiors. This political manipulation and suppression of accurate statistics makes it
difficult to get accurate disease-specific data out of many states, particularly those with
authoritarian regimes (such as the former Zaire, Nigeria, China, Myanmar). Democracies
that possess greater political transparency allow for improved data collection capacity
from those nations.

At the systems level, data collection capacity is improving as nascent surveillance
regimes (such as ProMED) report daily outbreaks and occurrences via electronic media.
As mentioned above, the World Health Organization and the US Centers for Disease
Control issue quarterly reports on prevalence levels of certain notifiable infectious
pathogens, and weekly updates such as Morbidity and Mortality Weekly Report offer
tallies of disease incidence within the US population. However, for the majority of the
population of states the data are marred by certain inaccuracies at this level as well. First
of all, it is very hard to obtain accurate data on the incidence of and/or prevalence levels
for certain pathogens (Hepatitis) within specified national populations (Sierra Leone).
Sentinel data are available for selected diseases in selected communities on various dates,
but it is difficult at best to derive national seroprevalence levels from these scattered
studies.\textsuperscript{54} Secondly, in some cases the agencies that are expected to monitor prevalence
levels have simply bowed to the enormous prevalence levels in certain regions and

\textsuperscript{54} The global set of HIV national seroprevalence levels for 1997 was recently published online and
represents the first opportunity for scientists to correlate HIV prevalence with State Capacity.
Unfortunately, these prevalence statistics are only available for 1997, but they permit some preliminary
evaluation of AIDS effect on human development at the global scale.
stopped collecting data on selected pathogens. This is the case in Sub-Saharan Africa where the WHO has admitted that it no longer has the capacity to monitor the prevalence of malaria in the region, and that rates are simply off the scale. Finally, there are occasional scientific inconsistencies in the data collection, interpretation and dissemination processes from the various reporting sites to the WHO. While WHO attempts to harmonize the data as much as possible, it is likely that some inaccuracies will remain in the data. As local, regional, state, and WHO infrastructure improves over time the data will as well.

Given that statistics cannot tell us everything we need to know about the relations between variables (i.e. causation), I employ qualitative data during process-tracing analysis to fill the theoretical gaps in the model. For the process-tracing I employ extensive primary source epidemiological and microbiological data obtained from the Population and International Health and Countway Libraries at the Harvard School of Public Health. I have also used the ProMed global disease surveillance system, the US Bureau of the Census HIV Surveillance sentinel site database, the World Health Reports, Morbidity and Mortality Weekly Reports, journals such as Science, Nature, the Economist, the Journal of the American Medical Association, New England Journal of Medicine, Lancet, Emerging Infectious Diseases and numerous health-related internet sites as core data sources. Additional material is derived from US government documents such as NSTC-7, USAID, and speeches made by Secretary of Health and Human Services, Donna Shalala. Moreover, primary source documents from the WHO, PAHO and CDC have been useful, while secondary source documents from the World Bank, UNDP-AIDS, and USAID are also used.
Despite certain deficiencies noted above it is still possible to derive generalizable and empirically testable scientific hypotheses from the data. To ensure that our conclusions are simultaneously demonstrable and accurate, I employ statistical data analyses. Statistical analysis of empirical data provides the correlations which confirm or disconfirm the various hypotheses, and allow us to discriminate between potentially important causal linkages and ones that are marginal to the subject at hand. Quantitative techniques can tell us whether there are any potentially causal relationships.

I engage in a bi-variate statistical analysis using Pearson’s correlation coefficient to test the strength of the hypothesized relationships between the variables. The correlations that we derive from these tests tell us the strength of the association between two variables between the values of 1 and -1, and the significance of the association given the size of our sample. Significance tells us that we have a real and important relationship between the variables, and that we can generalize these findings to hold across the total population.

This study employs t-tests (test of statistical significance) on Pearson’s $r$ (the correlation) which tells us whether the correlation differs significantly from 0. To demonstrate that IM (as a proxy) predicts SC we need to use Ordinary Least Squares multiple regression analysis. The above tests can either support or refute the respective hypothesis within a 95% confidence interval. The margin of error based on sample size is $+/10 \%$.

There are several statistical techniques that are not used for various reasons. I do not employ log-linear, logistic regression because these techniques are used for categorical data. Cluster analysis is also inappropriate because we have no respondents,
so there is nothing to cluster. Factor analysis is also not used because we have nothing to
reference the SC variable against, and you would need at least 10 years of data per
variable to do the job. Therefore, the five variables within the computed variable of SC
would require a minimum of 50 years of available data to work with. Having less than the
required 10 years per variable makes the reliability of the factor analysis suspect. We
cannot yet test for validity because this requires factor analysis. Reliability tests indicate
how reliable the measure is as a computed variable by measuring the internal consistency
of observed variables vis a vis one another and the overall computed variable. Regardless
of the sample size we then require an alpha of at least 0.6 in order to say that there is a
strong inter-correlation between the observed variables that taken together comprise the
computed SC variable. Following the computation of the alpha (0.64) we can attest to the
firm inter-correlation between the five SC indicators that comprise the aggregate SC
measure.

These statistical analytic processes can tell us a lot, but they cannot firmly specify
the nature of the causal relations within the model. Initially, I correlate the independent
and dependent variables for the entire 1950 to 1991 time period. I then lag the variables
to see if the strength of the correlation changes downstream. Theoretically, disease-
induced mortality and morbidity are likely to impair state capacity, but this effect is not
likely to be immediate. For example, following the colonization of the human host,
pathogens often take differential amounts of time to generate disease within that given
host. Thus, the debilitating effects of certain diseases with long germinating periods (such
as HIV/AIDS and Hepatitis B, C) will logically grow stronger with the passage of time.
Therefore, we lag the variables to see if the downstream effect of ERIDs on SC grows or diminishes with the passage of time.

ERIDs pronounced negative effect on child life-expectancy will reduce the downstream availability of healthy and capable workers available to a given society. By lagging the variables this study analyzes the import of the differential time lag between increasing ERID values and state capacity outcomes. This will help to predict the downstream economic and political effects of rising ERID levels, help us to formulate more effective policy measures to deal with the ERID problem, and help us to predict downstream state capacity based on current population health indicators.

Given that we cannot currently run a factor analysis on the relationships between the variables due to the unavailability of quantitative data we need to explore the question of causality using available non-quantifiable data. Process-tracing case studies allow us to distinguish spurious correlations from probably-causal relationships, and can help us get a handle on certain interactions that are difficult to correlate because of operationalization and measurement problems. Mapping the complex threads of causation between the independent, intervening, and dependent variables illuminates the probable causal connections between them. For example, while statistical data analysis may demonstrate a high correlation between the burden of disease on a society, and its levels of productivity, the scientist must utilize process-tracing techniques to determine the causal linkages between the variables and to determine which variable is driving the other.
Falsifiability

As stated above, we are testing the hypothesis that increasing infectious disease prevalence diminishes state capacity. The hypothesis is easily falsifiable if (a) we find that increasing levels of disease do not correspond to declining state capacity, or conversely, (b) if falling infectious disease rates do not correlate with increasing state capacity. Since most of the data available comes from the most successful anti-microbial era in human history (i.e. 1950 to 1991), we can empirically test the hypothesis by looking at the impact that the decline of disease-induced morbidity and mortality had on state capacity in the post World War II era. If we find that the inverse relationship holds between proxy indicators for both disease prevalence and state capacity, then we can generalize that the negative association between ERID and SC will also hold over time, and across geographical regions. Thus, if the hypothesis is correct we can argue that the expanding pandemics of HIV/AIDS, tuberculosis, malaria, and hepatitis (to name just a few) will have negative implications for state stability and development in the future.

If I find what I expect to find during this analysis, I will have established the importance of public health as a major determinant of downstream state capacity. Given the theorized empirical centrality of ERID as a stressor on SC, any major threat to population health thereby jeopardizes state prosperity, governance and survival. Thus infectious disease can be seen as a major contributing factor in the breakdown of governance, poverty, and state failure in affected regions. Conversely, and of equal importance, declining disease rates should therefore lead to greater state capacity and by extension greater prosperity, stability and power in healthier states.
The present analysis of the impact of infectious disease on the populations of states and its resultant effect on state prosperity and stability obviously has broad ramifications for the majority of (if not all) human societies. If we can understand the relationship between rising levels of ERIDs and the associated decline in prosperity and stability of states and societies, then we gain the ability to address the break points in the chain of causation in order to formulate more effective policies for the surveillance and containment of infectious disease. We may also gain some ability to predict future events and processes that may be detrimental to a given state, such as disease related socio-economic decline, insurrection, rebellion and (in extreme cases) state failure.

This dissertation illuminates a significant parameter of state capacity, population health. A parameter is defined as one class of phenomena that exerts a general effect on another dependent phenomena. In this sense, broadly defined constructs such as population, environment, poverty, and now health may generate significant positive or negative effects on state capacity. The central concern of any study of parameters of state capacity is to determine the importance of each parameter relative to the others, and what effects they exert on state capacity. Can we empirically demonstrate that health is a more significant parameter than environmental degradation, for example? Hopefully the answer will be in the affirmative. However, the question of determining the relative weight of different parameters will have to follow the completion of this project, and bias-free systematic studies of the other parameters.
Chapter Three

Statistical Evidence

From independence in 1980 and for nearly a decade thereafter, Zimbabwe made stunning health advances. But AIDS has already erased all the life expectancy gains made since then. Further, if the worst projections come to pass, by about 2010 life expectancy will return virtually to where it stood the day I was born, in what was then Southern Rhodesia, half a century ago.  

- Callisto Madavo, World Bank

In the wake of Hiroshima and the widespread destruction of human life and property during the World Wars, theorists of international relations had ample empirical evidence that war and weapons of mass destruction held the potential to cripple governments and eviscerate entire societies. With the rise of ‘low politics’ in the Post-Cold War era, political scientists are faced with a number of novel (and often competing) claims concerning the changing nature of threats to both national and international security. More often than not, a lack of empirical data and analysis has plagued the evolution of new domains of international relations theory, and this is particularly true of nascent sub-fields such as the environmental, population, information, and health security paradigms. The following chapter provides substantial empirical evidence that there is in fact a strong and significant negative association between infectious disease rates and state capacity, buttressing the claim that health security is a viable new realm for scientific exploration and analysis.

Despite the minor data problems that force us to rely on proxies for the independent variable, the statistical analysis that follows shows a clear and strong negative relationship between ERID and SC. The core of this chapter is to establish whether in fact we can dismiss the null hypothesis. As such the statistical analysis presented here compliments the process-tracing employed in the following chapter, that should allow us to flesh out the likely causal relations between variables.

As discussed in the previous chapter, the covariance between the proxy indicators for ERID and our computed State Capacity measure will either confirm or disconfirm the null hypothesis, namely that infectious disease has no effect on State Capacity. Using standard Pearson’s two-tailed correlation to measure the covariance we find that IM does in fact correlate strongly (negative association) with the various measures of state capacity for each country in the sample. Similarly, we find that Life Expectancy (LX) displays a strong positive correlation with the respective State Capacity variables. Significant trends in the data indicate a strong negative association between the prevalence of infectious disease and individual indicators of state capacity within a particular society. Additionally, there are minor outliers that do not follow the general trends within each of the state capacity categories, but this is to be expected given the wide range of societies and cultures in our sample. I will discuss the presence of outliers with regard to each variable of SC.

Of course, the raw data employed in the analysis is derived from twenty randomly selected countries, and ranges chronologically from 1951 to 1991 A.D. National data quality ranges from excellent (e.g. Japan, Netherlands) to rather sub-optimal (e.g. Uganda, Saudi Arabia). In general, the mortality data (IM and LX) tend to be available
across this time period for all countries in the sample. Ergo, measurements involving the independent variable are not subject to data gaps and do not result in significant problems for the data analysis. Conversely, certain gaps and inconsistencies do exist in the data for individual countries concerning certain SC variables at certain times. For example, there is no currently available data for Government Expenditure in Saudi Arabia over the given time period. Similarly, Iceland has no armed forces and therefore does not have any data for Military Expenditure. Additionally, single and/or multi-year gaps exist in the data for several developing nations, particularly Uganda and Rwanda. These gaps notwithstanding, all available data for the entire time period per specified country has been employed in this analysis. To the extent that these data gaps exist they may have minor impact on the strength of the correlations, but these same gaps will complicate the work of all social scientists involved in the analysis of these variables. Given the existence of imperfect data we must be reasonably cautious in our conclusions, yet the correlations will provide the answer as to whether the null hypothesis can in fact be discredited or not.

The following tables display the trends in the data using a 0 year lag.

Table 3.1: Infant Mortality vs. Gross National Product

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rwanda</td>
<td>-.978**</td>
<td>.956</td>
</tr>
<tr>
<td>Brazil</td>
<td>-.956**</td>
<td>.913</td>
</tr>
<tr>
<td>Colombia</td>
<td>-.955**</td>
<td>.912</td>
</tr>
<tr>
<td>Haiti</td>
<td>-.950**</td>
<td>.902</td>
</tr>
</tbody>
</table>

Note that *, in the following tables, stands for a correlation that is significant to <.050, and ** signifies a correlation that is significant to <.010.
<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malawi</td>
<td>-.931**</td>
<td>.866</td>
</tr>
<tr>
<td>Netherlands</td>
<td>-.926**</td>
<td>.857</td>
</tr>
<tr>
<td>Botswana</td>
<td>-.912**</td>
<td>.832</td>
</tr>
<tr>
<td>Kenya</td>
<td>-.910**</td>
<td>.828</td>
</tr>
<tr>
<td>South Africa</td>
<td>-.903**</td>
<td>.815</td>
</tr>
<tr>
<td>Peru</td>
<td>-.899**</td>
<td>.808</td>
</tr>
<tr>
<td>Thailand</td>
<td>-.881**</td>
<td>.776</td>
</tr>
<tr>
<td>Iceland</td>
<td>-.871**</td>
<td>.758</td>
</tr>
<tr>
<td>Norway</td>
<td>-.860**</td>
<td>.740</td>
</tr>
<tr>
<td>India</td>
<td>-.853**</td>
<td>.727</td>
</tr>
<tr>
<td>Italy</td>
<td>-.852**</td>
<td>.726</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>-.783**</td>
<td>.613</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>-.769**</td>
<td>.591</td>
</tr>
<tr>
<td>Japan</td>
<td>-.760**</td>
<td>.578</td>
</tr>
<tr>
<td>Tanzania</td>
<td>-.348</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>.051</td>
<td>Ns</td>
</tr>
</tbody>
</table>

Note that in this case the data show a strong and significant trend indicating a negative association between infectious disease prevalence (represented by IM) and Gross National Product per capita over the 1951-1991 period. Of the total sample population, 18 states show strong and significant negative correlations between IM and GNP over the selected time period. The balance of evidence provided in the trends above gives us initial reason to question the validity of the null hypothesis. The total strength of this correlation across the sample is (-.950**) significant to (<.001) with an r-square of (.902). Based on the strength of these correlations we can argue that there is a significant and strong negative association between ERID prevalence and GNP per capita, and that this finding is generalizable to the sample population. Two countries of the sample (Tanzania and Uganda) display insignificant correlations, and this is likely due to small N in Uganda’s case. Specifically, the data available for Uganda on this given variable
contain several single and multi-year gaps based on national reporting to the World Bank and IMF. Such gaps may result from any number of exogenous factors, but are probably due to the chronic unrest and internal violence that has plagued that state over the past three decades. The negative association between ERID and GNP per capita has direct repercussions on dimensions of State Capacity such as fiscal resources, legitimacy, reach and responsiveness, resilience, autonomy and human capital.

Table 3.2: Infant Mortality vs. Government Expenditure

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tanzania</td>
<td>-.978**</td>
<td>.955</td>
</tr>
<tr>
<td>Rwanda</td>
<td>-.977**</td>
<td>.954</td>
</tr>
<tr>
<td>Netherlands</td>
<td>-.956**</td>
<td>.914</td>
</tr>
<tr>
<td>Japan</td>
<td>-.951**</td>
<td>.904</td>
</tr>
<tr>
<td>Haiti</td>
<td>-.948**</td>
<td>.898</td>
</tr>
<tr>
<td>Thailand</td>
<td>-.941**</td>
<td>.885</td>
</tr>
<tr>
<td>South Africa</td>
<td>-.930**</td>
<td>.865</td>
</tr>
<tr>
<td>Italy</td>
<td>-.923**</td>
<td>.852</td>
</tr>
<tr>
<td>Colombia</td>
<td>-.891**</td>
<td>.794</td>
</tr>
<tr>
<td>Malawi</td>
<td>-.858**</td>
<td>.736</td>
</tr>
<tr>
<td>Norway</td>
<td>-.825**</td>
<td>.680</td>
</tr>
<tr>
<td>Kenya</td>
<td>-.802**</td>
<td>.643</td>
</tr>
<tr>
<td>Botswana</td>
<td>-.730**</td>
<td>.533</td>
</tr>
<tr>
<td>Iceland</td>
<td>-.727**</td>
<td>.529</td>
</tr>
<tr>
<td>India</td>
<td>-.719**</td>
<td>.517</td>
</tr>
<tr>
<td>Brazil</td>
<td>-.680*</td>
<td>.462</td>
</tr>
<tr>
<td>Peru</td>
<td>-.649**</td>
<td>.421</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>-.381</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>.246</td>
<td>Ns</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Again, trends in the data indicate a strong negative association between ERID and Government Expenditure. Of the total sample population, 17 countries show a strong and significant negative correlation between IM and GOVEX. Ethiopia exhibits a trend
towards this relationship, however its correlation is insignificant. Saudi Arabia has no
data on government expenditure over the selected time period, and Uganda’s correlation
is again insignificant. The total strength of the correlation for the entire sample is a strong
and significant (-.988) significant to(<.001) with an r-square of (.976). Thus, the global
negative association between government expenditure and infant mortality is
exceptionally strong, providing additional preliminary evidence for the rejection of the
null hypothesis. Based on these correlations, we can argue that increasing levels of ERID
result in declining levels of Government Expenditure: undermining the capability of the
government to provide for the basic needs of its citizens. Ergo, ERID would seem to exert
a negative impact on dimensions of SC such as reach and response, legitimacy, human
capital, and resilience.

Table 3.3: Infant Mortality vs. Military Expenditure

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Netherlands</td>
<td>-.947**</td>
<td>.897</td>
</tr>
<tr>
<td>Italy</td>
<td>-.934**</td>
<td>.872</td>
</tr>
<tr>
<td>Thailand</td>
<td>-.903**</td>
<td>.815</td>
</tr>
<tr>
<td>Colombia</td>
<td>-.847**</td>
<td>.717</td>
</tr>
<tr>
<td>Kenya</td>
<td>-.840**</td>
<td>.706</td>
</tr>
<tr>
<td>Norway</td>
<td>-.831**</td>
<td>.691</td>
</tr>
<tr>
<td>Botswana</td>
<td>-.800**</td>
<td>.64</td>
</tr>
<tr>
<td>Japan</td>
<td>-.800**</td>
<td>.64</td>
</tr>
<tr>
<td>Haiti</td>
<td>-.730*</td>
<td>.533</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>-.690**</td>
<td>.476</td>
</tr>
<tr>
<td>India</td>
<td>-.671**</td>
<td>.45</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>-.530*</td>
<td>.281</td>
</tr>
<tr>
<td>Tanzania</td>
<td>-.476*</td>
<td>.227</td>
</tr>
<tr>
<td>South Africa</td>
<td>-.452*</td>
<td>.204</td>
</tr>
</tbody>
</table>

3 Recall that for a correlation to be seen as strong and significant it must exhibit a correlation that is
significant to <.050.
In this instance, the data show a strong negative association between ERID and Military Spending per capita, per soldier (MX). Of the 20 countries in the sample 14 display a significant negative association between the variables. The correlations for five countries in the sample are insignificant, and Rwanda is the only significant outlier from the general trend. Iceland is notable in that it has a correlation of 0 because it has no regular armed forces. The overall correlation for IM vs MX is a powerful (-.884**) significant to (<.001) with an r-square of (.781). The data suggest that infectious disease prevalence does correlate in a negative association with the ability of the state to maintain its armed forces, and thereby ensure the security of that state from external predation and internal disruption. The data suggest that infectious disease might have a major negative impact upon on the ability of the state to provide for national security in the classic sense of deterring external aggression, maintaining internal stability, and projecting force abroad when necessary. Based on these measures we can infer that ERID has a negative effect on dimensions of SC including reach and responsiveness, resilience, and autonomy.

\[ \text{Country} \quad \text{Correlation} \quad \text{R-Square} \]
\[
| \text{Peru} | -.239 | \text{Ns} |
| \text{Iceland} | 0^* | 0 |
| \text{Brazil} | .171 | \text{Ns} |
| \text{Uganda} | .272 | \text{Ns} |
| \text{Malawi} | .709 | \text{Ns} |
| \text{Rwanda} | .380^* | .144 |

* Recall that Iceland has no regular army and therefore no spending per soldier per capita.
### Table 3.4: Infant Mortality vs. Secondary School Enrollment (%)

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Japan</td>
<td>-.988**</td>
<td>.976</td>
</tr>
<tr>
<td>Norway</td>
<td>-.988**</td>
<td>.976</td>
</tr>
<tr>
<td>Kenya</td>
<td>-.986**</td>
<td>.972</td>
</tr>
<tr>
<td>Brazil</td>
<td>-.983**</td>
<td>.966</td>
</tr>
<tr>
<td>Peru</td>
<td>-.979**</td>
<td>.958</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>-.977**</td>
<td>.955</td>
</tr>
<tr>
<td>Colombia</td>
<td>-.969**</td>
<td>.939</td>
</tr>
<tr>
<td>Netherlands</td>
<td>-.963**</td>
<td>.927</td>
</tr>
<tr>
<td>Italy</td>
<td>-.951**</td>
<td>.904</td>
</tr>
<tr>
<td>South Africa</td>
<td>-.950**</td>
<td>.903</td>
</tr>
<tr>
<td>Botswana</td>
<td>-.904**</td>
<td>.817</td>
</tr>
<tr>
<td>Haiti</td>
<td>-.902**</td>
<td>.814</td>
</tr>
<tr>
<td>Iceland</td>
<td>-.844**</td>
<td>.712</td>
</tr>
<tr>
<td>India</td>
<td>-.820**</td>
<td>.672</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>-.806**</td>
<td>.65</td>
</tr>
<tr>
<td>Tanzania</td>
<td>-.780**</td>
<td>.608</td>
</tr>
<tr>
<td>Malawi</td>
<td>-.797**</td>
<td>.635</td>
</tr>
<tr>
<td>Rwanda</td>
<td>-.779**</td>
<td>.609</td>
</tr>
<tr>
<td>Thailand</td>
<td>-.331</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>-.119</td>
<td>Ns</td>
</tr>
</tbody>
</table>

Once more, the data show a strong and significant negative correlation between disease rates and secondary school education as a percentage of the eligible population. The trend is exceptionally strong as 18 countries of the sample show significant negative association between the variables. Only two countries show insignificant correlations, and it is notable that Uganda once again falls outside of the general trend. The overall correlation for the sample between IM and Secondary School enrollment is (-.729**) significant to (<.001) with an r-square of (.531). This significance and polarity of this correlation suggests that rising levels of infectious disease prevalence has a strong negative effect on the ability of the state to provide for the education of its citizens.
Conversely, reducing the burden of disease would seem to allow for a greater provision of education to a given state's population. The data suggest that rising levels of ERID prevalence will undermine the formation and consolidation of human capital within affected societies: the causal processes detailing this relationship are delineated in the following chapter. Coupled with the effects of infectious disease on macroeconomic indicators this provides us with evidence to suggest that increasing levels of infectious disease have a persistent and significant negative impact on the formation and consolidation of human capital and on aggregate national development. The negative relationship between IM and SC affects facets of SC such as human capital, legitimacy, resilience, reach and responsiveness, fiscal strength, and autonomy.

Table 3.5: Infant Mortality vs. Net Long-Term Capital Inflow

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rwanda</td>
<td>-.877**</td>
<td>.769</td>
</tr>
<tr>
<td>India</td>
<td>-.783**</td>
<td>.613</td>
</tr>
<tr>
<td>Japan</td>
<td>-.680**</td>
<td>.462</td>
</tr>
<tr>
<td>Colombia</td>
<td>-.473*</td>
<td>.224</td>
</tr>
<tr>
<td>Haiti</td>
<td>-.464</td>
<td>Ns</td>
</tr>
<tr>
<td>Malawi</td>
<td>-.388</td>
<td>Ns</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>-.380</td>
<td>Ns</td>
</tr>
<tr>
<td>Tanzania</td>
<td>-.372</td>
<td>Ns</td>
</tr>
<tr>
<td>Kenya</td>
<td>-.280</td>
<td>Ns</td>
</tr>
<tr>
<td>Botswana</td>
<td>-.247</td>
<td>Ns</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>-.087</td>
<td>Ns</td>
</tr>
<tr>
<td>Norway</td>
<td>.153</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>.188</td>
<td>Ns</td>
</tr>
<tr>
<td>Brazil</td>
<td>.368</td>
<td>Ns</td>
</tr>
<tr>
<td>South Africa</td>
<td>.510*</td>
<td>.26</td>
</tr>
<tr>
<td>Peru</td>
<td>.608**</td>
<td>.370</td>
</tr>
<tr>
<td>Netherlands</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Italy</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>
Initially, the correlations between IM and CAPIN suggest that there is a weak negative association between ERID and the net intake of foreign capital into a given state. Of the total sample only 4 countries show strong and significant negative correlations between the two variables. Two countries show significant positive correlations between the variables, and the remainder of the sample show correlations that are insignificant, or do not have data for this particular variable for the selected time period. Thus, at first glance the observer is tempted to conclude that there is no conclusive relationship between the variables.

Interestingly enough, when the data for CAPIN is aggregated and run together as a comprehensive sample CAPIN measure against total sample IM, the global correlation becomes significant. This is likely due to the fact that the mean of the data points in the sample is filling in gaps within single national data fields. The overall correlation between IM and CAPIN is (-.686**) significant to (<.001) with an r-square of (.471). This suggests that on a global scale increasing levels of infectious disease prevalence will exert a negative effect on external capital flows into a given state over time. Therefore, states that bear increasing burdens of infectious disease will likely see foreign investment into their given state wane as the holders of capital seek safer havens for their wealth. Thus, ERID has a negative effect on dimensions of SC such as fiscal resources, resilience, reach and responsiveness, autonomy and legitimacy.

Life Expectancy versus State Capacity

As discussed in the previous chapter Life Expectancy (LX) is another proxy variable by which one can track fluctuations in the burden of certain infectious diseases on a given society. Certain pathogens (HIV, Hepatitis) that have a marked effect on LX
do not exhibit a similar effect on IM, and so we can use LX to explore the correlation
between ERID and SC. This is exceptionally useful in determining the prospective impact
of ERIDs such as AIDS, hepatitis and tuberculosis as they have a significant detrimental
effect on Life Expectancy in affected societies.

Table 3.6: Life Expectancy vs. Gross National Product

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>India</td>
<td>.976**</td>
<td>.952</td>
</tr>
<tr>
<td>Rwanda</td>
<td>.963**</td>
<td>.927</td>
</tr>
<tr>
<td>Norway</td>
<td>.960**</td>
<td>.921</td>
</tr>
<tr>
<td>Netherlands</td>
<td>.958**</td>
<td>.917</td>
</tr>
<tr>
<td>Brazil</td>
<td>.952**</td>
<td>.906</td>
</tr>
<tr>
<td>Haiti</td>
<td>.951**</td>
<td>.904</td>
</tr>
<tr>
<td>Iceland</td>
<td>.942**</td>
<td>.887</td>
</tr>
<tr>
<td>Colombia</td>
<td>.940**</td>
<td>.884</td>
</tr>
<tr>
<td>Thailand</td>
<td>.925**</td>
<td>.855</td>
</tr>
<tr>
<td>Malawi</td>
<td>.924**</td>
<td>.854</td>
</tr>
<tr>
<td>Kenya</td>
<td>.920**</td>
<td>.846</td>
</tr>
<tr>
<td>Botswana</td>
<td>.912**</td>
<td>.832</td>
</tr>
<tr>
<td>Peru</td>
<td>.895**</td>
<td>.801</td>
</tr>
<tr>
<td>South Africa</td>
<td>.883**</td>
<td>.78</td>
</tr>
<tr>
<td>Japan</td>
<td>.849**</td>
<td>.721</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>.752**</td>
<td>.566</td>
</tr>
<tr>
<td>Italy</td>
<td>.708**</td>
<td>.501</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>.669**</td>
<td>.447</td>
</tr>
<tr>
<td>Tanzania</td>
<td>.524*</td>
<td>.275</td>
</tr>
<tr>
<td>Uganda</td>
<td>.035</td>
<td>Ns</td>
</tr>
</tbody>
</table>

As the above figures indicate there is a significant and strong correlation between Life
Expectancy and Gross National Product over time for the countries in the sample. Of the
20 countries in the sample 19 showed strong and significant positive correlations between
LX and GNP. This means that rising life expectancy correlates well with increases in

\[\text{\textsuperscript{4} Keep in mind that this data is standardized to constant US dollar figures.}\]
GNP per capita, over time. Similarly, decreases in LX correlate with declines in GNP.

This means that pathogens such as HIV, which have a significant negative impact on LX, will likely erode GNP and undermine state capacity. Note that Uganda takes its usual role as an outlier, displaying an insignificant correlation between LX and GNP. The lack of reliable annual data from Uganda remains problematic and may account for the lack of significance. The overall correlation between LX and GNP is \(0.950^{**}\) significant to \(<0.001\) with an r-square of \(0.903\). The negative association between ERID and GNP per capita has direct repercussions on dimensions of State Capacity such as fiscal resources, legitimacy, reach and responsiveness, resilience, autonomy and human capital.

**Table 3.7: Life Expectancy vs. Government Expenditure**

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>India</td>
<td>.979**</td>
<td>.958</td>
</tr>
<tr>
<td>Tanzania</td>
<td>.979**</td>
<td>.958</td>
</tr>
<tr>
<td>Netherlands</td>
<td>.966**</td>
<td>.933</td>
</tr>
<tr>
<td>Rwanda</td>
<td>.953**</td>
<td>.908</td>
</tr>
<tr>
<td>Thailand</td>
<td>.949**</td>
<td>.901</td>
</tr>
<tr>
<td>Haiti</td>
<td>.949**</td>
<td>.901</td>
</tr>
<tr>
<td>Norway</td>
<td>.939**</td>
<td>.882</td>
</tr>
<tr>
<td>Japan</td>
<td>.926**</td>
<td>.857</td>
</tr>
<tr>
<td>South Africa</td>
<td>.901**</td>
<td>.812</td>
</tr>
<tr>
<td>Iceland</td>
<td>.891**</td>
<td>.794</td>
</tr>
<tr>
<td>Malawi</td>
<td>.885**</td>
<td>.783</td>
</tr>
<tr>
<td>Colombia</td>
<td>.847**</td>
<td>.717</td>
</tr>
<tr>
<td>Kenya</td>
<td>.837**</td>
<td>.701</td>
</tr>
<tr>
<td>Botswana</td>
<td>.710**</td>
<td>.504</td>
</tr>
<tr>
<td>Peru</td>
<td>.632**</td>
<td>.399</td>
</tr>
<tr>
<td>Brazil</td>
<td>.672*</td>
<td>.452</td>
</tr>
<tr>
<td>Italy</td>
<td>.514</td>
<td>Ns</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>.433</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>-.396</td>
<td>Ns</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>
The data above show a strong and significant positive association between LX and Government Expenditure per capita at the national level. Of the total population of the sample 16 countries display strong and significant correlations, Ethiopia follows the general trend while approaching significance, and Uganda takes its usual place as an insignificant outlier. The balance of evidence suggests that pathogens such as HIV and tuberculosis, which have a significant effect on life expectancy, will exert significant negative pressures on state capacity at the national level. Again Uganda’s correlation is insignificant and this suggests that the relatively low N of data points is affecting the correlation. Recurrent warfare in the state may be affecting the availability of N, or alternatively, inconsistencies in the reporting of data may account for Uganda’s continual status as an outlier. Again when we combine the national data into a global measure and correlate it with global SC, we find a strong and significant global correlation of (.778**) significance to (<.001) with an r-square of (.605). This implies that changes in disease-induced mortality have an effect of significant magnitude on variation in government expenditure and that this relationship holds across time and space to be generally applicable to all states in the sample. Ergo, ERID would seem to exert a negative impact on dimensions of SC such as reach and response, legitimacy, human capital, and resilience.

Table 3.8: Life Expectancy vs. Military Expenditure

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Italy</td>
<td>.974**</td>
<td>.949</td>
</tr>
<tr>
<td>Netherlands</td>
<td>.961**</td>
<td>.924</td>
</tr>
<tr>
<td>Thailand</td>
<td>.943**</td>
<td>.889</td>
</tr>
<tr>
<td>Norway</td>
<td>.924**</td>
<td>.854</td>
</tr>
</tbody>
</table>
From the data in the above chart we observe a relatively strong and significant positive correlation between LX and Military Expenditure (per capita/ per soldier). Of the sample population 12 countries show significant positive associations between LX and MX, six countries show correlations that are insignificant, and there are two notable outliers (Iceland and Botswana). Iceland boasts a correlation of 0 which is to be expected given that Iceland has no armed forces, which is hardly surprising given its geographical isolation and lack of historical enemies. Botswana, on the other hand is surprising, but explainable given a low overall population base, and high rates of military expenditure likely due to proximate military threats posed by forces in Angola, South Africa, Namibia, Zimbabwe and Zambia. Following the aggregation of the data into global measures we find that LX and MX show a strong and significant positive correlation of (.894**) significant to (<.001) with an r-square of (.799). This provides us with further

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Japan</td>
<td>.899**</td>
<td>.808</td>
</tr>
<tr>
<td>Kenya</td>
<td>.854**</td>
<td>.729</td>
</tr>
<tr>
<td>Colombia</td>
<td>.820**</td>
<td>.672</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>.654**</td>
<td>.428</td>
</tr>
<tr>
<td>India</td>
<td>.652**</td>
<td>.425</td>
</tr>
<tr>
<td>Haiti</td>
<td>.711*</td>
<td>.505</td>
</tr>
<tr>
<td>Tanzania</td>
<td>.513*</td>
<td>.263</td>
</tr>
<tr>
<td>South Africa</td>
<td>.477*</td>
<td>.228</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>.459</td>
<td>Ns</td>
</tr>
<tr>
<td>Peru</td>
<td>.288</td>
<td>Ns</td>
</tr>
<tr>
<td>Iceland</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Brazil</td>
<td>-.185</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>-.316</td>
<td>Ns</td>
</tr>
<tr>
<td>Rwanda</td>
<td>-.371</td>
<td>Ns</td>
</tr>
<tr>
<td>Malawi</td>
<td>-.752</td>
<td>Ns</td>
</tr>
<tr>
<td>Botswana</td>
<td>-.788*</td>
<td>.621</td>
</tr>
</tbody>
</table>
evidence to discredit the null hypothesis, and gives credence to the theory that ERID
represents a significant threat to the military dimensions of state capacity, such as reach
and responsiveness, resilience, and autonomy.

Table 3.9: Life Expectancy vs. Secondary School Enrollment (%)

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saudi Arabia</td>
<td>.994**</td>
<td>.891</td>
</tr>
<tr>
<td>Brazil</td>
<td>.985**</td>
<td>.970</td>
</tr>
<tr>
<td>Kenya</td>
<td>.985**</td>
<td>.970</td>
</tr>
<tr>
<td>Peru</td>
<td>.970**</td>
<td>.941</td>
</tr>
<tr>
<td>Japan</td>
<td>.968**</td>
<td>.937</td>
</tr>
<tr>
<td>Colombia</td>
<td>.960**</td>
<td>.922</td>
</tr>
<tr>
<td>Netherlands</td>
<td>.944**</td>
<td>.891</td>
</tr>
<tr>
<td>Norway</td>
<td>.933**</td>
<td>.87</td>
</tr>
<tr>
<td>Iceland</td>
<td>.924**</td>
<td>.854</td>
</tr>
<tr>
<td>India</td>
<td>.924**</td>
<td>.854</td>
</tr>
<tr>
<td>Thailand</td>
<td>.915**</td>
<td>.837</td>
</tr>
<tr>
<td>Haiti</td>
<td>.905**</td>
<td>.819</td>
</tr>
<tr>
<td>Botswana</td>
<td>.866**</td>
<td>.75</td>
</tr>
<tr>
<td>Malawi</td>
<td>.863**</td>
<td>.745</td>
</tr>
<tr>
<td>Tanzania</td>
<td>.796**</td>
<td>.634</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>.763**</td>
<td>.582</td>
</tr>
<tr>
<td>Italy</td>
<td>.718**</td>
<td>.516</td>
</tr>
<tr>
<td>Rwanda</td>
<td>.708**</td>
<td>.501</td>
</tr>
<tr>
<td>South Africa</td>
<td>.912</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>.082</td>
<td>Ns</td>
</tr>
</tbody>
</table>

The data show a very strong and significant positive association between LX and
Secondary School Enrollment (SS). Of the sample population, 18 countries show
significant positive association between the two variables. South Africa follows the
general trend and its correlation is approaching significance, while Uganda fulfills its
traditional role as spoiler exhibiting once again an insignificant correlation. The global
correlation between LX and SS is a significant and strong positive association of (.876**)
significant to (<.001) with an r-square of (.767). Based on this data we can conclude that
those pathogens that exert an exceptional negative effect on LX will generate significant
negative pressures on this dimension of state capacity at the national level. In other
words, as the prevalence of lethal ERIDs increases within a given society there will be a
corresponding drop in the ability of the state to provide basic educational services to its
citizens, and this may result in the long-term erosion of human capital within seriously
affected states. The negative relationship between IM and SC affects facets of SC such as
human capital, legitimacy, resilience, reach and responsiveness, fiscal strength, and
autonomy.

Table 3.10: Life Expectancy vs. Net Long-Term Capital Inflow

<table>
<thead>
<tr>
<th>Country</th>
<th>Correlation</th>
<th>R-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rwanda</td>
<td>.895**</td>
<td>.801</td>
</tr>
<tr>
<td>India</td>
<td>.852**</td>
<td>.726</td>
</tr>
<tr>
<td>Japan</td>
<td>.633**</td>
<td>.401</td>
</tr>
<tr>
<td>Colombia</td>
<td>.491*</td>
<td>.241</td>
</tr>
<tr>
<td>Haiti</td>
<td>.471*</td>
<td>.222</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>.401</td>
<td>Ns</td>
</tr>
<tr>
<td>Tanzania</td>
<td>.360</td>
<td>Ns</td>
</tr>
<tr>
<td>Malawi</td>
<td>.307</td>
<td>Ns</td>
</tr>
<tr>
<td>Kenya</td>
<td>.258</td>
<td>Ns</td>
</tr>
<tr>
<td>Botswana</td>
<td>.247</td>
<td>Ns</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>.138</td>
<td>Ns</td>
</tr>
<tr>
<td>Norway</td>
<td>-.197</td>
<td>Ns</td>
</tr>
<tr>
<td>Uganda</td>
<td>-.217</td>
<td>Ns</td>
</tr>
<tr>
<td>Brazil</td>
<td>-.389</td>
<td>Ns</td>
</tr>
<tr>
<td>South Africa</td>
<td>-.534**</td>
<td>.285</td>
</tr>
<tr>
<td>Peru</td>
<td>-.633**</td>
<td>.401</td>
</tr>
<tr>
<td>Iceland</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Italy</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Netherlands</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Thailand</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>
Predictably, the correlation between LX and Net Long-Term Capital Inflow (CAPIN), exhibits a weak positive association at the national level with only 5 countries out of the sample exhibiting strong and significant positive correlations. Indeed, four countries in the sample have no data for CAPIN, and nine states show insignificant correlations. Furthermore, both South Africa and Peru show a significant negative association between the variables and represent strong outliers. In the South African case this apparent effect may be attributable to the extended reign of apartheid, and the drop in foreign investment that accompanied it over the decades. Peru's status as an outlier is enigmatic and deserves further attention in subsequent analyses. It is interesting to note that when the data are combined into global measures the correlation between LX and CAPIN is a promising (.682**) significant to (<.001) with an r-square of (.465).

The data suggest that over time, and on a global scale, improvements in public health correlate with increases in foreign capital inputs into the state. Conversely, as infectious agents take hold on a global scale it will result in declining capital flows into seriously affected states and regions from external sources. Thus, countries that are located in regions of the world with exceptional pathogenic virulence and prevalence (e.g. Sub-Saharan Africa) will experience notable reductions in foreign investment over time. In as much as CAPIN is a logical indicator of SC this provides us with further evidence to reject the null hypothesis. Thus, ERID has a negative effect on dimensions of SC such as fiscal resources, resilience, reach and responsiveness, autonomy and legitimacy.
National Correlations

Persuant to the evidence for these trends on an indicator by indicator basis it is advantageous to examine the relationship between our proxy variables for ERID and a computed SC measure for each state within the sample. The aggregate State Capacity variable was computed courtesy of combining the standardized individual component variables into an additive measure (each with equal weighting) and then dividing so as to obtain a yearly mean sum. There appears to be significant variation at the national level in terms of the relations between IM, LX and State Capacity. Again the tendency is towards a significant negative association between IM and SC, and a positive association between LX and SC. The following correlations delineate these trends.

Table 3.11: ERID Proxies v. National State Capacity

<table>
<thead>
<tr>
<th>Country</th>
<th>IM</th>
<th>Sig.</th>
<th>LX</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botswana</td>
<td>-.697**</td>
<td>&lt;.001</td>
<td>.717**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Brazil</td>
<td>-.761**</td>
<td>&lt;.001</td>
<td>.760**</td>
<td>.001</td>
</tr>
<tr>
<td>Colombia</td>
<td>-.641**</td>
<td>&lt;.001</td>
<td>.583**</td>
<td>.001</td>
</tr>
<tr>
<td>Ethiopia</td>
<td>-.410*</td>
<td>.024</td>
<td>.499**</td>
<td>.005</td>
</tr>
<tr>
<td>Haiti</td>
<td>-.723**</td>
<td>&lt;.001</td>
<td>.727**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Iceland</td>
<td>-.659**</td>
<td>&lt;.001</td>
<td>.702**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>India</td>
<td>-.665**</td>
<td>&lt;.001</td>
<td>.696**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Italy</td>
<td>-.738**</td>
<td>&lt;.001</td>
<td>.682**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Japan</td>
<td>-.425*</td>
<td>.017</td>
<td>.538**</td>
<td>.002</td>
</tr>
<tr>
<td>Kenya</td>
<td>-.629**</td>
<td>&lt;.001</td>
<td>.639**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Malawi</td>
<td>-.386*</td>
<td>.032</td>
<td>.429*</td>
<td>.016</td>
</tr>
<tr>
<td>Netherlands</td>
<td>-.886**</td>
<td>&lt;.001</td>
<td>.941**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Norway</td>
<td>-.819**</td>
<td>&lt;.001</td>
<td>.915**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Peru</td>
<td>-.364*</td>
<td>.044</td>
<td>.402*</td>
<td>.025</td>
</tr>
<tr>
<td>Rwanda</td>
<td>-.670**</td>
<td>&lt;.001</td>
<td>.708**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>-.553*</td>
<td>.002</td>
<td>.504*</td>
<td>.006</td>
</tr>
<tr>
<td>South Africa</td>
<td>-.841**</td>
<td>&lt;.001</td>
<td>.844**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Tanzania</td>
<td>-.763**</td>
<td>&lt;.001</td>
<td>.822**</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
From the above data we observe a strong and significant negative association between IM and SC. Of the sample all 20 countries show significant negative association between the variables, ranging from the strongest correlations in Netherlands, Norway, and South Africa down to Peru and Malawi at the lower end. Interestingly, and for the first time, there are no outliers (significant or insignificant) as each country in the sample shows significant negative correlations between IM and SC. However, within the national correlations between LX and SC, Uganda rears its head again as an insignificant outlier. It is reasonable to assume that greater availability of accurate data from Uganda might drive the correlation toward the general trend of significant positive association. Perhaps inaccurate reporting of data is responsible for this apparent bucking of the overall trend of the sample.

Regional Correlations

Following the strong evidence of a negative association between ERID and SC at the national level it behooves us to aggregate the data and look at the effects that ERID might have on SC at the regional level. One seeks to answer the question as to whether the correlation between IM and SC holds across regions, or are some regions less vulnerable than others? This question is particularly important as those who live in the developed world frequently assume that the dynamic between ERID and SC is largely a
feature of the developing world, and that the global proliferation of ERID does not represent a threat to developed nations.

(1) Americas:

In the subset of countries from the sample we find Brazil, Colombia, Haiti, and Peru. We find that the correlation between IM and SC for this region (0 year lag) is (-.865**) significant to (<.001) with an r2 of (.748). The correlation between LX and SC for the region is (.851**) also significant to (<.001) with an r2 of (.724).

Table 3.12: ERID Proxies vs. SC Indicators, Americas

<table>
<thead>
<tr>
<th></th>
<th>IM</th>
<th>Sig.</th>
<th>LX</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAPIN</td>
<td>.192</td>
<td>.313</td>
<td>-.211</td>
<td>.347</td>
</tr>
<tr>
<td>GNP</td>
<td>-.969**</td>
<td>&lt;.001</td>
<td>.962**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>GOVEX</td>
<td>-.968**</td>
<td>&lt;.001</td>
<td>.961**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MILEX</td>
<td>-.482*</td>
<td>.050</td>
<td>.487*</td>
<td>.048</td>
</tr>
<tr>
<td>SK2</td>
<td>-.810**</td>
<td>&lt;.001</td>
<td>.809**</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

(2) Europe:

The subset of countries in the European region are Iceland, Italy, Norway and Netherlands. The correlation between IM and SC for this region (0 year lag) is (-.908**) significant to (<.001) with an r2 of (.824). Similarly, the correlation between LX and SC is (.948**) significant to (<.001) with an r2 of (.898).
Table 3.13: ERID Proxies vs. SC Indicators, Europe

<table>
<thead>
<tr>
<th></th>
<th>IM</th>
<th>Sig.</th>
<th>LX</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAPIN</td>
<td>.169</td>
<td>.452</td>
<td>-.167</td>
<td>.459</td>
</tr>
<tr>
<td>GNP</td>
<td>-.901**</td>
<td>&lt;.001</td>
<td>.915**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>GOVEX</td>
<td>-.923**</td>
<td>&lt;.001</td>
<td>.846**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MILEX</td>
<td>-.840*</td>
<td>&lt;.001</td>
<td>.904**</td>
<td>.048</td>
</tr>
<tr>
<td>SK2</td>
<td>-.974**</td>
<td>&lt;.001</td>
<td>.939**</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

(3) Asia:

The Asian countries from the global sample are India, Japan, Thailand, and Saudi Arabia. The correlation between IM and SC for this region (0 year lag) is (-.709**) significant to (<.001) with an r2 of (.503). The correlation between LX and SC is also strong at (.718**) significant to (<.001) with an r2 of (.515).

Table 3.14: ERID Proxies vs. SC Indicators, Asia

<table>
<thead>
<tr>
<th></th>
<th>IM</th>
<th>Sig.</th>
<th>LX</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAPIN</td>
<td>-.588**</td>
<td>.003</td>
<td>.622**</td>
<td>.002</td>
</tr>
<tr>
<td>GNP</td>
<td>-.935**</td>
<td>&lt;.001</td>
<td>.953**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>GOVEX</td>
<td>-.820**</td>
<td>&lt;.001</td>
<td>.801**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MILEX</td>
<td>-.798**</td>
<td>&lt;.001</td>
<td>.804**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SK2</td>
<td>-.437*</td>
<td>.014</td>
<td>.846**</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
(4) Africa:

Due to the random selection of the sample eight states fall into this category; these include Botswana, Ethiopia, Kenya, Malawi, South Africa, Tanzania, Uganda, and Rwanda. The correlation between IM and SC for this region (0 year lag) is \((-0.640^{**}\) significant to \(<0.001\) with an r-square of \(0.410\). The correlation between LX and SC, also remains strong at \(0.641\) significant to \(<0.001\) with an r-square of \(0.411\).

<table>
<thead>
<tr>
<th>Table 3.15: ERID Proxies vs. SC Indicators, Africa</th>
</tr>
</thead>
<tbody>
<tr>
<td>IM</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>CAPIN</td>
</tr>
<tr>
<td>GNP</td>
</tr>
<tr>
<td>GOVEX</td>
</tr>
<tr>
<td>MILEX</td>
</tr>
<tr>
<td>SK2</td>
</tr>
</tbody>
</table>

This shows that infectious disease prevalence exhibits a significant negative association with state capacity across the various regions of the globe. While there has been some speculation that disease poses only a regional threat to areas such as Sub-Saharan Africa it is evident that the problem is not localized to any particular region. Based on the data, we can conclude that the negative correlation between infectious disease and state capacity holds across all regions over broad stretches of time. Thus, Europeans and North Americans should also be concerned about the potential threat that the global diffusion of disease represents to all states and societies.
It is interesting to note that the regions with the best data collection capacity also boast the stronger correlations. This leads one to suspect that regions with inadequate data collection and dissemination infrastructures will show lower correlations than one might expect as data gaps increase in frequency (as it is more difficult to get significant correlations). This data problem may explain why the correlation appears to strengthen as you move from lower capacity regions (e.g. Africa) to higher capacity regions such as Europe. An alternative hypothesis that may help to explain the divergence of effect strength according to regional positioning resides in the notion that greater standard deviation may result in insignificant correlations within certain states, and drive down the overall correlation of the South vis a vis the North. Specifically, IM is low and LX is high in industrialized countries and these values do not vary much. Since IM is much higher and LX generally lower in the developing world (with larger standard errors as a result) the correlations in the developing world are likely to be weaker. In other words, the higher correlations in the North might not be the result of better quality data at all but rather the result of the data themselves. This leads one to conclude that while the correlations remain significant and strong at the national and regional levels, the relatively greater standard deviation inherent within the Southern data is likely pulling the strength of the correlation downwards.

As a means to determine whether this trend might hold I then aggregated national data (0 year lags) into groups based on a North (developed world) vs South (developing world) axis. The northern group consisted of Iceland, Italy, Japan, Norway, and the Netherlands. The Southern group consisted of Botswana, Brazil, Colombia, Ethiopia, Ethiopia,

---

5 This idea was derived from conversations with David A. Welch.
Haiti, India, Kenya, Malawi, Peru, Saudi Arabia, South Africa, Tanzania, Thailand, Rwanda and Uganda. Again following the assumption that the data are more complete for the developed North than for the developing South I ran the correlations between IM and SC. and found that the correlation for the North was \((-0.695**)\) significant to \((<0.001)\) with an r-square of \((0.483)\); whereas the correlation for the South remained significant but came in at \((-0.483*)\) significant to \((0.019)\) with r-square of \((0.233)\). A similar effect is observed in the correlations between LX and SC. The North shows a strong correlation of \((0.743**)\) significant to \((<0.001)\) with an r-square of \((0.552)\). The South shows a weaker correlation of \((0.513**)\) significant to \((0.012)\) with a r-square of \((0.263)\). Again, this seems to re-encorese the logical speculation that smaller standard errors within the data serve to intensify the significance of the correlations.

There may be an unfortunate tendency to misinterpret the data and claim that because of the disparity in national and regional correlation strength, that the industrialized states of the North are therefore more vulnerable to the effects of infectious disease. While it is true that the correlation strength is marginally stronger in the North, the correlations in the South remain strong and significant as well. One would expect that the South would show a very similar correlation strength to the North’s given adequate data reporting and/or similar levels of standard error.

Global Correlations

Given the strong negative association between the infectious disease and SC, the logical next step is the examination of the correlation between computed global measures
for IM, LX and SC. Using the sample means from each variable for each year and comparing that against the mean sample IM and LX for each year - we can compute the correlation between global IM/LX and global SC over the entire 1950-1991 time period. The resulting correlation is a strong (-.939**), significant to (<.001) with an r-square of (.882). This demonstrates a powerful negative association between the variables over the selected time period, and gives us more evidence to discredit the null hypothesis. In other words, increasing infectious disease prevalence levels would seem to compromise state capacity.

Similarly, global LX was run against global SC and the correlation turned out to be a powerful positive association of (.939**), significant to (<.001) with an r-square of (.882). This demonstrates that increasing Life Expectancy correlates positively with increasing State Capacity. However, it is helpful to keep in mind that IM is more sensitive to fluctuations in disease prevalence within a society than is LX, therefore I consider the IM vs SC statistic to be more informative.

Lags

One of the principal questions voiced concerns the directionality of causation within the model. In other words, do changes in population health affect SC, or conversely do changes in SC drive health. This question of causality is extremely important because if health drives SC it will have important academic and policy implications as greater investment in population health may be a means to ensure greater long-term prosperity and stability within a given society. Following the analysis of the
global lags between SC and IM and LX over the time period of our study, the data permits further analysis to determine the nature of causality in the equation. By lagging the independent variable from 0 to 15 years we can observe trends over time in the correlations to determine whether IM is in fact driving SC or whether SC is driving IM and LX. Theoretically, of course, there should be a feedback loop involved.

Table 3.16: Global IM vs. Global SC

<table>
<thead>
<tr>
<th>Years Lagged</th>
<th>Correlation</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-.939**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>1</td>
<td>-.937**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>2</td>
<td>-.934**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>3</td>
<td>-.933**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>4</td>
<td>-.935**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>5</td>
<td>-.937**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>6</td>
<td>-.940**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>7</td>
<td>-.941**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>8</td>
<td>-.942**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>9</td>
<td>-.940**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>10</td>
<td>-.764**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>11</td>
<td>-.755**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>12</td>
<td>-.758**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>13</td>
<td>-.739**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>14</td>
<td>-.734**</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>15</td>
<td>-.688**</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Interestingly, when IM was run against SC over a 15 year lag period, the correlation was (-.939**) at 0 years, increasing to (-.942**) at the eighth year lag interval. Following the peak of this effect in the six to nine year lag window the strength of the correlation declines rapidly after the ninth year and then weakens in linear fashion until the end of the lag period at year 15 (-.688**). It should be noted that even at the 15th year lag interval the effect of IM on downstream SC is still powerful.
Table 3.17: Global LX vs. Global SC

<table>
<thead>
<tr>
<th>Years Lagged</th>
<th>Correlation</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>.939**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>1</td>
<td>.934**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>2</td>
<td>.929**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>3</td>
<td>.924**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>4</td>
<td>.923**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>5</td>
<td>.926**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>6</td>
<td>.930**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>7</td>
<td>.933**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>8</td>
<td>.937**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>9</td>
<td>.937**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>10</td>
<td>.937**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>11</td>
<td>.936**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>12</td>
<td>.934**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>13</td>
<td>.933**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>14</td>
<td>.933**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>15</td>
<td>.931**</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

This effect is even more powerful, with LX being a significant driver of downstream SC. At 0 years it is (-.939**) significant to (<.001) with an r-square of (.882). A secondary peak occurs at the 8-10 year window. Ergo, significant negative changes in the life expectancy of a given national population will have significant implications for the future capacity of affected states. Thus those diseases that have a particularly onerous impact on LX will have a significant detrimental effect on SC over time. Given that many states in sub-Saharan Africa boast HIV seroprevalence rates ranging from 10-25 per cent of the population, we can expect substantial reductions in state capacity in the coming decade.
### Table 3.18: Global SC vs. Global IM

<table>
<thead>
<tr>
<th>Years Lagged</th>
<th>Correlation</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-.939**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>1</td>
<td>-.939**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>2</td>
<td>-.943**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>3</td>
<td>-.924**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>4</td>
<td>-.924**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>5</td>
<td>-.929**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>6</td>
<td>-.936**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>7</td>
<td>-.958**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>8</td>
<td>-.441</td>
<td>.100</td>
</tr>
<tr>
<td>9</td>
<td>-.506</td>
<td>.065</td>
</tr>
<tr>
<td>10</td>
<td>-.433</td>
<td>.139</td>
</tr>
<tr>
<td>11</td>
<td>-.197</td>
<td>.539</td>
</tr>
<tr>
<td>12</td>
<td>.066</td>
<td>.847</td>
</tr>
<tr>
<td>13</td>
<td>.365</td>
<td>.300</td>
</tr>
<tr>
<td>14</td>
<td>.578</td>
<td>.106</td>
</tr>
<tr>
<td>15</td>
<td>.703</td>
<td>.052</td>
</tr>
</tbody>
</table>

### Table 3.19: Global SC vs. Global LX

<table>
<thead>
<tr>
<th>Years Lagged</th>
<th>Correlation</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-.941**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>1</td>
<td>-.941**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>2</td>
<td>-.942**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>3</td>
<td>-.942**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>4</td>
<td>-.955**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>5</td>
<td>-.959**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>6</td>
<td>-.957**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>7</td>
<td>-.960**</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>8</td>
<td>-.721**</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>9</td>
<td>-.754**</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>10</td>
<td>-.704**</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>11</td>
<td>-.530</td>
<td>.076</td>
</tr>
<tr>
<td>12</td>
<td>-.273</td>
<td>.416</td>
</tr>
<tr>
<td>13</td>
<td>-.090</td>
<td>.804</td>
</tr>
<tr>
<td>14</td>
<td>-.405</td>
<td>.280</td>
</tr>
<tr>
<td>15</td>
<td>-.601</td>
<td>.115</td>
</tr>
</tbody>
</table>
As expected, when SC was run against IM over a similar 15 year lag period we found that it exhibited a correlation of (-.939**) at 0 years and a correlation of (-.924**) at four years. Thereafter, the correlation increases to (-.958**) at seven years, but the following year the correlation falls apart with the correlations in subsequent years declining to insignificant levels and reversing in polarity. Only in the fifteenth year do we again attain significance, however, the polarity of the correlation is reversed from the initial trend seen in the first seven years. Conversely, SC vs IM exhibited extreme variance in the strength, polarity, and significance of the equation over the rest of the lag period, suggesting that while SC may have a significant effect on ERID levels in the short-term, it does not have a significant effect over the long-term. This leads one to suspect that while SC has a powerful reciprocal influence on IM during the initial seven-year window, it is not driving the incidence or prevalence of ERID within a state over the long term. This makes logical sense when one considers that high capacity states such as the United States and The United Kingdom can have higher levels of HIV infection than lower capacity states such as Iran, due to cultural factors in disease transmission.

Similarly, SC drives LX up until the 10th year but again the correlation falls apart in the 11-15 year range.

Following on this intriguing (and surprising) evidence we can make several claims regarding the hypotheses that we are testing in this project. First, the balance of evidence allows us to reject the null hypothesis, as there is a consistently strong and significant negative correlation between IM and SC, and positive association between LX and SC at the national, regional, and global levels. Moreover, the strong and significant correlation over the lag period (the scale of the effect) indicates that the long-term impact
of infectious disease on population health has a major impact on downstream state capacity. Second, we can confirm that SC has an effect on infectious disease levels, but only within a short window of time (7-10 years), after which the correlations become volatile and insignificant.

This means that there is in fact a negative spiral dynamic or feedback loop operating between ERID and SC such that increasing ERID reduces SC and lowered SC results in increasing levels of ERID. Conversely, positive spiral dynamics may result when low levels of ERID result in increasing levels of SC, and greater levels of SC correspondingly reduce ERID in the immediate downstream window. Notwithstanding the cyclic nature of this dynamic between ERID and SC, the data indicates that ERID has a greater long-term effect on SC than the opposite and thus we can reject claims that SC is driving ERID emergence and prevalence. We can also reasonably conclude that ERIDs exert a significant effect on SC over time and will continue to do so.

Critics

It is true that ERID appears to be primarily a scourge of the developing world, and there is a disturbing tendency on the part of some Western scholars, policy-makers and the media to dismiss ERID as a threat to their populations. First of all, this rationale of denial is shortsighted and bound to lead to serious downstream losses for their respective societies as we exist in the global web of life and changes on the other side of the globe will eventually affect us as well. A prime example of global interdependence was the global capital crash of the Summer of 1998, wherein economic corruption and the lack of
fiscal regulation in Asian economies sent the world into an economic tailspin. The natural world is of course infinitely more complex and interdependent, and as the human species continues to alter the global environment, we will produce corresponding responses from that environment such as the continuing emergence of pathogens.

Critics will argue that only case-specific data per pathogen per year should be correlated against SC, but as I have pointed out at length in the methodology chapter, severe data limitations on a global scale prevent that type of undertaking at this point in time. However, with an eye to this critique I have run U.S. tuberculosis data for the 1950-91 period against SC measures for the United States. Despite the fact that tuberculosis is but one of many ERIDs endemic within the U.S. population, there is still a strong and significant negative association between this one disease and American State Capacity over this time period. The correlation between TB cases (incidence) and U.S. State Capacity is a strong (-.954**) significant to (<.001) with an r-square of (.910). The following table delineates the strength of the relationship between this ERID and US State Capacity over the Cold War period.

Table 3.20: Tuberculosis vs. SC Indicators (U.S.)

<table>
<thead>
<tr>
<th></th>
<th>TB Cases</th>
<th>TB Deaths</th>
<th>TB Rate</th>
<th>TB Death Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAPIN</td>
<td>(-.684**)</td>
<td>(-.472*)</td>
<td>(-.665**)</td>
<td>(-.439*)</td>
</tr>
<tr>
<td>MILEX</td>
<td>(-.827**)</td>
<td>(-.806**)</td>
<td>(-.830**)</td>
<td>(-.803**)</td>
</tr>
<tr>
<td>GNP</td>
<td>(-.832**)</td>
<td>(-.818**)</td>
<td>(-.849**)</td>
<td>(-.819**)</td>
</tr>
<tr>
<td>GOVEX</td>
<td>(-.840**)</td>
<td>(-.867**)</td>
<td>(-.914**)</td>
<td>(-.841**)</td>
</tr>
</tbody>
</table>
This relationship between Tuberculosis incidence/prevalence and State Capacity in the United States is important as it demonstrates the strong correlation between declining levels of infection and a corresponding increase in State Capacity over a forty year period using data from one specific (globally ubiquitous) pathogen. These correlations also show us that the negative association between SC variables and tuberculosis is significant and strong for both morbidity (cases, rate of infection) and mortality figures (TB deaths, death rate). Of note, the correlations between the morbidity variables and SC measures are marginally higher than those of mortality and SC. This would seem to confirm the hypothesis that ERID-induced debilitation combines with mortality to exert a comprehensive negative effect on state capacity. Thus, mortality indicators in and of themselves will not give us a true picture of the toll that ERID exacts upon SC in a given society. The strength of this relationship between Tuberculosis and SC can be extrapolated to the rest of the world, which exhibits lower SC, and in general much higher rates of TB. Thus the U.S. correlations can be seen as a conservative benchmark by which we can gauge the effect of one single pathogen on SC over a broad stretch of time.
Table 3.21: U.S. State Capacity vs. Tuberculosis Incidence/Prevalence

<table>
<thead>
<tr>
<th>SC (years lagged)</th>
<th>TB Cases</th>
<th>TB Rate</th>
<th>TB Deaths</th>
<th>TB Death Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>-.954**</td>
<td>-.972**</td>
<td>-.903**</td>
<td>-.924**</td>
</tr>
<tr>
<td>1</td>
<td>-.945**</td>
<td>-.965**</td>
<td>-.876**</td>
<td>-.900**</td>
</tr>
<tr>
<td>2</td>
<td>-.935**</td>
<td>-.952**</td>
<td>-.811**</td>
<td>-.838**</td>
</tr>
<tr>
<td>3</td>
<td>-.899**</td>
<td>-.922**</td>
<td>-.718**</td>
<td>-.767**</td>
</tr>
<tr>
<td>4</td>
<td>-.775**</td>
<td>-.851**</td>
<td>-.636*</td>
<td>-.711**</td>
</tr>
<tr>
<td>5</td>
<td>-.564*</td>
<td>-.749**</td>
<td>-.558*</td>
<td>-.645*</td>
</tr>
<tr>
<td>6</td>
<td>-.281</td>
<td>-.606*</td>
<td>-.474</td>
<td>-.585*</td>
</tr>
<tr>
<td>7</td>
<td>-.065</td>
<td>-.494</td>
<td>-.611*</td>
<td>-.741**</td>
</tr>
<tr>
<td>8</td>
<td>.069</td>
<td>-.402</td>
<td>-.734**</td>
<td>-.786**</td>
</tr>
<tr>
<td>9</td>
<td>.116</td>
<td>-.401</td>
<td>-.727**</td>
<td>-.782**</td>
</tr>
<tr>
<td>10</td>
<td>.074</td>
<td>-.476</td>
<td>-.700**</td>
<td>-.787**</td>
</tr>
<tr>
<td>11</td>
<td>.134</td>
<td>-.448</td>
<td>-.724**</td>
<td>-.780**</td>
</tr>
<tr>
<td>12</td>
<td>.132</td>
<td>-.339</td>
<td>-.743**</td>
<td>-.701**</td>
</tr>
<tr>
<td>13</td>
<td>.036</td>
<td>-.375</td>
<td>-.762**</td>
<td>-.750**</td>
</tr>
<tr>
<td>14</td>
<td>.027</td>
<td>-.341</td>
<td>-.777**</td>
<td>-.711**</td>
</tr>
<tr>
<td>15</td>
<td>.213</td>
<td>-.163</td>
<td>-.909**</td>
<td>-.891**</td>
</tr>
</tbody>
</table>

The above chart shows the relationship between Tuberculosis morbidity/mortality and State Capacity in the United States over a fifteen-year lag period. It is interesting to note that in the initial five years that tuberculosis-induced morbidity seems to generate a greater negative effect on SC than does TB-induced mortality within the same period, although both the morbidity and mortality effects are powerful in and of themselves. The morbidity effect displayed above tends to weaken in the fifth and sixth year lag intervals and becomes insignificant in the seventh year. This finding concurs with the conclusions drawn in reference to the previous chart in that END-induced morbidity has a powerful negative short-term effect on SC. Conversely, TB-induced mortality over the same time period shows a weaker effect than that of disease-induced morbidity with the strength of the correlations weakening significantly in the fifth and sixth year lag intervals. However, following the seventh-year interval, the effect of TB-induced mortality on state capacity begins to intensify, reaching high levels of significance at the end of the 15th year lag
interval. Initially, this might seem peculiar but it accurately reflects the depletion of human capital within a society as a result of ERID and the significant downstream effect that death has on SC. Thus we can conclude that disease-induced mortality has a significant and strong long-term effect upon SC. This is particularly noteworthy as one of the principal questions raised by this research pertains to which pathogens are most inimical to SC. As the data show, pathogens which induce high-levels of debilitation (i.e. morbidity) in the population likely exert a significant negative short-term effect on SC. Diseases in this range include malaria (plasmodium vivax), cholera, dengue, diptheria, onchocerciasis, schistosomiasis, etc. However, pathogens that generate high levels of mortality will have a greater total impact on both current and downstream state capacity. Ergo, highly lethal pathogens such as HIV, hepatitis (various subtypes), tuberculosis, malaria (plasmodium falciparum), and yellow fever will generate significant negative long-term influences on state capacity.

Given the strength and significance of these correlations, we shall now set about determining how ERID compromises SC, mapping out the causal pathways and mechanisms involved at the state level. This process-tracing approach permits greater understanding of the mechanisms involved and allows us to formulate effective policy measures which can intervene at the break-points in the chain of causation.
Chapter Four

Disease and Economic Development

*Beyond the enormous suffering of individuals and families, South Africans are beginning to understand the cost (of HIV/AIDS) in every sphere of society, observing with growing dismay its impact on the efforts of our new democracy to achieve the goals of reconstruction and development.*

- Nelson Mandela, 1997\(^1\)

Over the centuries there has been considerable debate concerning the sources of industrialization and the nature of the development of economies and societies. The most prominent explanations have been modernization theory and dependency theory. I argue that previous theories of development have generally overlooked a significant biological parameter that lies at the core of international development, specifically the burden of infectious disease on the productivity and the consolidation of human capital in a given population. Following the lead of Robert Fogel, I argue that the mastery of high morbidity and mortality rates in a given population has been a central driver of state prosperity and economic strength throughout recorded history. Similarly, I argue that the continuing and unchecked proliferation of emerging and re-emerging infectious disease represents a considerable threat to the economic development, stability and prosperity of states throughout the world.

Let us perform a mental exercise at this point. Picture a Mercator projection map of the globe and note the global distribution of wealth between states. Wealth is generally

---

located in the temperate regions of the world, with the exception of the Gulf Oil States. Societies in these temperate regions tend to be highly industrialized relative to those countries in the tropics. Even extremely cold countries, such as Canada and the Scandinavian nations are exceptionally prosperous. I argue that there is a biological foundation for development, and that this bioeconomic axiom holds both across time and globally across diverse human societies.²

Pathogens have historically impeded the economic and social development of many societies, particularly those that lie within the tropical regions of the world. If we think of the ‘burden of infectious disease’ as X (the independent variable) and ‘state prosperity’ as Y (the dependent variable) then we can empirically test the association between these variables to determine (1) whether X has an effect on Y, (2) whether the effect of X on Y is significant, (3) what the nature of the association between these variables is, (4) whether this association holds over time and across societies. Based upon the preceding evidence, this chapter argues that the proliferation of infectious disease can compromise the economic and social development of countries, and that the onerous burden of disease in tropical regions may partially explain the vast economic development differential between societies in the tropics and their richer counterparts in the temperate zones.

Over the span of centuries, historians and economists have speculated that infectious disease has played a significant (if enigmatic) role in the rise and fall of societies and empires. Fogel argued that much of England’s prosperity, if not the

² The geographical distribution of industrialized and developing nations, and its possible relation to disease prevalence was discussed with Daniel Deudney during a conversation in Toronto, on November 18 1997.
Industrial Revolution itself, resulted from the conquest of high morbidity and mortality in Britain during the late 18th and early 19th century. \(^3\) This conquest of mortality and morbidity was largely the result of significant advances in public health and in the increasingly equitable distribution of food. Conversely, William McNeill noted that the arrival of the Black Death (bubonic/pneumonic plague) in Europe during the 14\(^{th}\) century had significant and pervasive negative economic and social effects on the European societies of the time, generating widespread economic and political instability throughout the continent.

The buoyancy and self-confidence, so characteristic of the thirteenth century...gave way to a more troubled age. Acute social tensions between economic classes and intimate acquaintance with sudden death assumed far greater importance for almost everyone than had been true previously. The economic impact of the Black Death was enormous.... In highly developed regions like northern Italy and Flanders, harsh collisions between social classes manifested themselves as the boom times of the thirteenth century faded into the past. The plague, by disrupting wage and price patterns sharply, exacerbated these conflicts.... Employers died as well as labourers....\(^4\)

Sheldon Watts also notes the economically destructive power of the Black Death on the fortunes of the once powerful city-state of Venice, and its ramifications for the downstream reduction of its economic power in the international realm.

While Venice was closed down and its plague-dead leadership was being replenished from youthful entries in its Golden Book...Dutch and English entrepreneurs moved into its traditional marketing territories around the

---


\(^4\) William McNeill, Plagues and Peoples, p. 162.
Adriatic and eastern Mediterranean. Once in possession they stayed. Shorn of its major markets and burdened with leaders suffering from sclerosis (young in body but old in mind), Venice soon found itself only a regional power with no economic clout. From this it was but a short step to becoming a museum city.\(^5\)

The role of pathogens as central agents has been emphasized in the explanation of the outcomes in the Peloponessian War, the fall of Byzantine Rome, the collapse of the feudal order in Europe, the conquest of the Americas by European forces, and even in the resolution of the US Civil War.\(^6\) While historians make references to the economic and social dislocation generated by the introduction of a new pathogen to the human ecology, they cannot test directly the empirical significance of infectious disease for prosperity and economic development.

At the national level we have seen strong negative correlations between ERID and macroeconomic measures such as GNP, Government Expenditure, and to a lesser extent Net Long-Term Capital Inflow. On a regional and global scale, the negative associations between ERID and each of these indicators of state capacity remain strong and significant, and improve markedly in terms of Net Long-Term Capital Inflow. Taken together, these correlations indicate a strong negative association between ERID and economic dimensions of State Capacity over time. This in itself is an important finding, and merits further statistical analysis as our data improve over time. However, the statistical analysis only takes us so far in our understanding of the processes by which ERID undermines the economic capacity of a given state. Process-tracing at the state

---


level allows us to map the pathways of the effect of ERID on microeconomic units, from households and firms to its broader effect on economic sectors (agriculture, mining, etc) and finally to its macroeconomic impact on the state. I now examine how disease acts as the sand in the economic engine of a nation.

A Theory of Reciprocal Causation

Historically, the dominant conception of the relationship between health and prosperity in the social sciences held that improvements in population health were the product of greater prosperity. However, many medical and social scientists have recently argued that growing economic productivity often does not contribute to greater population health, since it is associated with such factors as increasing population density, exposure to toxins in the workplace, declining urban public health conditions and sanitation capacity.\^7 The empirical evidence presented in the last chapter supports the reverse hypothesis, namely, that improving public health contributes to the economic productivity of a given society. This growing 'economic capacity' may in turn be channeled back into public health infrastructure to create a positive feedback loop. However, we must recognize that the data show that infectious disease levels have a significant downstream effect on SC over the 15 year lag period; whereas SC only affects

ERID levels in the initial seven year lag window. Thus, improvements in public health have a greater effect on downstream SC than SC on public health.

There are many instances throughout history where industrialization and increased economic productivity have in fact led to sub-optimal health conditions for the local population. One only has to think of eighteenth and nineteenth century Britain to recall the barely tolerable conditions that most urban labourers lived in during the early age of industry, and the surge in environmental and infectious illnesses that accompanied massive shifts towards urbanization. Moreover, enormous inequalities persist across societies that have experienced consistent and comprehensive economic growth.8

Empirical research has confirmed the hypothesis that investment in basic human needs such as education and public health are investments in human capital that promote economic growth and productivity over the long term.9 Similarly, Bruce Moon and William Dixon argue that higher rates of economic growth do not improve a nation’s

---


ability to meet the basic needs of its population. Employing quantitative data for a large sample of countries over a twenty-five year time period, Moon and Dixon found that higher rates of growth did not lead to proportionately higher rates of basic needs improvement. Real product growth does seem to result in absolute gains in basic needs attainment, but the rates of change in basic needs provision do not suggest that growth is conducive to improving the provision of basic needs in developing countries. Indeed...higher rates of growth may have a negative impact upon subsequent basic needs improvements.\(^\text{10}\)

King subsequently tests Moon and Dixon’s findings and confirms their previous conclusions that “…[economic] growth has no clear impact upon basic needs, and what effect it does appear to have is negative.”\(^\text{11}\) He concludes, “Although higher growth rates do seem to exert a negative impact upon basic needs outcomes in the short term of roughly half a decade, the longer-term impact of growth upon basic needs appears to be negligible.”\(^\text{12}\) Given the mounting evidence that economic status does not determine the level of public health in the short-term, and that our data support the hypothesis that rising levels of infectious disease have a significant negative effect on SC over the long term, we can conclude that poverty and economic growth do not seem to result in the emergence of infectious disease. In fact, the evidence leads us to posit that the prevalence of infectious disease in a given society has in fact been a significant contributor to the economic decline of that society over time.

These effects are of course reciprocal in that both negative and positive feedback loops can and do occur between ERID and SC. Countries with low or declining SC (e.g. Haiti, Russia) will not have or be able to maintain the health infrastructures to contain

\(^{10}\) Cited in King, p. 387.
\(^{11}\) Ibid, p. 394.
emerging pathogens. Thus, infection will spread throughout the population, further
depleting human capital and government coffers, which will in turn lead to diminished
resources available to shore up crumbling health infrastructures. A classic example of this
type of negative spiral exists in modern day Russia, which is seeing a dramatic and
worrisome rise in HIV and tuberculosis seroprevalence levels fueled by its collapsing
socio-economic and health infrastructure.12

The short-term influence of low SC on ERID is no surprise, but what is interesting
is the finding that over time ERID can have a reciprocal negative influence on SC.
Anecdotal (and now empirical) evidence bears this out, demonstrated by the Black Death
which swept all of Europe sparing neither peasants in the field nor the powerful elites in
the city-states of Venice, Florence and Milan.14 Indeed, regardless of the initial capacity
of the ‘state’ in question, the Black Death seems to have struck with equanimity.
Similarly, the influenza pandemic of 1918 claimed victims from the developed and
developing world alike, and initial State Capacity did not seem to matter in the
distribution of deaths. In fact, it can be argued that those states exhibiting higher levels of
State Capacity which exhibited greater urban population densities succumbed to the
Spanish Flu faster than lower capacity states with greater proportions of the population

13 Based on evidence presented by Laurie Garrett at the CIS/Medicine Conference on The Social and
Political Impact of Emerging Infectious Diseases, University of Toronto, October 30th, 1998.
14 See Watts, Epidemics and History, pp. 1-38; David Herlihy and Christiane Klapisch-Zuber, Tuscans and
their Families: A Study of the Florentine Catasto of 1427, (New Haven, CT: Yale University Press, 1985),
pp. 73-78; Paul Slack, The Impact of Plague in Tudor and Stuart England (London, Routledge and Kegan
Paul, 1985); Richard Rapp, Industry and Economic Decline in Seventeenth-Century Venice. (Cambridge:
Harvard University Press, 1976); Ann G. Carmichael, Plague and the Poor in Renaissance Florence.
and Economic Consequences”, in Daniel Williman, ed., The Black Death: The Impact of the Fourteenth
Century Plague, (Binghampton, NY: Center for Medieval and Early Renaissance Studies, 1982); and M.W.
living in rural regions. Furthermore, North America and Western Europe were among the first regions to incubate the global HIV pandemic despite having arguably the highest levels of SC on the planet.

Based on the empirical evidence provided in the previous chapter we can reasonably claim that disease prevalence does in fact influence economic capacity, a significant dimension of SC. While the correlations presented in Chapter 3 show this negative association at the global, regional and state levels, it is imperative that we understand exactly how these causal processes function within states. Only if we trace these pathways will we be able to develop and evaluate policies that can mitigate the negative impact of ERID on state capacity and development. Infectious disease exerts this negative impact on state economic productivity and development in myriad ways. The optimal means to analyze the general economic effect of ERIDs is to examine their impact at the three standard levels of economic analysis: Microeconomic (individuals, households, firms), Sectoral, and Macroeconomic.

Microeconomic Analysis

Microeconomics deals with the economic behavior of individual decision-making units in a free-market enterprise system, analyzing consumer spending and saving patterns, the maximization of profits by firms, and the pricing of resources and products. Thus microeconomics is the study of individual components of an economy, such as


firms, households, and prices of goods and services. Process-tracing techniques, which involve a qualitative examination of the probable relations between variables, allow us to demonstrate how ERIDs erode household productivity. These maps may be able to illuminate the 'break-points' in the chain so that effective policy can be formulated to mitigate the negative effects of ERID on productivity.

Within the household unit ERIDs undermine prosperity and generate significant shifts in family spending and saving behavior. Households are defined as one or more individuals who represent both a consumption unit and a production unit.\(^{16}\) Given their endowments of land, other wealth, and the time of their members, households engage in satisficing when they make production, consumption, and savings decisions. At the micro level increased disease incidence and lethality exert a significant negative effect on the household by debilitating and killing productive members, which in turn generates shifts in savings and consumption patterns and results in supply and demand-induced shocks that destabilize the household as an economic (and social) unit.

It is necessary to distinguish between directly and indirectly affected households. Directly affected households are those in which a member of the family unit is ill or has died from an infectious disease. Indirectly affected households are those that directly assist affected households by taking in orphans, helping to pay funeral expenses, and providing additional labour inputs.\(^{17}\) Note that ERID-related adult mortality and morbidity will tend to affect macroeconomic aggregates such as wages and savings, and

---


so everyone in an affected society will be influenced by the resurgence of infectious
disease.

Depending on their virulence and transmissibility, ERIDs reduce the number of
breadwinners in the household, lower household income, and alter patterns of
consumption and savings. At the household level, pecuniary or direct costs that result
from ERID-induced illness consist of personal health care expenditures, costs of
prevention, diagnosis, treatment of illness, and costs of death, (which involve
expenditures for funerals, mourning ceremonies and coffins). Increasing medical costs
and higher funeral expenses will diminish general current expenditures including those
dedicated to savings. Households with infected members may try to increase savings rates
in anticipation of paying onerous funeral costs in the near future.18 These direct costs of
death can be particularly burdensome on the poorer segments of society. For example, in
southern Zambia, coffins cost from $66 to $200. The family of the deceased also pays for
the food, lodging, and transport of mourners. In Kinshasa, the funeral of a pediatric AIDS
death costs a family an average of $320, or the equivalent of 11 months income.19

Other direct costs of ERIDs include nonpersonal costs for educational campaigns,
biomedical research, and blood screening. Arguably, the single most onerous health care
costs for ERID patients are inpatient costs incurred during hospitalization. Annual
inpatient costs reflect the treatment received (drugs, lab procedures, surgery, etc), the
duration of hospitalization, and the frequency of episodes of hospitalization required in a

15 See John Cuddington. "Modeling the Macroeconomic Effects of AIDS, with an Application to
19 Opsit, p. S65.
year. In their study of the economic affect of AIDS in Thailand, Myers et al. found that the annual cost of AIDS treatment was approximately $1000 or 25 000 Baht per case, resulting in the loss of over 50% of the average annual household income. Similarly onerous treatment costs are also reported in Sub-Saharan Africa: in Tanzania, the average cost of each adult AIDS patient over the duration of the patient's illness is roughly 50 000 T Sh, and for children, the corresponding figure is 34 000 T Sh. Given that per capita income in Tanzania was roughly 12 500 T Sh in 1988, it is obvious that these AIDS-related health care costs will become a tremendous burden on the household as the epidemic worsens.

ERID-induced adult deaths can force vulnerable households into poverty. Even in countries such as Tanzania, where the government bears much of the burden of health costs, HIV-affected rural households in 1991 spent $60 (roughly the equivalent of annual rural income per capita) on treatment and funerals. Poonawala and Cantor observed similarly deleterious effects from ERIDs on household savings and productivity. They estimated that AIDS hospitalization in Zaire costs on average four months' wages for the average worker, while a funeral costs 11 months' wages. For indigent families, the reduced per capita income, coupled with the needs of a chronically ill patient, usually

---

result in substandard diets, increased labour-substitution resulting in reduced school attendance for the children of affected households, and lowered standards of living.24

Indirect or non-pecuniary costs result from the lost value of market and non-market output due to increased morbidity or mortality resulting from illness. Indirect costs are the foregone earnings of infected patients and the value of any household services they would have provided. Nonpecuniary costs also include the value that infected individuals, their families and friends, and other members of society place on the pain and death of affected persons, and changes in behavior to avoid contracting or transmitting ERIDs.25 Other ERID-induced indirect costs to the household consist of negative outcomes such as loss of remittances from labour, diminished agricultural inputs, diminished export and food crop production, reduced participation in labour-intensive and nonfood crop production, diminished household assets, and increased levels of malnutrition within family members.26 The combined direct and indirect costs of a lethal pathogen such as HIV/AIDS can have significant long-term detrimental effects on the annual net income of the household. Ainsworth and Over have determined that in severely affected states, roughly 75 per cent of annual household revenue may be lost as a result of HIV/AIDS infection.27 The U.N. estimates that, “the indirect costs of AIDS range from $890 to $2663 in Zaire and from $2425 to $5903 in...Tanzania, which

---

indicates that indirect costs represent roughly 95 per cent of the total costs associated with an AIDS infection.\textsuperscript{28}

The enormous economic burden of disease is in part a function of the synergistic interaction between various pathogens within both individual hosts and societies at large. For example, sexually transmitted diseases (Herpes, Gonorrhea, etc) often create access points for HIV to enter and weaken the immune systems, whereupon pathogens such as tuberculosis then kill the host. Of course, due to data collection problems outlined in Chapter 2, it is extremely difficult to estimate co-infection rates throughout the developing world. Thus, most of the economic analyses to date have focused on the effects of a single pathogen upon a given population. It should be noted that HIV/AIDS has received the lion’s share of this recent attention based on its lethality and rapidly increasing global prevalence rates. Despite the focus on HIV, other ERIDs including malaria, tuberculosis, leprosy, and river blindness also generate negative micro and macroeconomic outcomes. One example of how ERID-induced morbidity affects individual productivity is found in the effects of leprosy.

A study of lepers in urban Tamil Nadu, India, estimates that the elimination of deformity would more than triple the expected annual earnings of those with jobs. The prevention of deformity in all of India’s 645,000 lepers would have added an estimated $130 million to the country’s 1985 GDP. This amount is the equivalent of almost 10 per cent of all the official development assistance received by India in 1985. Yet leprosy accounted for only a small proportion of the country’s disease burden, less than 1 per cent in 1990.\textsuperscript{29}


Based on the evidence provided above, I argue that ERIDs in general and HIV in particular tend to generate economic ‘shocks’ to household savings and consumption patterns. Morbidity and mortality resulting from ERID infection erodes the economic capacity of households as it reduces the time and labour available from members of the household, impairs the supply of education and health of the family unit, and through inheritance customs, reduces the holdings of land, housing, and live-stock available to the household.

Households adjust to these initial shocks over time by attempting to rationally re-distribute their limited human and financial capital in order to overcome the burden of disease, and change their behavior concerning production, expenditure, savings, and investment. Non-infected individuals may spend an increased amount of time caring for the debilitated, additional time working to make up for the lost productivity of the sick, and less time in school. To pay for medical care, they may also draw upon savings, sell assets, borrow from others, or diminish their investments. In many developing nations, affected families may have to sell assets such as land and livestock in order to maintain economic subsistence levels. Indeed, according to World Bank figures ill-health is the cause of 24% of land transactions in Kenya.

Despite the overwhelming (and largely justified) concern regarding the negative economic effects of HIV/AIDS, other ERIDs such as malaria represent resurgent impediments to development. While generally less lethal than HIV/AIDS, malaria

---

30 For additional information on these re-adjustment processes see Ainsworth and Over, “The Economic Impact of AIDS on Africa,” p. 564.
32 With the obvious exception of the highly-lethal falciparum malaria.
frequently debilitates the populations of tropical societies, particularly during the peak months of the rainy growing seasons. In their seminal study on the economic effects of malaria in Africa, Donald S. Shepard et al. estimated the direct and indirect costs imposed by this disease on African societies.

In 1987, a case of malaria cost $9.84 (in 1987 U.S. dollars) - $1.83 in direct costs and $8.01 in indirect costs. As the average value of goods and services produced per day in Africa was $0.82, this cost is equivalent to 12 days of output. By 1995, the average cost of a malaria case is projected to rise to $16.40 (still in 1987 U.S. dollars) due to increasing case severity and chloroquine resistance. At the same time, per capita output is predicted to fall to $0.77, so the burden of one case will rise to 21 days of output. In per capita terms, the burden of malaria is forecast to rise from $1.34 to $4.02. For Africa as a whole, the annual economic burden of malaria was $0.8 billion as of 1987 and will rise to $1.7 billion in 1995; it represented a 0.6% share of GDP previously, and a 1.0% share for 1995.\(^\text{33}\)

Konradsen et al. note that malarial infection results in the loss of approximately 18 percent of annual household net income in Sri Lanka, a significant shortfall considering the synergistic burden of disease in the developing world.\(^\text{34}\)

ERIDs tend to exhibit a greater effect on poorer households than on rich ones. Poorer households often bear a much greater economic burden from infectious diseases than their wealthier counterparts, and thus infectious disease tends to reinforce income inequalities within societies and exacerbate income disparities between classes. In their study of malaria's effect on household income in Malawi, Ettling et al. observed that "very low income households carried a disproportionate share of the economic burden of malaria, with total direct and indirect cost of malaria among these households consuming


32 per cent of annual household income..." Conversely, malaria consumes only 4.2 per cent of the annual income of the Malawian elite household. The implication is that while all Malawians suffer the deleterious economic drag from the plasmodium, the economic burden of the disease falls mainly on the poor, exacerbating the income gap between the average worker and the upper classes and driving marginalized populations into deeper poverty. One of the principal negative effects of ERID on societies is that it increases both perceived and real inequalities between the rich and poor, particularly in the case of diseases like malaria, tuberculosis and cholera that tend to affect marginalized populations. Predictably, the economic gains from improved health are comparatively greater for poorer families, who are usually most handicapped by illness and stand to gain significantly from the development of improved human capital in the form of fewer work days lost to illness, worker productivity, per capita income, improved education and nutrition, greater opportunity to obtain better-paying jobs, longer working lives, and the ability to use previously underutilized natural resources.

---


ERID and Orphans

One effect of the HIV pandemic that is commonly overlooked is the enormous increase in the numbers of orphaned children. The direct and indirect economic costs of caring for orphaned children will impose additional financial burdens on afflicted societies. In 1994 Timothy Wirth estimated that “between 10 and 15 million children will be orphaned as a result of this disease (HIV/AIDS) as we enter the Twenty-First Century.” Within those states that display exceptionally high HIV seroprevalence rates (e.g. Sub-Saharan Africa) AIDS will result in the loss of one and frequently both parents, and will likely claim several aunts and uncles as well, depriving children of stable family structures, education, health care, and training. Both orphans and the elderly will find themselves reduced to destitution as their sole providers of income succumb to the epidemic.

USAID predicts that the number of orphans resulting from the HIV pandemic will greatly surpass Wirth’s estimates, due to the fact that earlier demographic estimates of seroprevalence had significantly underestimated national rates of infection throughout the developing world. Based on the recent findings of USAID studies of 23 countries in the developing world, J. Brian Atwood predicts that more than 34.7 million children will have lost one or both parents to HIV by 2000, with the figure increasing to 41.6 million in 2010. No figures are yet available for global estimates of the likely number of HIV orphans. Aside from the immense human suffering involved, such great numbers of

orphaned children will create immense financial and social strains on heavily afflicted
societies. Atwood predicts dire consequences as a result of the extreme winnowing of the
adult population in many countries.

With children who have lost their parents eventually comprising up to a
third of the population under 15 in some countries, this outgrowth of the
HIVAIDS epidemic will create a lost generation – a sea of youth who are
disadvantaged, vulnerable, undereducated and lacking both hope and
opportunity. What we are seeing here are the seeds of crisis. The creation
of such a large and disaffected demographic “youth explosion” could
propel some of these societies to significant unrest and destabilization over
the long term. The threat to the prospects for economic growth and
development in the most seriously affected countries is considerable.40

This means that for those Sub-Saharan African countries that now exhibit general
population HIV seroprevalence rates of 10 per cent or greater, we can expect the number
of AIDS orphans in those societies to grow exponentially within the decade. In certain
societies such as Zimbabwe, Zambia and Botswana (which now have HIV infection
levels in excess of 25 per cent of the general population), one might reasonably expect
that the rapid increase in orphans might generate increasing socio-economic disruption
over the long-term.

Based on the statistical evidence presented in Chapter 3, we can reasonably
conclude that the proliferation of infectious disease threatens the economic welfare of the
family unit in all societies. However, the relationships illuminated through process-
tracing suggest that ERID poses a relatively greater threat to poorer, marginalized
families, particularly those in the developing world and in societies that rely on privately
funded health-care systems. One of the most significant problems associated with the

40 J. Brian Atwood, Speech on World AIDS Day, December 1, 1997. Available at
proliferation of pathogens is the strong possibility that it will exacerbate economic disparities between upper and lower economic strata within a given society. This may in turn contribute to perceptions of deprivation on the part of the marginalized. We shall pursue this point (and its attendant social and political ramifications) further at the end of the chapter.

In sum then, ERID generates the following costs to the household:

*Direct Costs*

- Increased personal health care expenditures, costs of prevention, costs of diagnosis, treatment costs, costs of death
- non-personal costs such as blood screening, biomedical research, and preventative educational campaigns

*Indirect Costs*

- Lost value of market and non-market output due to illness,
- Lost potential future earnings of debilitated dead members and the loss of household services that they provided.
- The normative/emotive/social value that a given society places on the pain and death of infected members.
- Changes in the behavior of individuals in order to avoid disease contraction.

*Behavioral Changes*

- Labor Substitution
- Re-allocation of time of household members. Greater amounts of time spent working and caring for infected members results in decreased time spent in school.
To pay for medical care, members reduce investments, sell household assets such as land and livestock, draw on savings, or borrow externally.

**Outcomes**

- Lower educational attainment for directly and indirectly affected individuals (resulting in the long-term depletion of human capital for that given state).
- Lower consumption per capita, decreasing aggregate demand.
- Depletion of household income, savings and assets.
- Diminished level of household health.
- Growing inequality between prosperous and marginalized households.

Households are not the only actors at the microeconomic level which are negatively affected by the proliferation of infectious disease. Individual firms, which have endowments of land, buildings, equipment, and a trained workforce, are also subject to the negative effects of ERID. The "environment" in which these firms operate includes market prices, levels of morbidity and mortality in the community, infrastructure, climate, rainfall, and government policies. The proliferation of ERID will likely affect individual firms in several negative ways: increased sick leave and absenteeism, diminished productivity, increased worker turnover, the loss of highly skilled managers, greater training costs, and larger expenditures on health and death benefits.41 The World Bank argues that in severely affected states, the work force will become younger, and lack adequate training. For example, Tanzania and Uganda are already witnessing greater AIDS-induced absenteeism and declining productivity. The majority of macroeconomic

---

41 Ainsworth and Over, "The Economic Impact of AIDS on Africa," p. 566.
models predict that adult AIDS-related deaths will greatly slow the rate of per capita economic growth as compared to a non-AIDS scenario.  

Extensive work has been done on the relationship between investment in human capital and the positive downstream effects on labour productivity.  

ERIDs (notably HIV/AIDS) exert a negative impact on firms through the reduction of the local labour supply (particularly skilled workers), which will over time generate wage increases and cut the profit margins of firms. "The negative labour productivity effect will arise because sick or worried workers are less productive than happy, healthy workers. Even the productivity of those who do not have AIDS may fall as infection and illness rates among friends, families and coworkers rise." This reduced labour supply and productivity combined with increasing wages will undermine the productivity and profit-margins of firms in affected regions. For this reason, multinational firms may choose not to invest in areas where ERID endemicity is particularly high. As we have seen from the data presented in the previous chapter, this is in fact the case: Net Long-Term Capital inflow tends to increase as disease prevalence falls over time. Thus, the proliferation of ERID (particularly HIV) does over time generate disincentives to invest capital within severely affected societies. In extreme cases, exceptionally high rates of ERID infection in local

---

populations may spur capital flight from affected regions to safer havens that have lower levels of endemic disease and hence are more productive.

Sectoral Analysis

*AIDS kills those on whom society relies to grow the crops, work in the mines and factories, run the schools and hospitals and govern nations and countries, thus increasing the number of dependent persons. It creates new pockets of poverty when parents and breadwinners die and children leave school earlier to support the remaining children.*

- Nelson Mandela, Davos 1997

In the broadest sense ERIDs have a negative impact at the sectoral level of the economy, adversely affecting a host of economic mechanisms and generally undermining productivity throughout society. In the formal sector, the proliferation of infectious disease threatens the expansion of industry and the private sector, which exhibit a deep reliance on skilled workers, entrepreneurs and managers. In his 1997 address to the World Economic Forum at Davos, Sir Richard Sykes stated that the (HIV/AIDS) epidemic is already generating a negative effect on the global workforce, markets, and overall business climate. Sykes cited studies done in Southern and Eastern Africa by the African Medical and Research Foundation (AMREF) for AIDSCAP, which concluded that the HIV/AIDS epidemic generates significant negative economic outcomes: the loss of skilled personnel; the need for greater resources to hire and maintain replacement workers; an increase in labour turnover and absenteeism; and a reduction in

---

productivity.\textsuperscript{46} Reductions in labour-supply resulting from increased mortality will also impose shocks to industry. The following Ugandan example is informative.

The impact of AIDS on the economy will be felt through its effect on two key inputs into economic activity - labour and capital. The most immediate impact will be through changes in both the productivity and size of the labour force. By causing premature mortality to a significant number of workers between the ages of 15 and 60, AIDS will reduce both the size and growth of the labour force. By the year 2010, there will be about 2 million fewer in the (Ugandan) labour force age group or approximately 12 per cent less than without AIDS.\textsuperscript{47}

Callisto Madavo notes that in a representative sample of 20 Zambian firms, worker mortality increased from 500 to 800 per cent between 1987 and 1992, largely as a direct result of HIV/AIDS.\textsuperscript{48}

Further examples of the negative impact of HIV on skilled personnel include the loss of the majority of the senior managers of Barclay’s Bank of Zambia to AIDS, while in Malawi nearly 30 per cent of all schoolteachers are HIV positive.\textsuperscript{49} These highly skilled workers represent significant investments in human capital on the part of their employers and home states, and it will be very expensive and time-consuming to replace them. Indeed, African elites have generally been severely affected by HIV/AIDS. For example, in the former Zaire, “among the [largely male] employees at a Kinshasa textile mill, managers had a higher infection rate than foremen, who in turn had a higher rate


\textsuperscript{49} Dr. Piot is currently the head of UNAIDS in Geneva.
than workers." ° Because HIV is so prevalent throughout African elites it is likely to have significant negative effects on senior management structures within firms, undercutting the reservoir of human capital within firms, sectors, and macroeconomies. Peter Piot of UNAIDS argues that HIV/AIDS costs companies operating in Kenya roughly 4 per cent of annual profits, and that as a direct result of the HIV epidemic Kenya's GDP will be 15 per cent less than it would have been (in a non-AIDS scenario) by the year 2005. 51

Besides the economic shocks to firms, resulting from pathogen-induced shortages in labour supply, Hancock and Cuddington agree that ERIDs will undermine the economic productivity of nations as they exert general and profound negative effects on the overall size and quality of the labour force. 52 ERIDs (particularly HIV/AIDS) will affect individual sectors through both demand and supply-side shocks. One example of supply-side shock occurs when a company which is dependent on an ERID-infected labour pool manufactures products for export. While international demand for the product is not affected by the local AIDS epidemic, the firm's labour supply and productivity are diminished, increasing costs and thus narrowing the company's profit margin. 53

Conversely, a healthy and stable work force enables employers to reduce the expense of allocating slack into their production schedules, permits greater investment in staff training that consolidates human capital, and provides employers with the benefits of

specialization. Cohen argues that labour costs will rise as productivity declines because of increasing morbidity and absenteeism: additional training costs will also result from greater labour turnover. Firm expenditures on health and other social programs will also grow, such that the public and private outlays of firms, will rise as a proportion of aggregate expenditure. Thus, the savings available to firms used for financing capital expenditures is diminished.

Other effects of ERID-induced morbidity and mortality on firms include reduced functional capacity in that workers may not be able to do their former jobs. Labour-substitution practices may result as other workers may be required to assume the former responsibilities of ill coworkers. In their 1992 study of the microeconomic effects of AIDS on Thailand, Myers, Obremsky and Viravaidya concluded, that the AIDS epidemic would have a significant negative effect on the performance of the Thai economy. In addition to greater health care costs and foregone income, HIV would likely result in a shortage of skilled labour, increased absenteeism, and significant training/re-training costs.

ERIDs also generate a number of negative effects on the workplace at the sectoral level which range beyond mortality, absenteeism, and reduced productivity of workers. Companies and firms will experience higher recruitment and training costs as well as incurring larger medical and insurance expenditures for benefits and funerals.

---

Health

The global resurgence of ERIDs will hurt the health care sector in affected nations through both supply and demand-side shocks. As greater numbers of the population become infected and develop illness, the demand for medical care will soar, and the associated costs will drain national accounts where governments are the major providers of medical care. Similarly, the increasing prevalence of infection will also debilitate and kill those medical professionals who must minister to the needs of the general population. Some ERIDs, particularly HIV, have the ability to generate significant supply-side shocks within the health sector, as the societal stock of medical personnel are infected, debilitated, and die. Additionally, the HIV pandemic will generate much greater health care costs, as states need to employ greater numbers of healthcare staff. As well, the burdens of medical insurance, disability payments, and life insurance premiums will add to the societal burden of healthcare costs.

Such health sector costs are by no means limited to the developing world, and are doubtless more costly (in absolute terms) to the economies of the developed world. Joshua Lederburg points out that nosocomial (hospital-acquired) infections result in the deaths of over 20,000 Americans per year, and cost the economy $5-10 billion annually.

In absolute terms ERID is likely to have a much greater negative effect on the health care sector throughout the states of the developing world. For example, the World

---

57 Myers, Obremsky and Viravaidya, The Economic Impact of AIDS on Thailand, p. 15.
59 Sir Richard Sykes, Private and Public Partnerships in the Fight Against HIV/AIDS, p. 3.
Bank estimates that the downstream costs of treating all current cases of AIDS cases in Tanzania will consume approximately 40.6% of the public health budget and nearly 25 per cent of combined public and private spending. Similarly, the expense of treating current AIDS cases in Rwanda will absorb an astonishing 60 to 65.5% of the public health budget.61 What this means is that either far less money will be available to treat other health problems (i.e. malaria, tuberculosis, onchocerciasis, heart disease), or that money that would have gone into more productive sectors such as infrastructure or education will have to be diverted to cover national health expenditures. In many cases, the health care system will be completely overwhelmed by the strains of ERID.

According to the U.N. “the direct costs (drug costs, doctors fees, hospitalization care, food, etc.) of treating AIDS patients are enormous and greatly surpass available resources in Africa. For instance, it has been estimated that the direct cost of treating one AIDS patient in Zaire ranges from a low of US $132 to $1585; in...Tanzania, from $104 to $631. But the annual national budgets per capita for all health care were less than $5.”62

Additionally, developing countries are generally unable to afford expensive medications required for the treatment and care of their AIDS patients. The drug AZT, for instance, is estimated to cost $20 000 (U.S.) per patient per annum in 1994. Given the low health care funding available on a per capita basis throughout most of the developing world, it is easy to see how ERIDs will drain government coffers, putting the expensive protease-inhibiting multi-drug cocktails of Western medicine out of reach for the majority

---

of the world's inhabitants. Furthermore, hopes for the development of effective and inexpensive vaccines for the prevention of HIV and medications for the treatment of AIDS patients have been unsuccessful, further constraining countries unable to afford the costs needed for the treatment of its AIDS patients. In Thailand, Viravaidya et al. estimated AIDS health care costs at between $658 and $1016 per year, per infected person. Given that the national average GDP per capita was roughly $1270 in 1991, AIDS-related costs will drain households, and the government is forced to intercede when the family's resources are exhausted. The macro level cost of AIDS to the Thai economy, include lost future earnings of $22 000 per death and ten-year aggregate costs of between $7.3 and $8.7 billion.

The case of India is instructive in that surging HIV prevalence levels threaten to overwhelm the Indian health budget. If the HIV epidemic follows its current trend of proliferation throughout Indian society, by the year 2010 the government will have to spend about one-third more on health care than in a hypothetical non-AIDS scenario. This will necessitate an onerous $2.5 billion increase in the health care budget. Such increases in the health budget are negative for most countries as the revenue must come from other government portfolios, usually education and the provision of other basic human services. Nelson Mandela has also recently voiced his concern that AIDS will

---


drain the coffers of the South African government. "It is anticipated that if current trends continue then AIDS will cost South Africa one per cent of our GDP by the year 2005; and that up to three quarters of our health budget will be consumed by direct health costs relating to HIV/AIDS. Even creative low cost alternatives to hospital care will leave us with a significant impact on our health care budget." Moreover, diseases such as AIDS, malaria, and tuberculosis will combine to accelerate the fiscal drain from competing sectors of government expenditure. Thus, the proliferation of ERID (HIV in particular) constitutes a very real fiscal problem for governments across the globe, and particularly those in the developing world. In a very real sense, the proliferation of ERID will force governments to divert funding from other key sectors (such as education, law and enforcement) into the health sector. This will likely result in increasing government deficits, greater debt, and generally undermine the ability of the government to provide for the basic needs of the population.

Agriculture

A major consequence of the improvement in rural health such as seems to have taken place in England in the century after 1650 was a notable increase in the efficiency of agricultural labour. Healthy people work better - and more regularly; and, as is obvious, losses to agricultural production resulting from inability to do necessary work at the right time of the year disappear in proportion as labourers cease to suffer from debilitating fevers and similar afflictions which tend to crest during the growing season. As health improved, fewer workers could therefore feed larger numbers of city folk.

- William McNeill

---

66 Nelson Mandela, AIDS: facing up to the Global Threat, p. 3.
The global spread of ERID will also have an enormous impact on the labour-intensive agricultural sector in affected regions. The UNDP notes that countries that bear the economic brunt of the HIV pandemic tend to be those that are most reliant on agriculture. Diseases such as malaria, dengue, onchocerciasis, schistosomiasis, and HIV have the greatest ability to undercut the productivity of affected agricultural workforces. Thus, the negative consequences of HIV on the agricultural sector will be substantial throughout developing societies.

The impact of ERID proliferation on agricultural systems is likely to be varied and complex depending on crop type, cash remittances, degree of labour-intensive cultivation practices, and size of holding. In sub-Saharan Africa, the absolute number of individuals infected with HIV is likely to be higher in rural than in urban areas, because the majority of the population continues to live in rural areas. Furthermore, the age groups most seriously affected by the AIDS epidemic are those that are the most productive in the labour-intensive agricultural sector.

Of course, agriculture is a core sector of many developing economies, particularly in Africa, Asia and South America. Agriculture contributes a large share of GDP and for many countries it contributes the majority share of the value of exported products through cash crops. Increasing ERID prevalence rates will similarly afflict the agricultural sector with supply and demand-induced shocks. For example, as pathogens debilitate and kill

---


off the work force, the semi-skilled labour supply for this sector will be reduced, necessitating either labour substitution strategies within households/firms or the drawing of workers from other sectors. This problem of semi-skilled labour shortages tends to get particularly acute in the rainy season when vector-borne pathogens (e.g. malaria, dengue, etc.) attain the highest prevalence within populations. However, given the facts that most developing societies possess an abundant labour-supply, and that labourers can be retrained to work in the agricultural sector rather quickly, we should not be overly concerned about supply side-shocks in this sector. The only obvious concern would arise in the case that an entire region, such as Southern Africa, suffered a rapid demographic implosion due to high prevalence of HIV in the 15-45 year segment of its population distributions. With HIV rates in the area ranging from 15-26% of the total population, this remains a distinct possibility in the coming decade. Of course, imported labour from Central Africa would eventually be used to offset the demographic decline, but such massive adjustment would require some time to enact.

Demand side shocks may also compromise sectoral productivity. Those citizens that succumb to ERIDS are also consumers of agricultural products, therefore in seriously afflicted societies domestic demand for these crops will grow more slowly. If smallholders shift from the production of export to subsistence crops as a response to ERID-induced shocks, export revenues will also suffer over the long term. Barnett and Blaikie argue that

---

70 AIDS and the Demography of Africa, p. 4.
71 Southern Africa may be understood in this case to incorporate Botswana, Zambia, Zimbabwe, Namibia, and South Africa.
Those farming systems that are situated in the semi-arid tropics... will be most vulnerable to labour loss as a result of AIDS. ...the farming systems of southern and eastern Africa in Tanzania, Zimbabwe, Kenya, Botswana and Zambia seem particularly vulnerable, since seropositivity rates have already reached high levels in some rural areas of those countries. In all cases there will probably be implications for foreign-exchange earnings and urban food supplies.73

In her analysis of the Ugandan agricultural sector, Jill Armstrong found that the majority of production is concentrated within “smallholder” farms and tends to be exceptionally labour-intensive. She concurs that HIV/AIDS will compromise key farm production parameters such as labour availability and productivity and it will also limit investment in capital equipment to improve output. Thus, HIV/AIDS will reduce the amount of available disposable income that can be used to acquire agricultural inputs (e.g. occasional extra labour, new seeds or plants, fertilizer, pesticides, or oxen power).74 Armstrong concludes that as a result of increasing ERID infection “labor costs can be expected to increase, reflected in both market wage rates and the shadow wage rate implicit in family farm operations. This, in turn, could lead to a reversal in migration from urban areas or increased migration from other regions with surplus labour.”75 ERID has a number of other possible effects on smallholder agricultural production: (a) the working day may be lengthened, (b) land under cultivation may be reduced, (c) cash

---

74 AIDS and the Demography of Africa, p. 4.
crops may be substituted by less labour-intensive food crops, and (d) planting and weeding may be delayed leading to poor harvests of the loss of an agricultural season.\textsuperscript{76}

Furthermore, human tendencies towards risk-aversion will also result in reduced agricultural productivity under certain circumstances, particularly when vector-borne pathogens are factored into the equation.\textsuperscript{77} As a result of these vector-borne diseases, in many regions of the developing world risk-averse farmers often forego higher output in exchange for diminished income volatility. For example, farmers in malarious regions of Paraguay frequently choose to produce crops that can be grown outside the malaria season, but which are of relatively lower value.\textsuperscript{78} Thus the desire to avoid infection may result in sub-optimal economic outcomes for smallholder agricultural producers.

Under certain conditions health investments can dramatically increase the productivity of land. For example, the reduction of malaria prevalence in Sri Lanka from 1947-77 increased national income by 9 per cent in 1977. The cumulative cost of disease-containment was $52 million, compared with a cumulative gain of $7.6 billion in national income over the same time period. This results in an impressive cost-benefit ratio of $140 gain per dollar originally invested in containment. During the exercise, regions that had been previously rendered hostile by vector-borne disease became increasingly settled as migrants moved in, generating increased output.\textsuperscript{79}


\textsuperscript{77} For example, aerial vector-borne diseases such as malaria, dengue, yellow fever, and various strains of encephalitis have significant negative effects on agricultural productivity throughout the tropical regions of the planet.

\textsuperscript{78} World Development Report 1993: Investing in Health, p 18. Such crops include cheap staples such as maize, wheat, and potatoes.

\textsuperscript{79} Ibid. p. 18.
The World Bank's Onchocerciasis (River Blindness) control program covers 14 Sahelian countries in Africa and has been highly effective in opening new land and increasing agricultural productivity in the region. The program protects approximately 30 million people from Onchocerciasis at an annual cost of less than $1 per capita, and at least 1.5 million previously infected individuals have recovered. The Bank estimates that the program will have prevented at least 500,000 cases of blindness by the year 2000. Furthermore, the River Blindness control program liberated around 25 million hectares of previously endemic land for resettlement and cultivation, generating a significant increase in agricultural production. The total estimated cost of the OCP over the 1974 to 2000 period is about $570 million. The estimated range of increased internal national productivity ranges from 16 to 28 per cent, and the majority of benefits will flow to those in the rural agricultural sector.80

Other ERIDs, such as parasitic nematodes, can also have significant negative effects on the agricultural sector. In Nigeria, Dracunculiasis (Guinea Worm disease) contributed to significant morbidity in over 2.5 million Nigerians during 1987. Cost/benefit analyses revealed that the disease was the chief impediment to increasing rice production in Nigeria, with the net effect of the disease being a reduction in rice production by $50 million in foregone revenue. Modeling suggested that the benefits of a worm control program would exceed its costs after only four years.81 The WHO is currently involved in a Dracunculiasis control program within selected regions of Africa, and the fruits of disease control are growing increasingly apparent. For example,

reductions in Dracunculiasis prevalence within targeted regions have resulted in a roughly 40% increase in food production in those regions. The land under cultivation by farmers in these areas has increased by 25%, and school absenteeism has declined from the level of 60% to 13% in certain regions.82

One obvious lesson that can be drawn from ERID’s negative impact on agriculture is that the proliferation of disease threatens ‘food security’ (i.e. the ability of the state to provide adequate nutritive resources for its population). This may in turn lead to increasing marginalization, famine, and deprivation that will certainly contribute to increasing poverty and human misery. As HIV, tuberculosis, and vector-borne diseases spread across the globe, they have the potential to burden national economies that rely on labour-intensive agriculture to ensure both the sustenance of their own population and to generate exports in order to increase national revenue. The proliferation of ERID threatens urban food supplies and foreign exchange earnings in a number of developing nations, particularly in Sub-Saharan Africa, South and South-East Asia, and Latin America.

Education/Training

One peculiar characteristic of the global AIDS pandemic is that unlike many other ERIDs (i.e. rotavirus) HIV does not spare the elite. HIV prevalence rates in high-income, urban, and well-educated African men are as high (or frequently higher) as rates in low-

income and rural men. Since the elites have greater levels of consumption and investment and accrue higher wages, any disease affecting this group relatively more than other diseases is likely to have a greater impact per case.\textsuperscript{83} Thus, decisions to make long-term societal investments in education are risky in Africa, as these investments in human capital are lost to HIV/AIDS with increasing frequency.\textsuperscript{84} Cuddington argues that premature HIV-induced mortality will continue to erode national stocks of experienced workers, depleting existing reservoirs of human capital and limiting national output.

\[ \text{As AIDS becomes more prevalent, the perceived costs and benefits from undertaking new investments in human capital will change. Total expenditure will shift toward health care and away from schooling. To the extent that AIDS reduces expected lifetime, the incentives for individual workers or their employers to invest in education and training will also be reduced. Shifts in the relative wages of skilled and unskilled workers caused by differences in the prevalence of AIDS among various skill groups might also affect decisions to invest in human capital.} \textsuperscript{85} \]

In the previous chapter, I provided evidence that infectious disease levels exhibit a significant negative correlation with the percentage of the eligible population of a state that is enrolled in secondary school. The cases presented below demonstrate that outbreaks and/or rising levels of disease can prevent children from attending school, or doing well if they do attend school. This provides us with some additional evidence to help interpret the aforementioned correlations, and provides us with additional evidence to infer causality. As the prevalence of infectious disease continues to grow, the costs and benefits from current investments in human capital will likely shift. As a result, reduced

\textsuperscript{83} Ainsworth and Over, "The Economic Impact of AIDS on Africa," p. 561.
\textsuperscript{84} Gerald Helleiner recently noted that of the 20 agro-economists that his group had recently trained in Africa, 10 had died of HIV/AIDS within the last year. Author's conversation with Dr. Helleiner, December 18\textsuperscript{th}, 1998.
spending on education will likely be the consequence of increasing national expenditures on health care to counter the effects of ERID. Given that infectious diseases (particularly AIDS) tend to reduce life expectancy, the incentives for labourers or their employers to invest in training and education will diminish correspondingly. This results from the logic that there is little point in investing scarce resources in the education of someone who may be permanently debilitated by illness and therefore not likely to live to use the skills acquired through education. Furthermore, ERIDs may limit the efficacy of investing in human capital as poor health tends to undermine to capacity of children to learn and acquire skills. Dasgupta has observed that youth who have been prone to illness and malnutrition tend to have learning disabilities and reduced cognitive function which is directly related to their poor health status. The World Bank has found that healthier and well-fed children do much better in school, and enroll with greater frequency than their deprived counterparts. A recent study on Jamaican children infected with whipworm, showed that those debilitated by nematode infection scored 15 per cent lower before treatment than healthy children in the same school. Following treatment, the previously-ill children were retested and achieved scores significantly closer to those of the healthy control group.

Ainsworth, Over and Cuddington expect the HIV pandemic to produce demand and supply shocks resulting in negative outcomes within the education sector. They argue that AIDS will reduce the demand for education relative to an AIDS-free scenario for a

---

86 Ibid, p. 175.
87 For an extensive review of this argument see Partha S. Dasgupta, An Inquiry into Well-Being and Destitution.
number of reasons: reduced cohort size of those entering school, declining enrollment rates due to the fact that affected households may find school fees too burdensome or because children may be needed as labourers or caregivers. \(^9\) USAID figures show that HIV/AIDS in Tanzania will reduce the number of children attending primary school by 22 per cent and the number of children in secondary school by 14 per cent from the level that could be expected without HIV/AIDS. \(^9\) Supply side shocks include depletion of the number of teachers available (due to increasing morbidity and mortality), increased teacher training and turnover costs, and reductions in the efficiency of the education system. \(^9\) "(B)y 2010, Tanzania will have lost 14,460 teachers to AIDS. By 2020, some 27,000 teachers will have died. Training replacement teachers for the year 2020 will cost about $37.8 million (in 1991 dollars) in recurrent costs." \(^9\) Ultimately, these disruptions of the education sector will have a significant negative effect on the downstream formation and consolidation of human capital in severely-affected societies. Over time, as a result of these disruptions, the quality of the labour force will be significantly degraded, impairing long-term prospects for national growth. Demand-induced shocks to the education sector resulting from ERIDs will also have a significant negative effect on the downstream formation of human capital.

Cohen argues that rising adult mortality will also have a negative effect on training and education passed down to subsequent generations, in that rising adult

---

\(^9\) John Cuddington, “Modeling the Macroeconomic Effects of AIDS, with an Application to Tanzania,” p. 175.


mortality will diminish the passing on of acquired skills and knowledge from adults to youth, resulting in the gradual decline of labour productivity.\textsuperscript{93}

\textit{Mining}

As I have argued in the preceding pages, ERIDs have a particularly negative economic impact on those sectors associated with labour-intensive modes of production. Mining is therefore among the most vulnerable of these sectors to the increasing global proliferation of ERID. Mining also tends to make a significant contribution to the economic output of many nations, particularly those in the developing world where resource extraction generates significant employment and revenues from export that can be used to offset foreign debt. For example, the copper industry in Zambia, which depends on young workers for its labour-intensive operations, accounts for almost 25 per cent of the country’s Gross National Product, and 90\% of the country’s export earnings.\textsuperscript{94} The United Nations estimates that approximately 60 per cent of the copper industry’s labour force will be HIV-infected by year 2000. This AIDS-induced morbidity and mortality will impair individual productivity and increase man-hours lost to illness. As well, AIDS will generate increased expenditures resulting from benefits for ill employees and their dependents, death benefits, and the need to train new employees. The combination of these pressures will undermine industry profits and have a significant


negative effect on Zambia's downstream prosperity, particularly if global copper prices recover from their current nadir.\textsuperscript{95}

The mining sector in Namibia accounts for approximately 12\% of GDP and for some 3.5\% of national employment. Thus, mining is a major contributor to national output and generates more than 50\% of total national export revenues. The industry will have to cope with the direct costs associated with the epidemic, namely absenteeism, health costs for employees and dependents, retraining costs and additional recruitment costs. But the greatest costs of the epidemic will result from the loss of skilled managerial and supervisory workers. This erosion of national reserves of human capacity will be difficult, costly and time-consuming to replace.\textsuperscript{96}

\textit{Tourism}

Increasing levels of infectious disease may also disrupt tourism in severely affected regions with the attendant reduction in revenue from foreign sources. Many countries in the developing world extract significant economic benefit from their tourism industries, particularly countries in the Caribbean basin and Oceania. The proliferation of diseases such as malaria, dengue, cholera and HIV may have serious and long-term negative impacts on the tourism sector for many nations, particularly those in the tropics. States that depend on revenues from tourism for employment and foreign exchange are vulnerable to the myriad negative effects of ERID through the reduction of labour supply,

\textsuperscript{95} AIDS and the Demography of Africa, p. 4.
changing domestic demand priorities as ERID reduces income, and demand shifts as
tourists visit more benign destinations.97

In their study of the effect of AIDS on the Thai economy, Myers et al. concluded
that, "Thailand's $5 billion tourism industry may already be feeling the effects of AIDS.
Tourist arrivals are down for a variety of reasons, with the fear of AIDS certain to be a
significant factor in the future."98 Aside from attrition processes such as the HIV/AIDS
pandemic, outbreak events such as the sudden re-emergence of Ebola in Zaire, Plague in
India, and Cholera in Peru generate extreme levels of fear derived from the human
tendency of risk-aversion, and thus tourists avoid affected regions during the outbreak
period. For example, the outbreak of cholera (El Tor strain) in Peru during 1991 had a
significant immediate effect on tourism revenues within that country. Kimball and Davis
estimate that during the outbreak tourism revenues declined between 60 to 70 per cent in
the first quarter of 1991 as compared to the first quarter of 1990.99 Given that the fear of
contagion can drive down tourism and deplete sectoral earnings, afflicted states may have
an initial incentive not to report an outbreak, or to downplay its seriousness.

Macroeconomic Analysis

AIDS is fundamentally a development problem, not just a health problem.
(Our models) ... predict that AIDS will reduce GDP growth by more than it

---

98 Myers, Opremsky and Viravaidya, p.15.
reduces population growth, moving the attainment of reasonable living standards even further into the future. Regardless of the net macroeconomic impact of the epidemic, a profound distributional impact will make some households and communities much worse off, which should be of great concern to policy-makers.

-Martha Ainsworth and Mead Over

The empirical evidence presented in the previous chapter clearly demonstrated that infectious disease rates show a significant negative correlation with macroeconomic national indicators such as GNP per capita, Government Expenditure per capita, and Net Long Term Capital Inflow. It is increasingly apparent that the current resurgence in infectious disease promises increasing poverty and economic destabilization for severely affected countries. At the microeconomic level, we have noted the adverse impact of ERID on economic well-being within individual families and firms, on the general quality of the labour force, on human capital formation and maintenance, and on various sectors of the economy. It is logical to conclude that these microeconomic effects will generate, through multiplier effects, significant negative macroeconomic outcomes. Direct costs to the economy will be enormous, and indirect costs will include lost output due to increasing mortality and (to a lesser extent) ERID-induced morbidity.

The four factors of production are capital, land, technology, and labour. Ainsworth argues that “if the first three of these [factors] grow at a constant rate the slower growth in labour caused by the epidemic will...slow the growth of output”

ERIDs act synergistically, however, to exert negative effects on capital (both human capital and foreign investment) and land (diminishing access due to endemic infestation).

101 Ibid, p. 578.
This negative synergy should result in significant limits on national economic growth, and may result in growing poverty and national economic decline (particularly within the developing world). This supposition is borne out by the data in the previous chapter.

In their study of the effects of HIV on the Cameroonian economy, Kambou, Devarajan and Over argue that the worst economic effects of HIV manifest themselves when significant infection levels are prevalent in skilled urban workforces, and predict that HIV will result in a 2.2% drag on the annual national GDP growth rate in Cameroon. They posit that the growth rates of saving and investment will decline rapidly, undercutting the GDP growth rate. This contraction of real output growth will accompany the simultaneous erosion of macroeconomic competitiveness in international markets. These effects have already been demonstrated in the declining growth rates of exports, and increasing current account problems.\textsuperscript{102}

Thus, ERID impedes the investment of foreign capital, erodes human capital resources (limiting the endogenous supply of social and technical ingenuity within a given state), and renders some land and its natural resources economically useless. ERID may also affect technology as disease-induced mortality may reduce the pool of skilled individuals such as scientists and doctors that would have otherwise contributed to endogenous technological innovation.

**Foreign Investment**

The global correlation between CAPIN and IM is \(-.686^{**}\) (significant to \(<.001\)), with an r-square of \(.471\), while the association between LX and CAPIN is \(.682^{**}\) (significant to \(<.001\)), with an r-square of \(.465\). This means that as disease rates in a given state fall over time, it will have a positive effect on SC, such that foreign investors and other exogenous sources of capital may view that state as increasingly stable and profitable. Of course, correlation does not indicate causation, and this probabilistic relationship will require much deeper analysis before we can make any claims about causation.

CAPIN is one of our core indicators for SC because it measures the influx of economic capital into the state from exogenous sources over time. Rational investors\(^{103}\) will seek to put their capital into politically stable and economically productive societies, and thus this variable indirectly and partially measures state stability and prosperity.

The evidence suggests that high rates of infectious disease prevalence in a given state will generate a disincentive for foreign capital owners to invest in states with high endemic ERID rates. High disease rates tend to correlate with diminished state productivity, and such countries may be less able to attract exogenous capital in order to improve infrastructure and stimulate economic reforms. States with higher pathogen prevalence levels are, therefore, less productive and prosperous. Thus, soaring disease rates in much of the developing world will diminish the probability of increasing foreign

\(^{103}\) Of course, investors exhibit bounded rationality (as do policy-makers) in that they do not have perfect information, and are subject to a host of perceptual and cognitive factors that impair perfect rationality. For an interesting discussion on the limits of rationality see Robert Jervis, *Perception and Misperception in International Politics*, (Princeton, NJ: Princeton University press, 1976).
investment and undermine the ability of severely affected nations to generate economic growth.

Again, the global correlation between IM and Gross National Product (GNP) per capita is (-.950**) (significant to <.001) with an r-square of .903, while the association between LX and GNP stands at (.950**) (significant to <.001) with an r-square of .903. GNP measures the total monetary value of all goods and services produced by a nation’s citizens per annum. The significant negative correlations between IM and GNP, and tuberculosis rates and GNP in the U.S. show a powerful inverse relationship between disease rates and economic productivity. Conversely, the positive association between LX and GNP provides further evidence to support the hypothesis. As Fogel had suggested and as the data show, declining disease rates over time increase the productivity of a given state. It is important to note the centrality of human capital in the economic equation of state prosperity, and understand the significant role that public health plays in the generation and preservation of that valuable human capital. Thus, we may conclude that as public health improves, so does societal economic productivity. Conversely, as HIV, tuberculosis, malaria, and other ERIDs continue to proliferate, we can conclude that this will undermine the economic productivity of severely affected states such as those in Sub-Saharan Africa.

Another core economic indicator of SC is Government Expenditure, which measures the total fiscal outlay of the state on the provision of services (e.g. education, healthcare) to its population on an annual basis. As mentioned above, the correlation between global IM and GOVEX is (-.988**) (significant to <.001) with an r-square of .976, while the correlation between LX and GOVEX is (.778**) significant to <.001,
with an r-square of .605. These statistical findings demonstrate that as ERID rates decrease we observe reciprocal increases in government expenditure. The data show that as disease rates rise in a given country, government expenditure will shift to increased spending on health care and the withdrawal of funds from other sectors. This will eventually result in long-term economic decline due to falling economic productivity. As the growth rate of the economy declines, the revenues available to government will fall. This may well limit the state’s economic capacity to adapt to the crisis.

**Savings**

Infectious disease will also affect savings patterns at the microeconomic level to produce negative outcomes. In Cuddington’s analysis of the effect of AIDS on the Tanzanian economy, he predicts that one of the outcomes of the epidemic is a negative domestic saving effect. According to Cuddington, infectious diseases such as onchocerciasis, HIV, and malaria will affect savings patterns in several ways. First, the immediate effect of increased medical expenditures will diminish saving as well as non-health current expenditures to a certain extent. Second, infectious diseases may negatively alter savings patterns through their pernicious effects on the life expectancy, age structure, healthiness, and growth rate of the population. The attributes of national health delivery systems will determine whether the negative savings effect burdens the private or public sector respectively. Cuddington argues that the decline in domestic savings will generate a corresponding reduction in the formation of capital, and if the
savings decline is significant it will produce a correspondingly large negative effect on per capita income over the long term.\textsuperscript{104}

In developing countries, available savings and their use will significantly affect the growth rate of GNP. There are reasons to expect that the effects of HIV and the general proliferation of ERID will be to reduce total national savings, resulting in reduced investment, diminished productive employment, lower per capita incomes, a drag on GNP growth rate, and in all likelihood a lower level of GNP.\textsuperscript{105} Cohen concludes that ERID-induced declines in national savings resulting from domestic and exogenous sources, will generate a decline in the rate of investment, precipitating a fall in the GNP growth rate. The statistics presented in the last chapter would seem to support this argument as ERID levels demonstrate a significant negative association with GNP per capita over time. Ultimately expenditures on current output will likely grow resulting in fewer savings to generate capital formation. As domestic savings decline, less investment is likely.

Thus, exogenously supplied savings will decline in volume and will diminish relative to the growing needs induced by ERID. Furthermore, it is probable that domestic savings will also decline as most productive sectors will be affected by ERID (notably HIV). Additionally, declining foreign savings as a result of declining net long-term capital inflow will compromise national savings reservoirs over time.\textsuperscript{106}

\textsuperscript{104} John Cuddington, "Modeling the Macroeconomic Effects of AIDS, with an Application to Tanzania," p. 175.
\textsuperscript{106} Ibid, p. 4.
Trade

At the systems level, trade goods from ERID-affected areas may be subject to international embargo. This has been the keystone of the growing discordance between the United Kingdom and its European partners, as British beef and beef by-products have been banned by the rest of the European Community due to the fear of contamination by the BSE prion that causes a lethal new variant of Creutzfeld-Jacob disease (V-CJD) in humans. This trade embargo has seriously strained Britain’s relationship with Brussels, to the extent that Prime Minister John Major once declared ‘diplomatic war’ on the rest of the European Union in an attempt to disrupt the agenda of European unification. The economic damage to Britain has yet to be accurately measured but estimates run from a bare minimum of $8.4 billion (U.S.) to upwards of $48 billion (U.S.).

Similarly, the re-emergence of Cholera in Peru from 1991-1996 had significant effects on trade and national prosperity. The epidemic generated over 600,000 cases and 4,500 deaths in Peru and spread rapidly throughout equatorial South America. Peru experienced trade-related costs resulting from the epidemic, due to cancelled orders for exports of foodstuffs including fresh fruit and seafood. These export costs, including deterioration in prices of export goods, delayed sales, canceled orders, and increased inspection costs resulted in the loss of $700 million in export revenue for 1991 alone.

The Indian city of Surat saw an outbreak of plague in September 1994, resulting in mass panic and the rapid out-migration of peoples from the affected region. Because of the nature of the contagion, people became caught up in what eventually became a global

---

epidemic of fear. Despite the fact that only 56 people perished as a result of the contagion, the economic damage to India resulting from foregone export revenues was significant. Specifically, many Indian goods were the subject of international embargo for the duration of the epidemic, ranging from foodstuffs to fabric and diamonds. Costs of the epidemic to India range from $1.3 to $1.7 billion, a significant sum given the persistent frailty of the Indian economy.\textsuperscript{110}

It is important to differentiate between outbreak events and attrition processes when we evaluate the potential for ERID to increasingly disrupt trade. Attrition processes are highly unlikely to generate the significant levels of affect (fear) that may lead to an international boycott of a given country’s exports. Conversely, outbreak events tend to generate high levels of fear, which when coupled with the generalized human tendency to risk-aversion, result in extreme (and often inappropriate) measures, such as trade restrictions on Indian diamonds. Once the hysteria of an outbreak event dissipates, normal trade patterns will likely resume. Attrition processes, on the other hand, are likely to have greater negative long-term effects on trade, as every factory worker felled by contagion fails to produce a tradable good every minute, day, or year, depending upon the nature of the good in question.

National Costs

Despite the high state capacity of the United States of America, even it is still somewhat vulnerable to the effects of disease proliferation. The U.S. National Science

\textsuperscript{110} See Kimball and Davis, p. 7.
and Technology Council estimates that the annual financial cost of common infectious
diseases in the United States exceeds $120 billion per year. This figure includes
morbidity and mortality associated with:

- **Intestinal infections** $23 billion in direct medical costs and lost productivity
- **Foodborne diseases** $5-6 billion in medical and productivity costs
- **Sexually transmitted diseases** $5 billion in treatment costs (excluding AIDS)
- **Influenza** $5 billion (direct medical costs) and $12 billion (lost productivity costs)
- **Antibiotic-resistant bacterial infections** $4 billion in treatment costs
- **Hepatitis B virus infection** $720+ million in direct and indirect costs

Furthermore, many Americans suffer from infectious complications acquired in hospital.
Approximately 2 million such cases occur every year in the United States, resulting in
approximately 70,000 related deaths. The spread of these nosocomial infections imposes
onerous economic costs – on the order of $10 billion a year.\(^{112}\) In the case of tuberculosis
costs to the Thai economy, Sawert et al found that “Indirect costs resulting from
morbidity and mortality due to the TB epidemic… are U.S. $317 for a treated patient and
U.S. $1900 for a patient who remains undiagnosed. Death of an HIV-infected TB patient
causes an economic loss of U.S. $3490 to society, while the death of a non-HIV-infected
patient results in a loss of U.S. $19,400.”\(^{113}\)

**Malaria**

According to recent USAID estimates, more than 85 per cent of global malaria
cases occur in Sub-Saharan Africa where the disease is responsible for approximately 2.5

\(^{110}\) Ibid, p. 8.
\(^{112}\) Ibid, p. 22.
million deaths per year. The negative economic impact of malaria on the region is substantial and increasing: for example, the direct economic cost of malaria in Africa for 1987 was $800 million; it rose to $1.7 billion by 1995; and it is projected to reach $3.5 billion by the year 2000. Moreover, USAID estimates that health care costs for malaria on an out-patient basis account for approximately 40 per cent of current national public health expenditures in Sub-Saharan Africa. USAID predicts that the region can expect a 7-20 per cent annual increase in the burden of malaria-induced mortality and morbidity in the coming decade. As Shepard, Ettling, Brinkmann, and Sauerbom pointed out, the direct and indirect costs of disease are often greater than the amount of economic aid provided to afflicted regions by international donor agencies. "(I)n 1987 the cost of malaria in sub-Saharan Africa was about $791 million per year. This figure is projected to rise to $1,684 million by 1995. By comparison, the entire health assistance to Africa of a major bilateral donor, the U.S. Agency for International Development, was only $52 million for all conditions." Ettling and Shepard’s study of malaria’s economic cost to Rwanda demonstrated that malaria’s drag on the national GDP rose from 1 per cent in 1989 to an estimated 2.4 per cent in 1995, a 140 per cent increase over 5 years. Following the projected trajectory of increases in malaria incidence, the cost of malaria to the Rwandan GDP can

---

be expected conservatively to reach a minimum of 3.36 per cent by the year 2000. Recent studies by the World Health Organization in Burkina Faso, Chad, Congo and Rwanda, show that the cost of an average case of malaria in Sub-Saharan Africa is equivalent to about 12 days of productive output. The total cost for the area in 1995 was projected at $1684 million or 1% of GDP (having risen from $791 million or 0.6% of the GDP in 1987). This represents an increase in costs to the regional GDP of 66.7% from 1987 to 1995. Extrapolating conservatively from the trendline, malaria alone will exert a drag of 1.67% on the regional GDP by 2003, and an onerous 4.64% by 2012.

Of course, one must consider that the impact of malaria differs from state to state as South Africa has a relatively minor malaria problem compared to the tropical states in the Great Lakes region of Central Africa. Shepard estimates that malaria incidence in Rwanda increased an average of 21 per cent every year through the 1980s (an eightfold increase since 1979), while malaria incidence in Togo increased by a relatively modest 10.4 per cent/annum over the same time period. Furthermore, malarial resistance to quinine increased from 0 to 30 per cent for the whole of Africa, and rose to an astonishing level of 66 per cent in Rwanda, over the same time period. Regional resistance to another powerful anti-malarial agent (fansidar) is estimated at 34 per cent. Unfortunately, malaria is not confined to the African continent and its spread bodes ill for other tropical regions, particularly South Asia. In a detailed study, V.P. Sharma concludes that malaria generates an annual economic loss of anywhere between US $0.5 to $1 billion annually to

the Indian economy.\textsuperscript{120} As malaria spreads throughout South and South-East Asia, Oceania and Latin America, we should expect to see mounting damage to the economies of those affected regions.

\textit{HIV/AIDS}

\textit{We have every reason to assume that the epidemic in South-East Asia will soon be just as widespread as it is in Africa. And that East Africa’s experience – a slow down of its economy – will be replicated in Eastern Europe and the developing countries of Asia and Latin America.}

- Peter Piot, Director UNAIDS\textsuperscript{121}

The significant economic costs resulting from the HIV pandemic are now becoming clearer, draining government coffers and markets in both the developing and developed world. Hellinger notes the burdensome costs imposed by HIV infection on the American health care budget. He estimates that the lifetime cost of treating a person with HIV from the time of infection until death is approximately \$119,000.\textsuperscript{122}

Hanveldt et al. compared the societal impact of HIV/AIDS with other selected causes of male mortality to determine the indirect costs of lost future production in Canada. Over the period 1987-1991, the HIV/AIDS epidemic resulted in the loss of \$2.11 billion (U.S.) to the Canadian economy.

Assuming a 2\% annual growth in earnings and a 3\% annual real discount rate....Deaths due to HIV/AIDS accounted for 2.11 billion in 1990 US$.


\textsuperscript{122} Disaggregated, the estimated costs of individual care from HIV infection until the development of AIDS is \$50,000 while the estimated costs from AIDS development until death is approximately \$69,000. F.J. Hellinger, "The Lifetime Cost of Treating a Person with HIV, \textit{JAMA}, Vol. 270, No. 4, (28 July 1993), p. 74.
Future production loss due to HIV/AIDS more than doubled during the period from 1987 to 1991, from 0.27 to 0.60 billion 1990 USS. Our findings demonstrated HIV/AIDS mortality is already having a dramatic impact on future wealth production in Canada. If the past trend continues, the production lost in 1994 should exceed 0.86 billion 1990 US$ and will account for more than 10% of the total annual loss for men aged 25-64 years.\textsuperscript{123}

In 1993, D.C. Lambert estimated the costs of AIDS-related deaths in France. He concludes that the annual cost of AIDS-induced mortality in France is between 10 and 12 billion (SUS) in 1989 and between 18 and 20 billion in 1992. Based on the trendline of the epidemic he argues that the costs of HIV to the French economy will reach between S32.4 and S36 billion for 1995.\textsuperscript{124} Furthermore, Newton et al. examined the past and potential impact of HIV/AIDS in 19 English-speaking nations of the Caribbean, and estimate that the total annual costs of the epidemic will approach SUS 500 million per annum (in constant 1989 USS) or 2% of GDP in the low scenario, and will exceed SUS 1200 million per annum or 5% of GDP in the high scenario.\textsuperscript{125}

Bloom et al. recently concluded a study to estimate the significant negative impact of HIV on the Human Development Index. The researchers used a sample of 56 countries over the 1980-92 period, and concluded that HIV resulted in the global loss of 1.3 years of human development progress per country. Some countries in particular have borne an enormous toll: Zambia has lost ten years of development, Tanzania lost 8 years, and

Malawi and Zimbabwe lost approximately 5 years. It should be noted that the pandemic has greatly increased in seroprevalence levels since the 1980-92 era, and thus the negative developmental impacts will likely be significantly greater than Bloom’s original figures.

Recent studies by USAID conclude that AIDS will infect 25% of Kenya’s population by 2005, and reduce Kenya’s GDP by 10 per cent. Cuddington argues that HIV/AIDS may reduce Tanzanian GDP in the year 2010 by 15-25% in relation to a counter-factual no-AIDS scenario. Julia Dayton concurs, noting that the presence of AIDS in Tanzania will likely reduce the average real growth rate in the 1985-2010 period by between 15 and 28 per cent, from 2.9 to 4.0 per cent per annum. Over a 25-year period this decreases potential output by between (1980) Tsh 15 billion to Tsh 25 billion. The net impact of HIV on growth of potential GDP per capita is estimated at a reduction of 12 per cent (vs. a counter-factual non-HIV scenario).

Cohen argues that Botswana will witness a dramatic decline in its Human Development Index rankings as a result of HIV. Human development indicators in 2010 will decline below 1996 levels. He also posits that HIV/AIDS will likely result in a net loss of 10% to Namibia’s Human Development Index indicators over the period 1996-2006, and that the lion’s share of these losses will be incurred by the most deprived and marginalized in Namibian society.

---

Piot has warned that several of the most promising emerging markets, particularly China, India, and Thailand are likely to replicate the experience of Sub-Saharan Africa in a few years, which would be a disaster for the global economy.\textsuperscript{131} Myers et al. predict that the negative macroeconomic effects of HIV/AIDS in Thailand, will continue to slow growth "...The total annual health care costs plus the value of lost income is projected to grow from $100 million in 1991 to $2.2 billion by 2000 in the high scenario and from $97 million to $1.8 billion in the low scenario. Over a ten year period, $8.7 billion will be lost due to AIDS illness and death in the high case and $7.3 billion in the low case. These annual cost, both direct and indirect, equal about 16-18 times the per capita GDP."\textsuperscript{132}

A recent study commissioned by the UNDP’s Regional Bureau for Asia and the Pacific has examined the detrimental effect of the HIV/AIDS pandemic on development as measured by the Human Development Index (HDI). The study anticipates that the HIV pandemic will cost Thailand approximately nine years of human development, and will cost Myanmar (Burma) five years of developmental progress between 1992 and 2005. India, which will soon be the HIV/AIDS capital of Asia, will forego one year of development progress by 2005, with the loss accelerating thereafter.\textsuperscript{133}

Piot warns that by the year 2000, the world economic impact of AIDS could be as high as equivalent to 4 per cent of the GDP of the United States or the entire economy of

\textsuperscript{132} See Myers, Obremsky, Viravaidya, p. 8.
\textsuperscript{133} "AIDS Leading Threat to Public Health," \textit{HINDU ONLINE}, available at \url{http://www.webpage.com/hindu/950916/18/1516c.html}. 
India. In the 1994 version of the UNDP’s Human Development Report, the cumulative direct and indirect costs of HIV/AIDS throughout the 1980s have been estimated at $240 billion. “The social and psychological costs of the epidemic for individuals, families, communities and nations are also huge - but inestimable.” The report goes on to predict that “The global cost - direct and indirect - of HIV and AIDS by 2000 could be as high as $500 billion a year - equivalent to more than 2% of global GDP.”

Occasionally, critics argue that rising levels of infectious disease will in fact concentrate wealth in the hands of survivors and thereby raise per capita GDP and the overall standard of living in affected societies. However, models developed by Ainsworth and Over predict that the Malthusian hypothesis under which survivors will experience rising GDP is erroneous, and that the AIDS/HIV epidemic (among others) will in fact reduce GDP growth per capita across many scenarios.

Based on the balance of evidence culled from the SC studies, we can reasonably conclude that the global proliferation of ERID will impose a general drag on the productivity and prosperity of seriously affected states. Under extreme conditions of endemic pathogen infection within the national population pool (e.g. most countries in Sub-Saharan Africa), disease has the potential to destabilize economies and push entire regions into economic decline. Cohen warns that HIV’s “…impact on the economic, social and political systems may be fundamental and structural. The consequences of system collapse are not something that economists can predict or even comprehend. Such

---

collapse, however, may pose a threat to the continued functioning of some countries with high rates of seroprevalence. Unfortunately, social scientists have no idea where the threshold of collapse may lie, after which the system may rapidly slide into a negative spiral dynamic. Available models are based upon the anecdotal experience of historians and their observations of the effects that various forms of pestilence had upon local populations at distant points in time. Given the rapid and significant changes in population density, urbanization, migration, environmental decay, and travel times in the twentieth century, we have no clear idea of the levels at which pathogen seroprevalence in local populations may push societies across a poorly demarcated threshold into a negative economic spiral.

Given the evidence presented above it is reasonable to speculate that one of the principal barriers to the development of pathogen laden tropical areas is the economic burden that ERID inflicts on those societies. If we extrapolate from Fogel's arguments it becomes apparent that one reason for the significant gap between the developed states of temperate regions and the generally underdeveloped tropical states is the intensity of infectious disease in tropical regions. Such arguments are taken as obvious within the medical community, but have yet to be recognized by the community of scholars of international development in the social sciences.

---

138 As with many natural systems, the level of inputs into a system may cross boundaries at which the entire system suddenly and chaotically shifts, gradually establishing a new equilibrium that may be irreversible. This argument follows on Thomas F. Homer-Dixon's concept of 'threshold' dynamics as detailed in Thomas F. Homer-Dixon. "On the Threshold: Environmental Changes as Causes of Acute Conflict," pp. 43-83.
139 See Fogel, "The Conquest of High Mortality and Hunger in Europe and America: Timing and Mechanisms."
The strong association between health and prosperity is certainly evident in Sub-Saharan Africa, which is the area of highest ERID prevalence on the globe, and not surprisingly the poorest region of the world as well, despite the presence of significant natural resources. Besides having the highest prevalence of endemic regional pathogens, Sub-Saharan Africa is also host to the most virulent sub-epidemic of the AIDS pandemic. Now that the HIV pandemic is thoroughly entrenched in Africa, we should be concerned about the economic instability that the epidemic will bring in its wake as it spreads through both South and South-East Asia, and Eastern Europe. The UNDP has predicted that economic losses resulting from AIDS could soon exceed total foreign aid to seriously affected states.\textsuperscript{140} Based on the experience of Sub-Saharan Africa, and given the data we now have, it seems reasonable to predict that as ERID continues to proliferate on a global scale, we will see increasing poverty and economic polarization between social classes within societies.

High rates of ERID impose costs on the family unit, reduce per capita income, reduce savings, reduce government revenues and fiscal resources, divert government expenditure from more productive sectors into health care, produce disincentives to invest in child education, impede settlement of marginal regions and the development of natural resources, negatively affect tourism, and occasionally result in the embargoing of presumably infected trade goods, to produce a general negative effect on the state economy. If many states are similarly affected within a region, such as sub-Saharan

Africa, the net effect will be the underdevelopment of the region as a whole, which may in turn imposes a net drag on global trade.\textsuperscript{141}

Reviewing the evidence, we can draw the following conclusions regarding the economic impact of infectious disease on national productivity.

1. ERIDs impede the formation and consolidation of human capital.\textsuperscript{142}

2. ERID induced morbidity and mortality will generate supply and demand-side shocks to households, firms, and even to entire sectors. These shocks will reverberate across sectors and will tend to destabilize national economies, resulting in declining standards of living, per capita income, and buying power.

3. ERID may result in diminished levels of foreign capital inflow, and generate incentives to embargo infected trade goods.

4. ERID may increase and/or reinforce perceived and real income inequalities within afflicted societies. Such increasing inequalities may generate absolute deprivation and perceptions of relative deprivation in marginalized segments of society, possibly leading to increasing social dysfunction through crime, rioting, and low-intensity violence.\textsuperscript{143}


\textsuperscript{142} In this instance I use the term human capital denotes the level of education, training, literacy, knowledge, and ingenuity inherent in one individual or a group of individuals within a given society.

Ultimately, the global proliferation of ERID poses a significant long-term threat to the economic capacity of states, as ERID induced economic shocks force a contraction of the frontier of national production possibilities and threaten the development prospects of many societies. Indeed, ERID is already undermining hard-won economic and social gains throughout the developing world, particularly in the tropical regions of the globe. The available evidence suggests that the negative effects of infectious disease on economic productivity are, unfortunately, likely to be greatest in the developing world (due to initially low state capacity, geographical endemicity, and lower endogenous adaptation capacity). ERID constitutes a significant long-term threat to the economic stability of states and possibly entire regions, particularly those within the tropical zones of the planet.
Chapter Five

Conclusion

This study has presented several major arguments. First, the increasingly rapid proliferation of pathogens constitutes a significant threat to human life and welfare. Second, I have argued that increasing prevalence of infectious disease will generate greater levels of human mortality and morbidity, resulting in both the gradual erosion of State Capacity and increasing poverty. Thus, pathogen-induced economic decline will increase the demands of the population upon the state for the provision of basic services, even as the ability of the state to both provide those goods and to govern effectively declines. This combination of declining state capacity and increasing deprivation may contribute to increasing governance and development problems in affected states, and may in extreme cases contribute to state collapse.

Empirical Findings

First, indicators measuring disease-induced population mortality show a significant negative association with indicators of state capacity. Thus, these mortality indicators would seem to be excellent predictors of both current and downstream State Capacity. Second, infectious disease prevalence levels show a significant negative empirical association with measures of State Capacity. Given the large global effect that the proliferation of infectious disease will have on population mortality and morbidity,
the increasing global proliferation, lethality and transmissibility of pathogens may act as a stressor to compromise State Capacity in significantly affected states.

Third, the negative empirical association between ERID and SC holds at the state, regional, and global levels, and over time. This is important in that the negative association between the two variables is generalizable across different societies and state structures, geographical areas, and historical periods. Fourth, this study demonstrates the existence of an asymmetrical feedback loop between population health and SC. Lagging of the variables shows that ERID levels tend to drive downstream SC over the entire 15 year period of the lag, whereas SC only drives ERID within a 9 year lag window. The asymmetrical nature of the lags demonstrates that the effect of public health on state capacity is greater than the obverse. Thus, the increasing prevalence of pathogens within a given state’s population is likely to undercut that country’s SC over the long-term: this diminished SC will in turn lead to the increasing proliferation of infectious disease within that society in a negative self-reinforcing feedback loop.

Fifth, based on the analysis of the relationship between ERID and the economic/developmental indicators in the aggregate SC variable, the proliferation of ERID will probably tend to intensify poverty and general human misery in seriously affected states, which may contribute to greater relative or absolute deprivation. This study also articulates the probabilistic linkages between increasing disease prevalence and declining state economic productivity (at the micro and macro levels) in Chapter Four.

This analysis empirically confirms the hypothesis that the continuing global proliferation of infectious disease also poses an increasingly significant obstacle to the economic, political and social development of societies throughout the world. The
negative empirical association of infectious disease levels with the economic and social development indices within the aggregate SC variable suggests that ERID will have a pronounced effect in stunting the development of seriously affected societies. This may provide some explanation as to why sub-Saharan Africa (the historical cradle of many human pathogenic agents) has continually found itself at the bottom of the Human Development Index rankings, despite a wealth of natural resources in the region and the constant infusion of capital and social/technical ingenuity from exogenous sources such as World Bank, IMF, individual donor countries and NGO-sponsored development initiatives. Perhaps the findings of this study may help to inform the theoretical debate on the determinants of international development as well.

Theoretical Ramifications

This study reconceptualizes the theoretical linkages between infectious disease and security, empirically shows that the null hypothesis is false, and provides preliminary evidence that supports the hypothesis which links infectious disease to state instability. It also clearly maps out the probabilistic relations between the independent, intervening, and dependent variables, the probable directionality and polarity of associations between these variables, and then demonstrates the statistical strength of these associations. This study employs a research methodology that examines the interaction of complex human and natural systems. Thus, it contributes to the quest for consilient knowledge and serves as a nascent bridge between the natural and social sciences. Based on the empirical findings herein, we may logically conclude that changes in the natural world can and do
frequently have an effect upon social and economic phenomena, and these effects may contribute to political instability and sub-state violence.

Because of the significance and scale of the correlations between health indicators and State Capacity we may be able to employ population health data to predict downstream SC in a given country with some degree of success. This may in turn help social scientists devise increasingly accurate models to predict state stability over the long-term. However, further empirical analysis with a greater N and increased number of data points will be necessary before we can draw any hard and fast conclusions concerning the exact predictive value of health data in its relationship with SC.

Considering that the null hypothesis has been empirically falsified, we can justifiably conclude that population health does in fact have a significant effect on state capacity. This means that the natural world does have significant, although admittedly difficult to observe, effects upon human social structures. This provides additional ammunition for the theories of scientists such as Sir Francis Bacon, William Whewell, Edward O. Wilson, and William McNeill who have consistently argued that great truths may be found when we investigate the possibility of consilient knowledge between the great branches of scientific endeavor. Conversely, the continued fragmentation and compartmentalization of knowledge in the sciences as a general trend may lead us to bypass certain avenues of consilient scientific investigation, which may in turn induce a greater ignorance of the perils that may await humanity as we continue to alter our global ecology.

Furthermore, the finding that population health would seem to drive SC in a reciprocal spiral over a broad span of time is not only theoretically novel and intriguing...
in and of itself, but it provides significant evidence to buttress the claims of King, Moon, and Dixon that poverty is not in fact the principal determinant of population health, and that pathogens should rightly be seen as independent variables. This is significant because it allows us to reject the simplistic common assumption that disease is simply the product of the maldistribution of scarce economic resources: indeed there is in fact widespread evidence to the contrary. One only has to look at the fact that HIV spread throughout Europe and North America before it spread throughout Eastern Europe and South Asia to comprehend that poverty alone does not account for the proliferation of ERID. The finding that disease-induced mortality tends to have a greater long-term effect on SC over the fifteen-year lag period than the obverse may be of significant theoretical interest to those involved in development studies. This finding may help to explain the relatively rapid development of states in ‘the North’ (where fewer endemic diseases have historically afflicted the population), as compared to countries in the tropical regions of the globe, or ‘the South’. This hypothesis remains highly speculative, but it may partially explain why the South has industrialized at a slower pace than the North, why the South started from a lower initial base of SC during this process, and why tropical regions continue to experience significant problems in terms of economic development and effective governance.

Additionally, this examination of the feedback loop leads us to conclude that countries with lower initial levels of SC are in greater danger of pathogen-induced destabilization and poverty than those states which currently exhibit high levels of SC, owing to the reciprocal nature of the Population Health/State Capacity spiral. Thus, ERID represents a greater threat to states in the developing world and regions in decline (e.g.
South Africa, Colombia, and Russia) than to those developed states with relatively high initial levels of SC (e.g. Canada, Germany, Japan).

Given that ERID will act as a stressor on SC, and reverse development gains in seriously affected states, the resultant disease-induced poverty and general human misery may exacerbate relative and absolute deprivation on the part of the population. ERID may thus increase relative and/or absolute deprivation, while simultaneously increasing the demands of the populace upon the state for the provision of basic needs and services. All of this occurs as the state's capacity to respond to these demands correspondingly diminishes. Thus, if the deprivation/state weakness hypothesis is correct, the global proliferation of infectious disease may accelerate and/or exacerbate pre-existing societal tensions, and may in some cases contribute to increased criminal activity, low-intensity intrastate violence, and in certain extreme cases, contribute to state failure.

Finally, this analysis develops a new theoretical framework that examines the probabilistic relations between ERIDs, State Capacity, and societal deprivation, and may assist to some extent in the prediction of state failure and intrastate violence in the future. Such a conceptual tool may help to partially explain the breakdown in governance within states such as Zaire, Haiti, Rwanda, Burundi and Cambodia. This thesis does not, nor does it seek to, explain all civil and intrastate conflict, rather it seeks to shed some light on the increasing instability and state failures that we are currently observing throughout the developing world, and to warn the peoples of the developed world that they remain potentially vulnerable to the various negative macro effects of infectious disease.
Policy Ramifications

Drawing on the evidence provided in this dissertation, we can reasonably conclude that the proliferation of ERID will result in the increasing destruction and debilitation of the population base of seriously affected countries. ERID will also simultaneously increase poverty and misery throughout these societies, erode and/or prevent the consolidation of endogenous human capital, and through the erosion of State Capacity, ERID may in fact increase the probability of social unrest, governance problems, and political violence within states with high infection rates.

The resurgence of ERID may have significant implications for state survival, stability and prosperity, and ramifications for interstate relations as well. The premature death and debilitation of a significant proportion of a state’s population erodes worker productivity and undermines state prosperity, erodes human capital within a given society over the long-term, threatens the state’s ability to both defend itself and to project force, generates institutional fragility and generally impedes the state’s ability to govern effectively.¹

Plagues have probably contributed to the collapse of governance over the broad span of history: they hampered the Athenian war effort during the Peloponnesian war, contributed to the demise of Byzantine Rome and to the destruction of the feudal order in Europe, and were the primal force in the annihilation of the pre-Colombian societies in the Americas after their first contacts with Europeans.² This dynamic is not relegated to

¹ Of course, this assumes that states seek more than just survival and power as Classical realists, such as Hans Morgenthau, would have us believe. Liberalism argues, correctly, that states seek prosperity and stability in addition to survival and security.
² This conclusion is based on the aforementioned negative empirical association between infectious disease and SC. The statistical evidence shows that the arguments of historians such as McNeill, Crosby, Zinsser, Watts, and Oldstone (linking plagues with the collapse of empires and societies) are likely accurate. See
the annals of history but continues to affect state capacity in the modern era. Because of the negative association between infectious disease and state capacity, the global proliferation of emerging and re-emerging diseases (particularly HIV, tuberculosis, and malaria) is a threat to international economic development and effective governance at the state level. This allows us to draw several preliminary policy conclusions. First, the growing destabilization of sub-Saharan Africa may result in part from the exceptionally high ERID levels in the region, particularly that of HIV/AIDS, tuberculosis and malaria. Indeed, the extreme and persistent governance problems in the Great Lakes region of central Africa may be related to increasing ERID stresses on state capacity.

Global phenomena such as ERIDs frequently act in concert with other global collective action problems such as environmental degradation, resource scarcity, and overpopulation to strain state capacity. This synergy between stressors of state capacity will increasingly destabilize seriously affected states and in some cases entire regions (such as sub-Saharan Africa). We must foster increased communication and cooperation between the global policy and medical communities and provide increased resources for surveillance, containment, and co-operative policy measures to check the global proliferation of emerging and re-emerging diseases. Above all, we must bring the gravity of these issues to the attention of the heads of all governments, as the greatest requirement for stemming the global tide of infection is political will.

Tangible actions that governments should take include the establishment of a global disease surveillance system, incorporating the successful civil-society model of the ProMED network that currently monitors disease outbreaks. Governments should also undertake the collection of ‘health intelligence’ such that we can monitor the progression

Oldstone, *Viruses, Plagues and History.*
of diseases through the populations of states that either cannot provide accurate statistics on disease prevalence, or refuse to for political reasons. Policy-makers must also take action to reduce the pace of global environmental degradation, curb the abuse of anti-microbial medications within their societies, and provide increased funding for research to develop vaccines and other anti-microbial agents.

Although the World Health Organization has been the principal actor engaged in tracking disease emergence and proliferation, the WHO faces several problems in dealing with these issues. Although funding is increasingly diverted within the WHO to infectious surveillance, treatment and control, these programs are generally underfunded, understaffed and have proven generally less than effective in fighting the re-emergence of ERID on so many different fronts. Thus, greater resources must be given to the WHO to increase its capacity, and these funds should be specifically targeted to deal with the greatest current ERID threats (HIV, tuberculosis, and malaria). The United States and Japan are currently developing a policy framework for greater co-operation in checking the spread of ERID within and between their own territories. Furthermore, the G7 states are exploring the means by which they might collaborate to reduce the threat of emerging diseases to their populations. While these efforts have not produced any concrete results in the form of multilateral anti-contagion regimes, they are a step in the right direction.

The following policy recommendations can be made at this time. First, ERID poses a direct threat to stability and prosperity throughout the developing world. The region currently experiencing the greatest amount of ERID-induced poverty and instability is Sub-Saharan Africa. Rising ERID levels in South Asia, South-East Asia, and Eastern Europe are worrisome and demand greater surveillance. Vulnerable key states
that are experiencing rapid increase in ERID prevalence levels include South Africa, Russia, Ukraine, India, China, Brazil. Given that our resources to mitigate the growing crisis in public health are finite, we shall have to pick our spots and allocate funds to disease prevention with discretion in order to maximize the stability of key states and promote regional stability. Examples of such possible interventions might include massive campaigns against the proliferation of HIV in Russia, Ukraine, Brazil, India, China, and South Africa.3

Current statistical trends show that disease-induced mortality is indeed climbing throughout the developed world, and at some future point ERID may constitute a direct threat to political and economic stability in the developed world. However, short of the re-appearance of another rapid and lethal pandemic (e.g. Spanish Influenza), ERID does not present an immediate threat to the stability of states with high initial SC. Therefore, the globalization of disease is not a direct threat to the security of industrialized nations at the present time. However, infectious disease will continue to undermine stability throughout the developing world, compromising key foreign policy concerns of the developed states (such as global political and economic stability), and perhaps contribute to the development of indirect threats to the security of the developed world. Based upon the preceding evidence we can reasonably conclude that the accelerating global proliferation of infectious disease is a security and foreign policy issue area that deserves further analysis.

Given the nature of the global threat to public health, the policy community must both acknowledge the ERID problem, and marshal significant political will to deal with

3 Unfortunately, given South Africa’s soaring HIV and tuberculosis prevalence rates, it may in fact be too late for a successful anti-HIV intervention in that country.
the situation before it deteriorates much further. In both Uganda and Thailand, successful campaigns to limit the spread of HIV were only successful because political elites in those nations used their leadership skills to mobilize both the resources of the governments and aid from external donors. In the absence of informed and committed political leadership, disease continues to spread inexorably. Given the will, policy-makers can enact the required redistribution of fiscal resources, ingenuity, and technology to stem the rising tide of disease and promote global prosperity and stability.

In addition to the mobilization of political will and capital, nascent global ERID surveillance regimes must be strengthened and enlarged. Current systems in place such as ProMED, and the militarized USDOD GEIS system can be geographically enlarged and their information processing capacity augmented. Furthermore, a global ERID containment regime should be designed and built, requiring active cooperation by all states and leadership by the G-7 nations. The G-7 has the economic and technological capacity to take on such a role, and at the past several summits G-7 leaders have expressed growing concern about the proliferation of ERID. Such a regime would require significant cooperation between the majority of states in issue areas as diverse as; increasing the availability of public health data, strengthening local public health capacity, augmenting national and international communication structures, limiting humanitarian disasters and war, and stemming the misuse of antibiotic agents. In particular, global environmental degradation and overpopulation must be addressed and slowed, as these two processes affect viral traffic in their roles as disease amplifiers and probabilistically increase the chance of lethal new zoonoses taking hold within the human
ecology. Such a regime would obviously be extremely hard to both devise and enforce, and will require further study and conceptual development.

An Agenda for Future Research

First, and foremost, the findings of this paper are primarily based on the analysis of quantitative data for the random sample of 20 countries. These preliminary findings provide us with evidence to suggest that increasing pathogen-induced mortality and morbidity within a given country's population will have a negative long-term effect on its State Capacity (SC). However, it behooves us to explore this research in greater depth, and to refine our understanding of the hypothesized relationships. Future studies should try to increase the N, refine the indicators employed, and as appropriate employ different statistical measures.

On the State Capacity side, greater research needs to be done on modeling and quantifying this variable, either by substituting or adding different logical index measures, and perhaps weighting some of the individual measures of SC differently to reflect their logically greater importance within the equation. At this point in time it may be too early to tell which indicators (if any) are the most prominent in the determination of SC, and thus greater research in the form of case studies should precede any attempts at differential weighting. Regarding the independent variable, data on infectious disease prevalence and incidence within given populations are becoming increasingly available at the national and sub-national level for many states. This is particularly true of data on HIV/AIDS. As the data become available this will allow us to perform in-depth case studies of the relationship between ERID and SC within selected countries. This
increasing amount of specific data per pathogen, per country, may allow us to engage in a deeper analysis of the association between the prevalence of various pathogens and State Capacity at the sub-national, national, and regional levels. This will allow us to distinguish pathogens that generate great mortality and/or morbidity in a given population, and have a significant effect on SC (as HIV, malaria, and tuberculosis should), from relatively innocuous or rare diseases that probably result in little harm (e.g. rhinovirus and hantavirus respectively). By determining which pathogens have the greatest negative effect on SC we can target policy to be increasingly effective in mitigating the negative impact of ERID on states and their societies.

The case studies will increase the availability of data on pathogen prevalence and incidence in a given country, and enable the development of a comprehensive database of country specific indicators of SC for as many years as possible. The case studies will also allow us to see how pathogens interact with differentiated societal and political structures across the global population of states. This may provide us with answers as to why certain societies (e.g. Thailand) seem to adapt more effectively to the pathogen threat than others (e.g. the former Zaire), as certain structures of governance may prove more resilient in the face of the ERID threat. These case studies should be carried out on a global basis, drawing on cases from Africa, South Asia, East Asia, Eastern Europe, Latin America and the Caribbean, North America, and Europe. Case studies will also allow us to observe and document the varying strategies that states employ to counter the proliferation of disease within their populations. It is worthwhile to analyze the policy responses of certain countries (e.g. Thailand, and Uganda) which have had some success at curtailing the rise in HIV incidence within their populations, and comparing these
adaptive strategies with those of countries who have decisively failed to adapt to the HIV crisis (e.g. Zimbabwe, South Africa). Such a comparative policy analysis would undoubtedly provide significant value in terms of informing policy prescriptions for countries that are currently experiencing rapid disease proliferation (i.e. Russia, Ukraine, India). The study also points towards further research in that the linkages between State Capacity and political stability must be explored in greater depth, and this inquiry may in turn contribute to the nascent literature on state failure. This project may lead to deeper inquiries into the nature of state survival, power and stability: we should ask the question, under what conditions do states collapse, and how can we explain and predict the phenomena of state failure and intrastate violence?

There is also great interest within environmental policy circles concerning the possible relationship between environmental change, disease prevalence, and social violence at the sub-state level. For example, global climate change may affect variability in rainfall: this will of course in turn affect the levels of vector-borne diseases within certain regions. Following the deprivation hypothesis, increasing human misery and poverty resulting from increased disease levels will in turn generate increased levels of grievance in society that may manifest in terms of random social violence (e.g. theft, assault, homicide) or perhaps as organized violence and resistance against the state itself. I will investigate these linkages in subsequent research projects involving case studies of Kenya, Indonesia, and Brazil. The reciprocal impact of natural and human systems must be explored in greater depth, particularly with respect to their influence upon political outcomes.

---

4 See Holsti, *The State, War, and the State of War*. Also see Migdal, *Strong Societies and Weak States*, and the various works of Jack Goldstone and Thomas Homer-Dixon.
Given the strong empirical association between Population Health and State Capacity, it would seem logical to explore how other factors that induce human morbidity and mortality might also induce governance and deprivation problems. Given that phenomena such as global warming, ozone depletion, and environmental toxicity all compromise population health to a certain degree, one might then explore the possible relations between global environmental change, deteriorating human health, declining State Capacity, increasing deprivation, and social violence. One might also explore the effects of other non-environmental phenomena, such as alcoholism and narcotics-addiction, in the equation as they may also have long-term impacts upon human mortality. Such dynamics are obviously at work within present-day Russia and Ukraine, where falling State Capacity, massive substance-abuse and infectious disease are all feeding off of one another in a negative feedback loop.

Furthermore, other theoretical stressors of SC (such as environmental degradation/scarcity and demographic growth) must be quantified in order to evaluate the strength of their correlations with SC. Given an empirical examination of the effects of these stressors on SC we can evaluate the relative importance of these factors to the health component, and thus determine which of these variables poses the greatest future threat SC, and by extension, state stability. This will allow us to set accurately targeted research priorities and inform the policy-making process as to where we should concentrate scarce resources to meet the most significant threats.

Finally, social scientists must gather empirical evidence on the relative importance of intervening variables (disease amplifiers) in determining the spread and intensity of epidemics and pandemics. By determining which of these amplifiers have the
greatest effects in maximizing the global distribution and/or lethality of ERIDs we can then target policy in order to minimize amplifiers such as the misuse of antibiotics. Of course, reducing the frequency of war, famine, and environmental degradation will inevitably prove more elusive a goal. Finally, a significant amount of work needs to be done concerning the economic productivity and developmental implications of ERID.

This is one of the most important dimensions of this research, and tragically the influence of public health on economic and political development has been almost totally ignored over the years.
Bibliography


Bacon, F. The Advancement of Learning (1605).

--------- Novum Organum (1620).

--------- The Sylva Sylvanum (1626).

Barber, C.V. The Case Study of Indonesia, Occasional Paper, Project on Environmental Scarcities, State Capacity, and Civil Violence. (Cambridge: American Academy of Arts and Sciences and the University of Toronto, 1997).


Castilla, R.E. and D.O. Sawyer, “Malaria Rates and Fate: a Socioeconomic Study of Malaria in Brazil.” Social Science and Medicine, Vol. 37, No. 9, November 1993.


Coleman, W. *Death is a Social Disease: Public Health and Political Economy in early Industrial France*, (Madison WI: Wisconsin Publications in the History of Science and Medicine, 1982).


