ABSTRACT

AUSTIN, KELLY FAWCETT. A New Infection and the Forgotten Diseases: HIV, Tuberculosis, and Malaria in Less-Developed Nations. (Under the direction of Edward L. Kick, Ph.D.)

HIV, tuberculosis, and malaria represent a triple threat to development and well-being in less-developed nations. Despite amazing variation in HIV, TB, and malaria rates across nations, there are few cross-national assessments of the determinants of these diseases. Utilizing insights from neoliberal modernization and world-systems/dependency perspectives, the analyses examine how infrastructural, social, economic, and environmental factors influence HIV, TB, and malaria prevalence in less-developed nations. The results demonstrate the importance of TB and malaria alongside HIV in contributing to heightened cross-national life expectancy inequality, and also illuminate important indirect relationships among environmental decline, urban growth, GDP per capita, health infrastructure, and disease. In addition, environmental degradation associated with export agriculture is found to be a major factor contributing to elevated rates of malaria prevalence. Overall, the results bring attention back to the forgotten diseases of TB and malaria, and demonstrate important ways that development and dependency conditions combine to influence disease outcomes in poor nations, thereby reproducing conditions of global inequality.
A New Infection and the Forgotten Diseases: HIV, Tuberculosis, and Malaria in Less-Developed Nations

by
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DEDICATION

This dissertation is dedicated to my sister Holly, my uncle Brendon, and all others who have left this world too quickly and now live with us in our hearts.
Kelly Fawcett Austin was born and raised in the Santa Cruz Mountains of central California. There she enjoyed a childhood filled with hiking, camping, horseback riding, and exploring in the redwoods and large oaks with her parents and beloved dog, Roxy. During her teenage years Kelly and her parents moved to Ashland, Oregon, where she graduated from high school in 2001. Kelly attended college at Oregon State University, in Corvallis, Oregon. There she met her husband, John Austin, who shares her love for family and the outdoors. In June of 2006 Kelly graduated from OSU with a B.S. in Human Development and Family Sciences and a B.A. in Sociology. In 2007, Kelly and John moved to Raleigh, North Carolina. Kelly earned a Master’s degree in Sociology from North Carolina State University in 2008 and continued her research on global inequalities while pursuing a Doctorate degree. Starting in the fall of 2012, Kelly will be an assistant professor of sociology at Lehigh University in Bethlehem, Pennsylvania.
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CHAPTER 1
DISEASE AND DEVELOPMENT

1.1 Prologue

Despite declining global rates of tuberculosis (TB) and malaria throughout the majority of the 20th century, rates of these “old” infectious diseases have been increasing in many poor nations in recent decades (WHO 2011a; Packard 2009; Farmer 2001). Also, a relatively new infection, HIV or human immunodeficiency virus, is claiming over 2 million lives per year, 98% of which occur in less-developed nations (WHO 2011a). Even with the established links between national disease burden and underdevelopment (e.g. Acemoglu and Johnson 2007; Lorentzen, McMillian, and Wacziarg 2008; Ukpolo 2004), and the stark inequality in disease prevalence rates across nations (WHO 2011a), few studies have examined the cross-national determinants of infectious disease.\(^1\) Additionally, the exiting body of research on human well-being is dominated by methods that use only direct effects modeling approaches (e.g. Brady et al. 2007; Burroway 2010; Scanlan 2010; Jorgenson 2009; MacIntosh and Thomas 2004), which fail to take into account the relevant indirect relationships that characterize cross-national disease trends.

Indeed, the most consistent finding in comparative well-being research is that gross domestic product (GDP) per capita improves health outcomes (e.g. Burroway 2010; McIntosh and Thomas 2004; Scanalan 2010; Pritchett and Summers 1996; Firebaugh and Beck 1994);

\(^1\) For exceptions, see McIntosh and Thomas (2004), Burroway (2010), Scanlan (2010), and Shireliff and Shandra (2011).
however, authors are relatively vague on the specific mechanisms through which national economic growth enhances the health of the people. The consistency of GDP per capita in cross-national models of physical well-being has at least partially contributed to the near-exclusive focus of international development agencies (such as the World Bank) on economic liberalization and market-based approaches to enhance human welfare in poor nations (e.g. World Bank 2008; Davis et al. 2000; Auriol and Picard 2006). In fact, many of these strategies call for the reduction of health services or the privatization of current services to incite economic growth (e.g. Basu et al. 2000). This no doubt is at odds with research and theorization that points to human welfare gains as a means to development, not just an outcome of development (e.g. Sen 1999).

HIV has garnered overwhelming attention in the social and epidemiological sciences, despite the fact that TB and malaria afflict hundreds of millions more, contribute to a comparable level of mortality, and are – for the most part – treatable and preventable diseases (WHO 2011a, 2011b, 2011c; Farmer 2001). Many react to the global HIV pandemic, contending that we could solve the problem with a cure or a vaccine, but the current resurgence in TB and malaria should stand out as robust counter-evidence to that line of thinking. Nevertheless, TB and malaria are rarely thought of as they have been forgotten from public view, largely because these infections have ceased to bother the residents of affluent nations (e.g. Farmer 2000, 2001). Even this may change, however, with the emergence of antibiotic resistant strains of both infections, an issue that will be explored in more detail in following chapters. There are also other new patterns emerging with the neglected diseases; for example
epidemiologists have linked environmental degradation to increases in mosquito populations (e.g. Vittor et al. 2007), and malaria is also beginning to encroach into urban environments in many poor regions (e.g. Donnelly et al. 2005; Pattanayek et al. 2006).

Infectious diseases are acquired through bio-physical processes where viruses cause damage to or upset the human body (Bates et al. 2004). However, disease represents an important sociological issue as patterns in prevalence and mortality predominantly follow lines of international inequality. In fact, it is likely that the resurgence of these diseases is fundamentally tied to issues of persistent poverty in poor nations, including issues with sanitation, clean water access, and environmental decline (e.g. Norris 2004; Bates et al. 2004; Moore et al. 2003). Thus, patterns in disease prevalence are intimately linked to global development dynamics that include economic, social, and environmental realms. HIV, TB and malaria have retreated in certain nations, maintained a steady state in some, and surged forth among others, remaining to be the world’s leading infectious cases of preventable deaths (WHO 2011a; Farmer 2000, 2001; UN 2005). Understanding the underlying causes of comparative disease trends requires investigation of infrastructural, economic, social, and environmental forces that transcend conventional medical or biological reasoning (e.g. Bates et al. 2004; Farmer 2000, 2001; Burroway 2010). Although a disease may be specifically acquired through the bite of a mosquito or an exchange of bodily fluid, a number of structural and ecological characteristics have preceded or conditioned that bite or that exchange, thus making cross-national analysis an appropriate nexus through which to examine infectious disease.
This dissertation is divided into several parts. I will begin in this chapter by putting disease into a comparative context, first by briefly exploring the importance of disease from the standpoint of international public policy, then by thoroughly investigating the theoretical and prior empirical examinations of the relationships between development and human health. Next, I examine the contributions of and regional variations in HIV, TB, and malaria on life expectancy attainment over time. This analysis serves to bring attention back to the forgotten diseases and demonstrates important regional differences in the mortality burden of these infections, which also inform future sampling decisions. In the third chapter, I examine the cross-national factors associated with HIV prevalence, illuminating important interrelationships among urbanization and female empowerment which differ for Sub-Saharan African nations. The forth chapter looks at the causes of the forgotten diseases, modeling TB and malaria together, and finds that health infrastructure, urban slums, and environmental change play important roles in explaining heightened disease rates. The fifth chapter presents a separate analysis of malaria, which focuses on the rural issues associated with prevalence, including environmental degradation and export agriculture. I conclude the dissertation in the sixth chapter, which considers the results within the framework of comparative theories and international policies related to development, the environment, and human health in less-developed nations.
1.2 HIV, TB, and Malaria: A Triple Threat in Less-Developed Nations

HIV, TB, and malaria represent three of the most pervasive and deadly infectious diseases among less-developed nations (WHO 2011a). Although public attention on and health funding for malaria and TB are relatively much lower than for HIV (e.g. MacKellar 2005), the “triple threat” is becoming increasingly recognized by international institutions, as many policy makers acknowledge that HIV, TB, and malaria overlap in their patterns of prevalence and thus are likely to have similar socio-economic, infrastructural, and, in some cases, environmental determinants (e.g. Bates et al. 2004; UN 2005). Indeed, while HIV, TB, and malaria represent different viruses with unique attributes, I predict that their risk or vulnerability characteristics overlap more than they diverge.

The coinciding prevalence among HIV, TB, and malaria is recognized in a recent UN report (2005) titled, “Poverty, Infectious Disease, and Environmental Degradation as Threats to Collective Security,” where HIV, TB, and malaria are explicitly discussed together in the “disease section” as major risks to economic development and human well-being. The report focuses more on HIV than the other two diseases, however, arguing that “the challenge of containing the epidemic [HIV] and preventing new infections rests on advocacy, information and education campaigns, behavior change, condom distribution, and other key interventions.” Issues of poverty and environmental degradation were discussed separately in different areas of the report. Additionally, the UN and the WHO have recently partnered to support the development of a new agency, The Global Fund to Fight AIDS, TB, and Malaria, which has goals to dramatically reduce deaths from these three diseases by 2016 through increased provision of anti-retroviral
therapies (ARTs), TB and malaria antibiotics, and preventative interventions, such as condoms and mosquito bed nets, to people in less-developed nations.

Two things can be noticed by these efforts: (1) International agencies appear to continue to focus on the behavioral or individual-level factors that predispose people to disease, and (2) they continue to treat poverty, environmental degradation, and disease as stand-alone threats to development and well-being. The tactics taken by the UN and the WHO mirror approaches adopted by academic disciplines and scientific research, which focus more on the epidemiology of disease at it applies to biological factors rather than social dynamics. Even within the social sciences, respective fields of demography, economics, and sociology seem to be separately concerned with how sanitation infrastructure or economic growth or gender equality is related to human health, rather than considering the connections among these factors.

Despite these discrepancies, there is overwhelming consistency in the focus on economic development as being the key factor to enhance health and reduce disease in poor nations (e.g. Firebaugh and Beck 1994; Preston 1996; Brady et al. 2007; Burroway 2010; UN 2005). For example, the 2005 UN report conveys that to eradicate disease, “all states must commit themselves to the goal of achieving sustained economic growth.” However, the strategies for pursuing development, as well as the primacy of economic growth over other forms of development in enhancing health, are hotly contested within the comparative sociology literatures. I therefore begin by examining theories of international development and relevant empirical evidence.
1.3 Neoliberal Modernization and World-Systems/Dependency Perspectives on Development

Since its origins (e.g. Spencer 1895; Durkheim 1893), the field of sociology has been concerned with modernity and societal development. While current cross-national analyses most often measure development in terms of economic indicators, such as GDP per capita (e.g. Burroway 2010; Brady et al. 2007; MacIntosh and Thomas 2004; Jorgenson 2009; Shandra et al. 2004), the ultimate goal of development is often articulated as improvements in human welfare (Sen 1999). Although it is important to recognize human well-being as both a means and an outcome to development, most consider development to be the process that improves human well-being. Within the comparative literature, two major perspectives offer insights into the relationship between development and health. The first, modernization theory, emphasizes economic growth as the best strategy to improve human welfare (and therefore reduce disease) in less-developed nations, and is currently enacted through neoliberal approaches that call for market-based strategies to development. The second, world-systems/dependency theory, utilizes political-economy arguments to highlight the persistence of structural inequalities across nations, therefore suggesting that underdevelopment and its symptoms – including infectious disease – will be persistent features of some nations. Although both bodies of research attest that higher levels of development will be associated with lower levels of disease, each perspective has differing assumptions about the appropriate path to or possibilities for development in less-developed nations.
Modernization perspectives adopt both evolutionary perspectives (e.g. Comte 1964; Tonnies 1887; Spencer 1895) and functionalist theory (e.g. Parsons 1951) in the effort to illuminate the dynamics of development in less-developed countries. Under this line of thinking, social change is seen as unidirectional, where societies are hypothesized to reduce levels of infectious disease and enhance well-being as they move from a primitive to an advanced state. Building off the work of Durkheim (1893), modernized societies can be marked by their complex division of labor, which allows goods to be produced efficiently, as each member can specialize in what he does best and then trade with others. Thus, integration into a complex division of labor can lead to overall higher rates of output, benefitting all participating members (e.g. Friedman 2005; Ricardo 1817).

These ideas about the benefits of a division of labor are also applied to entire nation-states, under the doctrine of “comparative advantage” in neoliberal economics. It is assumed that all nations will grow economically through specializing in the production and export of a select number of global commodities (Smith 1776; Ricardo 1817; World Bank 2008). By producing according to natural or institutional comparative advantages, such as tropical nations producing agricultural products and highly-educated core nations producing information technologies, international trade will be extremely efficient and economically benefit all contributing nations (Ricardo 1817).

Under modernization thinking, the relationship between economic growth and enriched human well-being is inherent and natural; it is assumed that increased national economic growth trickles down to households and individuals, providing more resources to address factors that
foster good health (e.g. Firebaugh and Beck 1994; Pritchett and Summers 1996; Preston 1994; World Bank 2008). National economic growth through incorporation into international trade and financial markets also leads to expansions in industry, education, and urbanization, which are shown to promote more healthy behaviors and increase access to health resources, such as physicians (e.g. Soares 2007).

Modernization perspectives argue that tradition is one of the greatest barriers to economic development and enhanced well-being; traditional ways block the employment of advanced production technologies and integration into the economic world-system (Learner 1958). In the context of health, tradition also may prevent the use of medical technologies and interventions, as some societies consider disease to be the result of religious or spiritual forces (Soares 2007). In the 1960s, Rotow (1960) argued that as technologies and Western cultural models that emphasize achievement and progress are diffused around the world, less-developed nations which adopted them would modernize. This continues to be echoed today by those that view globalization, or the movement of industries and technologies to poor nations, as leveling the international development playing field (e.g. Friedman 2005). Additionally, the adoption of more rational and participatory value systems has been associated with higher rates of economic growth (Inglehart and Baker 2000).

Although modernization perspectives articulate development in terms that include cultural and social elements, including expansions in education, urbanization, technology, and instrumental value systems (e.g. Inglehart and Baker 2000; Inkeles and Smith 1974; Learner 1958), there has long been an underlying emphasis on economic growth as the key sign or
outcome of modernization, and thus development is largely conceived in economic terms (e.g. Rostow 1960; Firebaugh and Beck 1994; Friedman 2005). This is perhaps most salient today, as the World Bank (WB), the International Monetary Fund (IMF), and the World Trade Organization (WTO) actively promote trade integration, economic liberalization, and use of advanced technologies as the central means to incite development in poor nations (e.g. Friedman 2005; World Bank 2008). These types of policy prescriptions are often termed “Washington Consensus” or neoliberal models for development, and favor market-based strategies rather than state- or society-based approaches, broadly seeing economic globalization as a “project” that needs to be undertaken in less-developed societies to facilitate development (e.g. McMichael 2004). Trade liberalization is seen as a privileged vehicle for economic growth, where production is restructured around market freedoms rather than domestic needs and conditions (World Bank 2008; Basu et al. 2000). Indeed, many argue that the emphasis on global financial integration signals a new phase of globalization, as scholars demonstrate that the adoption of neoliberal economic policies become especially pronounced in the 1980s and 1990s in the wake of the debt crisis (e.g. Robinson 2004; McMichael 2004; Stiglitz 2002, 2007).

Although the vast majority of nations have implemented policies and provisions in line with neoliberal arguments, many demonstrate that rates of poverty have continued to increase in many developing nations (e.g. World Bank 2011; Stiglitz 2002, 2007). Although some nations such as China and India have achieved notable economic gains, national GDPs have continued to diverge with affluent nations achieving the largest income gains\(^2\) (e.g. Milanovic 2005; World Bank 2008).

\(^2\) It is important to acknowledge that comparative researchers debate the nature of global income inequality; analyses that utilize national-level data tend to show that income inequality between nations has increased over time,
Bank Data 2011). Some of these trends are in fact attributed to the adoption of neoliberal development models, as economic integration may make weak economies more vulnerable to the uncertainties and risks of the international market (e.g. Stiglitz 2002, 2007; Harvey 2006; McMichael 2004; Navarro 2007).

Thus, although neoliberal economic strategies spur short term economic growth, they may compromise human welfare and economic stability over the long term. Much of these critiques are currently being echoed by former Chief Economist of the World Bank, Joseph Stiglitz (2002, 2007), who argues that market-based approaches only concentrate wealth and create trickle-ups to the powerful elite, rather than trickle-down benefits to the masses. While the contributions by Stiglitz (2002, 2007) are fairly recent, the perils of neoliberal development strategies rooted in modernization perspectives have long been emphasized by world-systems and dependency scholars. In fact, the world-systems/dependency school largely arose as a reaction to modernization theory, arguing that development in the world-system actually represents a zero-sum game, where the advancement of core states is enabled through the underdevelopment of periphery states (Wallerstein 1974, 2004; Frank 1979; Amin 1976; Hornborg 2001; Chase-Dunn 1998). Thus depressed levels of human well-being will be a persistent feature of less-developed nations. This is because neoliberal capitalist development allows affluent nations to use less-developed regions as sources of cheap resources and labor,

while other estimates which are weighted by national population or rely on individual-level income data depict decreases in global income inequality over time (see Milanovic 2005 for full discussion). Although the exact nature of international income inequality is contested, statistics clearly illustrate that these forms of economic development and integration have not been successful in providing great reductions to poverty in less-developed nations, and that the global hierarchy of nations has remained largely unchanged over at least the last 50 years (e.g. Stiglitz 2002, 2007; Kick et al. 2011; World Bank Data 2011).
reproducing conditions of underdevelopment in poor nations (Hornborg 2001; McMichael 2004; Bunker and Ciccantell 2005; Amin 1976). The highly stratified system of states that we see today is the historical reflection of unequal exchanges and power asymmetries that formed with the international division of labor during colonial times (Emmanuel 1972; Wallerstein 1974, 2004; Frank 1979; Amin 1976).

The international division of labor describes that developed nations tend to produce high-wage, high-value goods for the world market, while less-developed nations largely produce low-wage, low-value goods (Wallerstein 1974, 2004; Chase-Dunn 1998; Hornborg 2001). Historically, this has been characterized by strong specialization in agriculture and other primary sector activities among periphery nations, and manufacturing and industrial production among more affluent nations. Although the international division of labor is becoming less rigid as secondary and tertiary sector activities spread to less-developed nations, extension of these forms of industry represents a “race to the bottom,” as the most labor-intensive and environmentally-damaging forms of production are those that move to less-developed nations, given the availability of cheap labor and lax or non-existent environmental and labor regulations (McMichael 2004). As the labor costs and profitability of the products that core and periphery nations exchange is unequal, international trade leads to transfers in surplus value from periphery to core states (Emmanuel 1972; Hornborg 2001; Rice 2007). Further, the establishment of agricultural and raw material enterprises throughout Latin America, Africa, and Asia during colonial times has created path dependencies on resource and labor intense production processes, such as raw material extraction, which undermine improvements in

Thus, inequalities in the world-system are constantly reinforced, and economic growth that does occur in poor nations is likely to be extremely uneven, failing to trickle down to benefit the masses. Indeed, technology transfers and market liberalization can often lead to over-urbanization, strains on public resources, reductions in public health spending, and heightened rates of poverty (Kantor and Boswell 2003; Bradshaw 1987; Davis 2006; Harvey 2006; McMichael 2004) – all of which have powerful implications for the prevalence of disease. In short, the world-systems/dependency scholars highlight that GDP per capita does not necessarily translate into improved well-being, it depends on how economic gains are pursued and used. In so doing, this perspective draws attention to broader strategies for development. Priority on economic growth is likely to compromise social and environmental sustainability; the most successful paths to development are going to likely include emphasis on social and environmental welfare as well (Brundtland Commission 1987; Bunker 1985).

1.4 Cross-National Evidence on Development and Physical Well-Being

Although the neoliberal approaches to modernization and dependency/world-systems perspectives have clear differences in their assumptions regarding processes of development and human well-being, GDP per capita proves to be one of the most consistent and robust cross-national indicators of human well-being measures, and the fact that more economically-developed nations have higher levels of human welfare is in large part consistent with both
perspectives. While level of economic development is demonstrated to have clear links to improvements in health, scholars draw on ideas from modernization and dependency perspectives to debate the primacy of economic growth over other factors related to development, such as education or urbanization, as well as the role of dependency in canceling out the beneficial effects of GDP per capita. Most analyses examine well-being measures that include life expectancy, infant/child mortality, and hunger3; it is likely that the developmental factors that influence these dimensions of health also impact disease rates in similar ways.

The current body of research overall demonstrates a positive relationship between per capita GDP and physical well-being measures (e.g. Firebaugh and Beck 1994; Pritchett and Summers 1996; MacIntosh and Thomas 2004; Jorgenson and Rice 2010; Brady et al. 2007; Shandra et al. 2004). Despite this, some researchers continue to question the efficacy of economic growth in improving quality of life outcomes in less-developed nations (e.g. Brady et al. 2007; Wickrama and Lorenz 2002; Soares 2007). This critical body of research includes many proxies for dependency, such levels of debt, foreign direct investment, or percent GDP from agricultural exports, and generally finds that these factors threaten human well-being gains in poor nation (e.g. Wimberley and Bello 1992; Ragin and Bradshaw 1992; Stokes and Anderson 1990; Shandra et al. 2004).

For example, numerous assessments report a negative relationship between trade dependence and physical quality of life, where increasing international exports (especially agricultural and raw material exports) are associated with declines in life expectancy (Wimberley

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3 See for example, Brady et al. 2007; Firebaugh and Beck 1994; Pritchett and Summers 1996; Wimberley and Bello 1992; Ragin and Bradshaw 1992; Shandra et al. 2004.
and Bello 1992; Ragin and Bradshaw 1992). Similarly, existing research also finds that investment dependence, often measured through indicators of foreign direct investment inflows, multinational corporate penetration, and national debt, have harmful effects on life expectancies and infant mortality rates (Shandra et al. 2004; Jorgenson 2009; Wimberley 1990; Bradshaw and Huang 1991; Shen and Williamson 1997). Other studies examine the effects of disarticulation on human welfare (e.g. Stokes and Anderson 1990; Wickrama and Mulford 1996), and find that physical quality of life is negatively linked to level of disarticulation in the economy, with the most pronounced harmful effects found among the poorest of nations (Stokes and Anderson 1990).

In general, the research described above finds both beneficial effects of increased GDP per capita on physical quality of life measures as well as unfavorable effects of dependency on human welfare in less-developed nations. Although these studies only tend to use direct effects modeling techniques, many authors still infer that while GDP per capita does contribute to improved levels of well-being, the avenues under which development is taken, such as increasing exports or multi-national corporate penetration, often produce harmful effects on physical well-being, thus offsetting the gains attributed to increased economic growth (e.g. Wimberley and Bello 1992; Ragin and Bradshaw 1992; Shandra et al. 2004; Jorgenson 2009). Some studies also present opposing findings, such as an analysis done by Firebaugh and Beck (1994), which in contrast to the body of work described above reports that investment dependence does not adversely influence well-being indicators of caloric consumption, infant survival probability, and life expectancy. Some models even depicted positive relationships between exports and physical
well-being measures (Firebaugh and Beck 1994), consistent with the propagations of neoliberal development models.

Some of the discrepancies apparent in this body of research may be attributed to differences in the control variables included, the well-being measures examined, and the modeling techniques employed across studies. In an attempt to address some of the shortcomings of earlier research, Brady and colleagues (2007) conduct a longitudinal assessment of multiple measures (infant/child survival, male/female life expectancy, caloric intake) from 1980-2003, and incorporate a multitude of relevant factors, including educational enrollment, the fertility rate, urbanization, debt service, exports and FDI inflows, thus representing one of the most comprehensive comparative analyses of well-being. Overall, their results illustrate that the fertility rate, secondary schooling, and urbanization have the largest effects on improving well-being in less-developed nations, while GDP has only modest influences when these controls are included (Brady et al. 2007). Perhaps most importantly, their analyses reveal that the positive effects of GDP growth on well-being are decreasing over time, which may indicate that economic growth is becoming increasingly decoupled from tangible improvements in human welfare. In other words, GDP growth may be less effective at improving well-being today than it was in decades past. Brady and colleagues (2007) conclude that increases in women’s health and education may be the best avenue for increasing well-being in less-developed nations.

Many studies in the field of demography also suggest that economic growth may not be the most important factor in improving well-being in less-developed nations (e.g. Soares 2007). For example, Preston (1996) argues that only about 10-20% of the global decline in infant
mortality can be explained by GDP, and other exogenous factors, such as education and sanitation improvements, account for 75-90% of the reduction. More recently, Kenny (2008) finds that less than 1% of the change in life expectancies across nations can be predicted based on income change. China represents a good example, as the rates of income growth in this nation have more than quadrupled from 1980-2000, but life expectancy had only improved by less than 3 years during this time (Kenny 2008).

Rather, improvements in health seem to be more directly tied to gains in education and increasing access to sanitation and clean water (Soares 2007; Riley 2005). Proponents argue that education can be a major vehicle to improved health for many reasons. Education tends to reduce risky behaviors as people become aware of different disease vectors. Schooling can also expose individuals to methods of Western medicine and dispel myths, such as that disease is caused by spirits or religious forces (Soares 2007; Elo 2009). Education is found to be especially important for women, and improving women’s access to education is linked to increased gender equality, a key contributor to enhanced well-being (Culter et al. 2006; Elo 2009; Kuhn 2010; Wickrama and Lorenz 2002; Shen and Williamson 1997; Scanlan 2010). Women tend to be much more vulnerable to infectious diseases in less-developed nations, as they often lack decision-making power or the resources necessary to get medical help (Elo 2009; Wickrama and Lorenz 2002; Shen and Williamson 1997; Scanlan 2010), thus increased gender equality and women’s empowerment tend to improve overall measures of well-being as women’s health increases. Female empowerment and education is already shown to be an especially important factor in reducing HIV prevalence (Scanlan 2010; Shircliff and Shandra 2011). Fundamentally,
education also provides people with a means of improving incomes and finding less dangerous types of employment (Elo 2009).

While the importance of education and female empowerment are common themes across both the sociological and the demography literatures, issues of sanitation and water access are almost exclusively considered in demographic research. Sanitation and clean water can improve well-being by reducing risks for diseases, as contaminated water is a major disease vector (Soares 2007; Moore et al. 2003). Despite developmental gains, access to sanitation is a continuing problem in less-developed societies, as a recent report by the WHO (2010) states that over 2.5 billion people in the world continue to lack adequate sanitation, with over 1.1 billion defecating out in the open. Indeed, education, female empowerment, and sanitation are expected to be important outcomes of modernization and economic growth, but dependency and neoliberal development schemes may limit the efficacy and scope of these interventions as social services become privatized and de-prioritized in favor of market liberalization (e.g. Navarro 2002, 2007; McMichael 2004).

Issues of sanitation and clean water are often considered alongside urbanization (e.g. Moore et al. 2003). Urbanization is commonly employed as an indicator of modernization, given that expanding education, technology, and industry promote the development of cities (e.g. Learner 1958), and moving into cities often increases access to medical services and employment that improve health (e.g. Brady et al. 2007; Soares 2007). However, many less-developed nations are experiencing very rapid urban population growth, leading to high rates of urban poverty and the proliferation of urban slums (Culter et al. 2006; Moore et al. 2003; Davis
Over-urbanization can be deeply tied to dependency dynamics, as this often represents a surplus population that has been displaced due to global economic shifts or issues of environmental degradation. For example, investment dependence contributes to land privatization, increased use of non-human technology, and expansions in low-wage factory work, which can all contribute to the emergence of an urban underclass (e.g. McMichael 2004; Davis 2006; Bradshaw 1987). In addition, rural displacement due environmental degradation and climate change represent important “push” factors of urban migration in certain areas (Hunter et al. 2011; McMichael 2006). As poor nations have increased dependence on agricultural production due to the structure of the world economy, urbanization which results from various forms of environmental decline reflects world-system theorizing.

Health interventions are also a common theme within the literature. While medical interventions have been important in contributing to gains in health historically, many are skeptical about the potential of vaccines and other health resources to impact levels of infectious disease in poor nations today (e.g. Riley 2005, Soares 2007; Kenny 2008; Elo 2009; Kuhn 2010). One reason is that support for non-HIV related global health interventions have decreased in the most recent years, with average levels of international funding for health campaigns (including TB vaccinations) decreasing over the previous decades (MacKellar 2005). Medical researchers acknowledge that there is low investment in life-saving malaria vaccines or antibiotics, as it is much more profitable to support the development of interventions for the ailments that plague people in affluent nations, including cancer and heart disease (Trouiller et al.
The regulation of patents and other agreements also prevents the dissemination of some medical interventions to poor nations (Navarro 2002).

Additionally, many point out that past health campaigns have already successfully targeted the easiest to reach segments of the population in developing regions (MacKeller 2005; Seckinelgin 2006; Cutler et al. 2006). On top of these issues, less-developed nations tend to have low levels and quality of health infrastructure, including doctors and hospitals, limiting the availability and success of treatments and preventative care. These dynamics are clearly linked to the dependency themes echoed above, as many rapidly growing economies have not provided a good public health environment or have moved away from it in their attempt to promote economic growth and adhere to neoliberal development models (Cutler et al. 2006; Harvey 2006; Stiglitz 2007; Navarro 2002, 2007).

Health interventions and technologies were certainly relevant in past decades, as the 20th century was marked by a series of “health transitions” which led to persistent improvements in average national life expectancy in all countries (Riley 2005; Soares 2007). Indeed, this time period was marked by decreasing inequality in life expectancy across nations (Riley 2005; Goesling and Firebaugh 2004; Mazumdar 2001; Cutler et al. 2006), as health technologies and

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4 Related to this, some studies have examined the influence of national inequality on health outcomes. The findings have been mixed, with many showing non-significant effects of economic inequality (GINI score) on life expectancy and infant mortality (e.g. Beckfield 2006; Brady et al. 2007). Others show that inequality has harmful effects on health outcomes in certain regions or with certain measures (e.g. Biggs et al. 2008; Cornia et al. 2009). In each of the following analyses, I tested for the effects of inequality using GINI estimates. Across all models, this indicator was not significant in predicting the disease outcomes; it was thus eliminated from the results presented here. Substantively, while economic inequality was not demonstrated to be an important factor predicting disease, it is likely that inequality in access to health resources is important, as the following chapters illustrate the importance of schooling, sanitation, and doctors in explaining cross-national patterns in disease. Additionally, relative deprivation may not be as important as absolute deprivation in explaining disease, as the absolute deprivation measures employed here (e.g. percent access to improved sanitation) are quite robust in predicting disease.
knowledge spread first throughout Europe and North America, then to developing regions in the 1940s-70s. In fact, it was during the middle of the century that TB and malaria were successfully eradicated from the United States, and shortly thereafter, interventions reduced TB and malaria rates in developing regions as well (Soares 2007; Farmer 2000). Also during this time many poor nations made significant gains in GDP and health infrastructure, such as sanitation, which allowed many less-developed nations to “catch up” to comparable levels of life expectancy achieved in developed nations (Riley 2005; Kuhn 2010). Some researchers are cautious in noting that although the world-system is historically marked by convergence in national life expectancies, there is significant variation in improvements, with certain nations continuing to lag behind (Mazumdar 2001; Kuhn 2010).

In the 1990s something unexpected happened – there was a sudden shift in national life expectancy trends, with inequality in average life expectancy across nations beginning to increase rapidly. The bulk of this divergence can be attributed to the poorest strata of nations, including those in Sub-Saharan Africa, as some have experienced actual life expectancy declines over the last few decades (Neumayer 2004; Riley 2005; Soares 2007; Cornia et al. 2009; Kuhn 2010; Cutler et al. 2006). For example in Zambia, the average life expectancy has decreased by 20 years since 1988. Indeed, the greatest inequality in life expectancy is among periphery nations themselves; research by Mazumdar (2001) illustrates that the standard deviation for life expectancy among high and middle income nations decreased from 1960-2000, but increased significantly over this time for the lowest income nations.
Many studies thus attribute life expectancy divergence to the advent of the AIDS pandemic, given the extreme levels of HIV/AIDS in Sub-Saharan Africa (Neumayer 2004; Riley 2005; Soares 2007; Kuhn 2010; Goesling and Firebaugh 2004). However, this assertion has not been empirically tested with proper controls, nor is there recognition that mortality from TB and malaria could also be contributing to life expectancy divergence among Sub-Saharan African nations. The forgotten diseases of TB and malaria have been forgotten by social scientists as well who attempt to explain growing cross-national life expectancy inequality. As life expectancy is most often promoted as the key indicator of well-being (e.g. Mazumdar 2001), examining the contribution of TB, malaria, and HIV to life expectancy rates represents an appropriate starting point for an investigation of infectious disease patterns in less-developed nations.

In addition to this, review of the research demonstrates that little is known about the cross-national or structural determinants of disease (e.g. Bates et al. 2004). Numerous studies have examined development and dependency effects on life expectancy (e.g. Brady et al. 2007; Bradshaw and Huang 1991; Firebaugh and Beck 1994), infant and child mortality (Shandra et al. 2004; Jorgenson 2009; Firebaugh and Beck 1994; Ragin and Bradshaw 1992), and to a lesser extent hunger (e.g. Wimberley and Bello 1992; Brady et al. 2007), but comparative studies of the causes of disease are almost completely lacking. Although epidemiological studies draw relevant links between environmental degradation and malaria (e.g. Vittor et al. 2009) and urban poverty and TB (e.g. Antunes and Waldman 2001) in individual-level studies, these patterns have not been analyzed cross-nationally, nor have the broader connections to international development dynamics been made.
1.5 Emerging Questions about HIV, TB, Malaria, and Well-Being in Less-Developed Nations

Considerations from neoliberal approaches to modernization and world-systems/dependency perspectives provide key insights into the developmental factors that are likely to promote disease in less-developed nations. However, these theorizations, as well as prior empirical examinations of human well-being, point to areas of research that demand further attention. It is essential to first demonstrate the importance of considering the neglected plagues of TB and malaria alongside HIV regarding issues of disease and development in less-developed nations. The recent patterns in cross-national life expectancy divergence are significant as they perhaps signal a new era of global health declines (at least among some nations), and emerging patterns with TB and malaria need to be brought into this discussion. I predict that TB and malaria prevalence are also contributing to growing inequality in cross-national life expectancies, alongside HIV, and that the effects of the forgotten infections may perhaps worsen over time with new patterns in antibiotic resistance and the potentials of anti-retroviral therapies to extend the life of HIV-infected individuals.

The broader concern of increasing incidence of HIV, TB, and malaria among poor nations also necessitates investigations into the cross-national determinants of these diseases. Current investigations of physical well-being are centered on measures of life expectancy and infant mortality, rather than the preventable causes of these deaths. In general, this body of work discounts or ignores other relevant factors and arguments in favor of the robust effects of GDP per capita. Additionally, while it is argued that GDP gains lead to improvements in
education or urbanization, these are most often set up as competing predictors, rather than assessing the indirect relationships among economic and non-economic factors that promote health. Other relevant variables, such as access to sanitation, are excluded due to issues with multicollinearity or a lack of interdisciplinary integration. Environmental factors have been almost completely absent from the global health literature, despite the probability that ecological degradation may influence population flows and proliferate certain disease vectors. I improve upon these shortcomings by modeling both direct and indirect effects on disease and utilizing latent constructs to estimate more comprehensive and inclusive models of HIV, TB, and malaria prevalence in less-developed countries. This requires the adoption of an interdisciplinary approach, as predictors commonly used in sociology, demography, economics, and the natural sciences are relevant to cross-national patterns in disease.

Infectious diseases have plagued the world-system since its onset; debilitating illness and resulting patterns in premature death have long contributed to underdevelopment in less-developed regions. Indeed, colonial domination by core powers was in large part enabled by the spread of disease to indigenous groups, eliminating entire populations that hampered European hegemony (e.g. Diamond 1997). The implications for the reproduction and exacerbation of global inequality in the current era are clear, given the well-established effects of disease on economic growth. HIV, TB, and malaria represent important dimensions of development that deserve sociological scrutiny.
CHAPTER 2
CROSS-NATIONAL LIFE EXPECTANCIES AND HIV:
THE FORGOTTEN ROLE OF MALARIA AND TUBERCULOSIS

2.1 Introduction

Infectious diseases represent the leading cause of death in less-developed nations, as communicable illnesses account for more deaths annually than non-communicable diseases, injuries, and hunger combined in poor countries (WHO 2011a). Scholarly research and public attention on disease has been devoted to the HIV/AIDS crisis, despite that many less-developed regions are experiencing a resurgence of other deadly infectious diseases, such as malaria and tuberculosis (TB). In fact, in Sub-Saharan Africa rates of tuberculosis infection have increased by over 30% and malaria prevalence has more than doubled since 1990 (WHO 2011a). Data on mortality also suggests that malaria and tuberculosis can be just as deadly, as these two diseases produce over one million more deaths per year than AIDS (WHO 2011a). Although malaria and tuberculosis are often “forgotten” from public view (Farmer 1999, 2001), these diseases pose continuing – and perhaps mounting – threats to well-being in poor regions.

TB and malaria afflict hundreds of millions of people, are especially deadly among young children and infants, and new trends in antibiotic resistance threaten the ability to treat these infections (WHO 2011b, 2011c); however, the effects of the “forgotten diseases” on cross-national life expectancy trends are indeed being completely neglected. Social scientists note that

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the sudden “turn-around” in life expectancy gains represents one of the most surprising demographic shifts of the current era, as cross-national life expectancies have been diverging since about the 1980s after over a century of life expectancy convergence (Neumayer 2004; Riley 2005; Soares 2007; Kuhn 2010; Goesling and Firebaugh 2004). The HIV pandemic is singled out as the cause of increasing international life expectancy inequality, despite that TB and malaria are also concentrated in the nations showing life expectancy declines (WHO 2011a). In other words, many of the robust findings for HIV prevalence on life expectancy inequality may be spurious, due to the embedded effects of TB and malaria. Exposing TB and malaria’s potential contribution to life expectancy declines would greatly aid in bringing attention back to the neglected diseases.

Although current studies tend to focus on the individual or biological causes of virus transmission, disease represents an important sociological issue as patterns in prevalence and death follow broader trends in global inequality. Poverty is a central risk factor for both acquiring and dying from disease (e.g. Farmer 2001), and poor nations have the lowest levels of life expectancy in the world-system (UN 2005; Mazumdar 2001; Kuhn 2010). Malaria, TB, and HIV are extremely rare among affluent nations, but represent the world’s leading infectious cases of preventable deaths in poor regions (WHO 2011a; Farmer 2000; UN 2005). As malaria and tuberculosis are curable and treatable diseases, and much less costly to address than medical interventions HIV/AIDS (e.g. MacKellar 2005), examining their contributions to life expectancy rates can help to direct public policy to the most appropriate and effective areas of need.
Comparative studies most often examine how development and dependency factors, such as GDP growth, education, urbanization, debt, and exports, are linked to life expectancy rates in less-developed countries (e.g. Brady et al. 2007; Shandra et al. 2004; Firebaugh and Beck 1994; Wimberley 1992; Ragin and Bradshaw 1994). A new line of research has also identified HIV rates in Sub-Saharan Africa as the cause of increased international life expectancy inequality (Goesling and Firebaugh 2004; Neumayer 2004). This chapter builds on these areas of research by examining the influence of malaria, TB, and HIV on life expectancy over time alongside prominent development and dependency factors, utilizing panel data from 1990-2009. In addition, analyses test for regional variation in the influence of disease on life expectancy, as well as comparing the magnitude of the effects of malaria, TB, and HIV on life expectancy rates across less-developed nations over time. I begin by briefly reviewing key insights from theorizations on modernization and dependency, followed by a discussion of the characteristics of malaria, TB, and HIV in less-developed nations.

2.2 Development, Life Expectancy and Disease: Theories and Research

Development has long been a theme within the discipline of sociology (e.g. Spencer 1895), and improvements in human well-being are generally seen as a key outcome or goal of development (Sen 1999). Human well-being is a multi-dimensional phenomenon, and thus, the measurement of human well-being is complex and controversial. There are many different views regarding how to best measure well-being, however life expectancy is often accepted as the major contributor to or indicator of well-being (e.g. Mazumdar 2001; Soares 2007; Riley
2005). Indeed, how long members of a society on average live reveals much about their overall quality of life.

Developmental theories clearly explain that life expectancy improves as societies modernize. However, processes of modernization are hotly debated in the field of global sociology, with some emphasizing that all nations can modernize (e.g. Rostow 1960; Friedman 2005) and others suggesting that international development represents a zero-sum game, where some nations are developed because other nations have remained poor (e.g. Frank 1979; Hornborg 2001). As was discussed in the prior chapter, neoliberal approaches to modernization draw attention to long-standing assumptions regarding economic growth and the division of labor in societies, arguing that increased economic integration will spur economic growth and therefore life expectancy gains in poor nations. Within this paradigm, the positive relationship between economic growth and measures of physical well-being is inherent and natural; increased economic productivity trickles down to enhance the purchasing power of citizens, providing more financial resources for factors that would improve life expectancies, such as proper housing, schooling, health care, and food security (e.g. Firebaugh and Beck 1994; Pritchett and Summers 1996).

In comparison to modernization perspectives on development, the prior chapter also explored how dependency/world-systems scholars are more skeptical about the role of economic growth and integration in poor nations. Many forms of economic globalization increase dependency and poverty in poor nations, as low-wage, dirty industries and technologies are those that spread out of the core to developing regions (e.g. McMichael 2004; Wallerstein
1974, 2004). Thus, economic growth does not necessarily translate into improvements in life expectancy; it depends on how it is achieved and used. Although the modernization and dependency/world-systems perspectives draw attention to relevant factors that influence life expectancy gains in poor nations, these indicators have been lacking from current demographic assessments of the effects of HIV on cross-national life expectancies.

Empirical work examining development and dependency influences on life expectancy rates provides partial support for both lines of theory. Economic growth, expansion of education, and rising urbanization are indicators of modernization that have been positively linked to improvements in life expectancy rates cross-nationally and over time (e.g. Brady et al. 2007; Soares 2007; Wickrama and Lorenz 2002; Firebaugh and Beck 1994; Pritchett and Summers 1996). There is also evidence that dependency is negatively associated with human well-being, as many find that export dependence, specialization in agriculture or raw materials, some forms of foreign investment, and mounting debt reduce physical well-being gains in less-developed nations over time (e.g. Jorgenson 2009; Shandra et al. 2004; Brady et al. 2007; Ragin and Bradshaw 1992). This is consistent with findings from demography, which have found that neoliberal or “Washington Consensus” provisions are slowing down the pace of life expectancy improvements over time in less-developed nations (e.g. Navarro 2002, 2007). For example, Cornia and colleagues (2009) emphasize the role of increasing income inequality, trade liberalization, and neoliberal development policies that de-prioritize social services in contributing to global health declines. In general, many assessments argue that the emphasis on GDP per capita gains may be overstated, as analyses demonstrate that education, access to
medical care, and fertility declines have been the most influential variables in improving life expectancy rates across nations and over time (e.g. Brady et al. 2007; Soares 2007; Riley 2005; Wickrama and Lorenz 2002).

Indeed, as previously suggested, the most recent decades have been marked by increasing inequality in life expectancy across nations, where the bulk of this divergence can be attributed to the poorest strata of nations, Sub-Saharan African nations, which have experienced actual life expectancy declines over the last few decades (Neumayer 2004; Riley 2005; Soares 2007; Cornia et al. 2009; Kuhn 2010; Cutler et al. 2006). Many studies attribute global life expectancy divergence to the advent of the HIV pandemic, which increases mortality rates for young and working-age adults, thus having a powerful impact on national life expectancy scores (Neumayer 2004; Riley 2005; Soares 2007; Kuhn 2010; Goesling and Firebaugh 2004). In fact, several studies show that when Sub-Saharan African nations are excluded from the analyses, life expectancy scores continue to converge through the 2000s (e.g. Soares 2007). However, a major shortcoming in this line of research is that the relationship between HIV prevalence and life expectancy is not tested with the development and demographic predictors commonly used in comparative assessments of well-being (such as education, urbanization, fertility, exports, debt etc). In addition, given the concentration of other deadly infectious diseases in Sub-Saharan Africa, including TB and malaria, it is possible that too much emphasis is being placed on HIV in explaining life expectancy declines.

Partial support for this line of thinking comes from an article examining patterns in international health assistance. MacKellar (2005) finds that international development assistance
for health has gone up considerably in recent decades, however the bulk of these resources are
devoted to the HIV/AIDS crisis. In fact, when funding for HIV/AIDS issues is removed from
the calculations, MacKellar (2005) finds that aid for health decreased as a proportion of total
development assistance. Thus, public health funding apart from HIV/AIDS has actually
declined over time in less-developed nations. These findings stand in stark contrast to disease
statistics on disability-adjusted life years, which demonstrate that communicable diseases apart
from HIV and other sexually transmitted diseases account for around 40% of the disease
burden, while sexually transmitted diseases including HIV account for only 5.8% of mortality in
less-developed nations (MacKellar 2005).

One argument to be made is that becoming infected with TB or malaria does not
necessarily result in death, thus resources are being disproportionately allocated to the most
severe or deadly infection. However, the effect of each of these diseases on life expectancy,
alongside relevant developmental controls, remains to be tested, and it is possible that TB and
malaria could have more pernicious effects on cross-national life expectancy rates than HIV,
given their elevated levels of prevalence, contributions to mortality particularly among children
and infants, and new patterns in antibiotic resistance which are likely to result in higher levels of
TB and malaria deaths in the most recent decades. An alternative, critical point of view on
health funding allocations is that tuberculosis and malaria have effective preventative treatments
(immunization for TB, bed-nets and insecticides for malaria) and can often be cured with
antibiotics (e.g. Bates et al. 2004), thus public funding for disease should be directed toward the
deadly diseases that can be addressed most successfully.
The development theories and existing empirical assessments point to important structural factors which are likely to impact life expectancy rates alongside disease in less-developed nations, such as GDP per capita, education, fertility, medical resources, debt, and participation in international trade. Although many infectious diseases may have significant ramifications for life expectancy, this is rarely considered beyond the impact of HIV/AIDS (e.g. Goesling and Firebaugh; Neumayer 2004). Indeed, many of the features of the HIV pandemic, including its scope, the characteristics of vulnerable populations, and patterns in transmission, cause it to be considered the key mechanism explaining heightened global life expectancy inequality. However, these attributes are also similar to the characteristics of TB and malaria, and it is possible that the overwhelming focus on HIV by social scientists and international institutions (e.g. World Bank) alike has contributed to a lack of consideration to these neglected diseases. Many of the robust findings for HIV on life expectancy inequality may be spurious, due to the embedded effects of tuberculosis and malaria. I now turn to a more detailed discussion of TB, malaria, and HIV, demonstrating that both this “new” pandemic as well as the “forgotten” diseases are likely to be combining to contribute to debilitating trends of life expectancy attainment across poor nations over time.

2.3 Tuberculosis, Malaria, and HIV: A Triple Threat to Life Expectancy Rates

Tuberculosis and malaria represent two of the most prominent deadly diseases of the developing world, as hundreds of millions of people worldwide suffer from these infections each year (WHO 2011a). Although scholarly attention to disease is largely restricted to the deadly
impact of HIV/AIDS, TB and malaria have similar patterns in vulnerability and transmission, and are also major contributors to premature death among the young and working-age adults. One thing that makes tuberculosis and malaria distinct from HIV is that the former are (for the most part) treatable and curable diseases. While AIDS research is advancing and use of anti-retroviral therapies (ARTs) which can prolong the life of infected HIV patients are spreading, these drugs still have limited use in poor nations due to their high cost and restricted availability of cheaper, generic varieties (Heimer 2007; UNAIDS 2009). Nonetheless, ARTs do not cure or prevent HIV, and the fact that TB and malaria have the potential to be treated with effective antibiotics makes their probable contributions to premature death especially discouraging.

Human immunodeficiency virus (HIV) is a retrovirus as it replicates host cells, and infection of HIV occurs during the transmission of human body fluids, most commonly through blood, semen, vaginal secretions, and breast milk (Barnett 2004; Heimer 2007; Garnett et al. 2001; UNAIDS 2009). The first case of HIV was officially detected in the United States in 1981, yet over 95% of HIV-infected individuals today are located in less-developed nations (UNAIDS 2009). While the HIV/AIDS crisis is a relatively new phenomenon, TB and malaria have afflicted human societies throughout history. In fact, TB was a leading cause of death globally in the 18th and 19th centuries (Farmer 2000; Gandy and Zamula 2002). The tubercle bacillus, or the TB virus, is a contagious respiratory infection that may be transmitted through sustained or intimate contact (WHO 2011b; Kim et al. 2005). Viable bacilli are also aerosolized by coughing and may remain in the air for several hours. Over one-third of people worldwide have Mycobacterium tuberculosis (M-TB), the latent organism that causes the TB virus (WHO 2011b;
Kim et al. 2005). M-TB does not cause active disease in all who are infected; among that fraction of the infected who do fall ill, however, the majority live in poverty (Kim et al. 2005; WHO 2011b; Gandy and Zamula 2002).

Malaria is distinct from the other diseases examined here in that it is a parasitic infection transmitted by mosquitoes. The malaria parasite enters the human host when an infected Anopheles mosquito takes a blood meal (WHO 2011c). Inside the human host, the parasite undergoes a series of changes as part of its complex life-cycle, which allows the plasmodia parasite to evade the immune system, infect the liver and red blood cells, and finally develop into a form that is able to infect a mosquito again when it bites an infected person (WHO 2011c; Sachs and Malaney 2002). Malaria produces symptoms of headache and fever, and can progress quickly into death as the parasite attacks red blood cells, causing comas and neurological abnormalities. In fact, many people who survive an episode of severe malaria may continue to suffer from neurological disorders, producing potential negative influences on life expectancy even among those who are “successfully” treated (Sachs and Malaney 2002; Pattanayak et al. 2006). Approximately 3 billion people who live in tropical regions endemic to Anopheles mosquitoes are at risk of acquiring malaria (WHO 2011c).

Although each of these infections is biologically distinct, HIV, TB, and malaria are all highly associated with poverty. Sub-Saharan Africa is the poorest region of the world-system, and houses 68% of global HIV cases, 30% of global TB cases, and 80% of global malaria deaths (WHO 2011a). Southeast Asia also has elevated disease prevalence; in fact rates of malaria are increasing most rapidly in this region, and Southeast Asia is home to over 35% of global TB
cases. Although relatively few cross-national studies of infectious disease exist, those that do clearly demonstrate that prevalence of HIV, TB, and malaria has been linked to lower rates of national economic growth (e.g. Garnett et al. 2001; Dixon et al. 2001b; Ukpolo 2004; Lozentzen et al. 2008; Acemoglu and Johnson 2007; Packard 2009; Sachs and Malaney 2002). Thus, not only are these diseases caused by poverty, but they also further contribute to it. These assessments find that HIV, TB, and malaria are likely to impede national economic growth by elevating pressure on medical resources, increasing the number of orphaned children, creating prolonged absences from work, lowering worker productivity, decreasing household incomes, and stimulating higher household medical expenses (e.g. Barnett 2004; Heimer 2007; Lozentzen et al. 2008).

Poverty is the key risk factor for contracting infectious diseases as poor people are less likely to use disease-prevention strategies (such as condoms or insecticide-treated bed nets), have limited access to medical care, live in crowded or unsanitary conditions, have low levels of education, and engage in work-migration patterns that facilitate transmission (e.g. Bates et al. 2004; Burrawoy 2010; Farmer 2000; Heimer 2007; Gandy and Zamula 2002; Pattanayak et al. 2006; MacIntosh and Thomas 2004). Less-developed nations also tend to have inadequate health care systems, which promote unsatisfactory interventions, often through a lack of antibiotics or other medical resources. Confounding this problem, poor people are less able to mobilize resources rapidly, so they often delay seeking treatment, which is a major obstacle as interventions for TB and malaria are most successful in the earliest stages of infection (Bates et al. 2004; Kim et al. 2005).
Besides persistent poverty in less-developed nations, many researchers note that increasing prevalence of TB and malaria in recent years may be due to the emergence of antibiotic resistant strains of these infections (Pattanayak et al. 2006; Bates et al. 2004; Kim et al. 2005). As antibiotic resistance develops when patients are exposed to sub-optimum concentrations of antibiotics, it is likely that the emergence of antibiotic resistant strains of both infections is linked to low quality health care and issues of poverty that prevent people from adhering to costly treatments in poor regions (e.g. Bates et al. 2004). Antibiotic resistance means that TB and malaria will be increasingly difficult to treat, and could also prompt increases in mortality from these diseases.

Rising rates of TB infection have also been linked to the HIV pandemic, as around 9% of all new TB cases in adults globally are attributable to HIV infection (WHO 2011b). In Sub-Saharan Africa, this association is even stronger, as the proportion of new TB cases in HIV patients is around 30% in this region. There was an estimated 1.8 million deaths from TB in 2010, of which 12% are attributable to HIV (WHO 2011b). People who develop latent M-TB typically have a 5-10% chance of developing active TB at a later point in their lives; however for HIV-infected individuals, this probability increases to around 50% (Kim et al. 2005). HIV is a potent risk factor for TB, as well as other infections including malaria, due to comprised immunity (Corbett et al. 2003; Gandy and Zamula 2002; Antunes and Waldman 2001). Medical research also notes that HIV-infected TB patients are much more difficult to treat effectively and may have many reoccurrences, or have the disease progress into mortality very quickly.

Increasingly, malaria is also becoming a factor in the transmission of HIV, as patients with severe malaria often require blood transfusions, and a significant portion of the blood supply in Sub-Saharan Africa is infected with HIV (Sachs and Malaney 2002). Clearly, there is much overlap between HIV, TB, and malaria, due not only to biological processes related to immunity and treatments, but also with poverty being a major contributor to the transmission of these infections (Bates et al. 2004). Although the HIV pandemic is singled out as the cause of increasing inequality in cross-national life expectancy estimates, it is clear that TB and malaria are deadly diseases that also deserve far more scholarly and public health attention.

2.4 Hypotheses

To explore how disease and development dynamics influence cross-national life expectancy rates over time, I employ random effects panel models on a sample of less-developed nations. (H1) I predict that HIV, TB, and malaria all have negative influences in life expectancy rates in less-developed nations over time, net of the influence of pertinent development and dependency predictors, given the similar patterns in vulnerability across nations to each of these diseases. Although researchers have recently emphasized the harmful influence of HIV on national life expectancies, this research has neglected to control for prominent developmental factors that are also associated with national life expectancy rates. Additionally, the influence of
TB and malaria on national life expectancies have been completely neglected, despite that these diseases could also account for emerging life expectancy divergence trends.

(H2) I also predict that the effects of disease on life expectancy vary significantly by region, as Sub-Saharan Africa and Southeast Asia have elevated rates of disease and are also among the most impoverished of nations, likely contributing to altered life expectancy trends in these regions. I will also make comparisons across the diseases, as it is possible that TB and malaria are responsible for comparable influences on life expectancy rates to HIV in less-developed nations.

I will first examine how HIV, TB, and malaria each influence life expectancy estimates alongside many of the most common predictors of well-being used in macro-comparative analyses. Following this, I investigate a second set of models which include pertinent regional interaction terms, for Sub-Saharan African and Southeast Asian nations. The last set of analyses allows for more careful comparison of the magnitude of the effects for disease over time in less-developed nations.

2.5 Methods

**Sample.** Many studies demonstrate that the determinants of life expectancy vary significantly across less-developed and affluent nations (e.g. Mazumdar 2001), and, by and large, infectious diseases such as HIV, TB, and malaria are rare in developed regions (WHO 2011a). Thus, the analyses are restricted to less-developed nations, which excludes the highest quartile of
nations based per capita GDP.\textsuperscript{6} The analyses include 118 less-developed nations, with data observations for the independent variables taken at 1990, 1995, 2000, and 2005; the data observations for the dependent variable, life expectancy, are measured at 1994, 1999, 2004, and 2009 to capture time lag between acquiring disease and mortality.\textsuperscript{7} The nations included in the sample are presented in Table 2.1. Considering the country-year as the unit of analysis, the total number of cases analyzed in each model varies from 471 to 176, depending on missing observations. To be included in the analyses, a nation must have data for at least 2 time points for predictor variables, and all time points for the dependent variable, life expectancy.

\textbf{Analytic Strategy.} Within the field of macro-comparative health research, heterogeneity bias is a major concern. Heterogeneity bias refers to the confounding effect of unmeasured variables that are omitted from the regression analyses (Frees 2004). Fixed and random effects models are often utilized to correct for the issue of heterogeneity bias, and have gained prominence as a methodological technique in areas of global sociology and health in recent years (e.g. Brady et al. 2007; Beckfield 2006; Jorgenson 2009). The major difference between fixed and random effects models is that fixed effects models treats non-inclusion error as fixed and estimatable, making no assumptions about the correlation between unobserved effects and the observed independent variables. Conversely, in random effects models error is randomly distributed and omitted time-invariant variables are assumed to be uncorrelated with

\textsuperscript{6} This is consistent with how prior researchers in this tradition have defined less-developed nations; see for example, Brady et al. 2007; Jorgenson 2008; Shandra et al. 2008.

\textsuperscript{7} Although HIV, TB, and malaria may progress into death at different rates, most notably with HIV taking at least a few years to cause death, while mortality from TB and malaria tend to be more rapid, data limitations preclude a 4-year time lag between disease and life expectancy. Preliminary assessments that included fewer time points for a larger time lag (capturing the unique dynamics of HIV) produced consistent results.
the included indicators (e.g. Hsiao 2003; Frees 2004). Although fixed effects models can offer a more stringent analysis, as error terms may often be related to the independent variables, random effects models have the advantage of greater efficiency relative to the fixed effect model leading to smaller standard errors and higher statistical power to detect relationships, which is especially relevant to cross-national data (Hsiao 2003). In addition, random effects models incorporate variation both between nations and within them, accounting for essential cross-national and historical factors. Fixed effects models only account for within-country, over time variation (Hsiao 2003; Frees 2004). As both the cross-national and historical variation is relevant to the topics under investigation, the models should allow the predictors to explain both types of variation. A Hausman test can discern if the fixed or random effects estimates are more appropriate, as it compares the fixed versus random effects under the null hypothesis that the individual effects are uncorrelated with the other regressors in the model (e.g. Hausman 1978). Non-significant findings for the Hausman test across the models presented here allow for the interpretation of random effects models.

In this study I utilize STATA (version 10) to estimate generalized least squares random effects models with robust standard errors. All models include unreported period-specific intercepts, which controls for the potential unobserved heterogeneity that is invariant within years. In addition to the predictors listed below, year is included in the models to control for changes in life expectancy rates over time, and to guard against spurious associations among omitted variables with common trends. Year is coded as follows: 1 (1990), 2 (1995), 3 (2000), and 4 (2005).
Dependent Variable. The dependent variable under investigation is total life expectancy, which measures the average number of years that a newborn baby is expected to live if the age-specific mortality rates effective at the year of birth apply throughout his or her lifetime. Life expectancy is considered to be a central dimension of human well-being (e.g. Mazumdar 2001), and this study seeks to build upon prior arguments regarding the role of HIV in facilitating patterns of global life expectancy divergence. Life expectancy data were obtained from the World Bank Database (2011).

Key Independent Variables. Key independent variables include HIV prevalence, TB prevalence, and malaria prevalence. Disease data were taken from the World Health Organization Statistical Information System (2011), and measure the number of people infected per 100,000. All three disease measures were log-transformed to reduce the influence of extreme outliers. Data on malaria prevalence are only collected by the WHO for malaria endemic nations. According to the WHO, “endemic” means that there is a constant measurable incidence both of cases and of natural transmission in an area (WHO Statistical Information System 2011). Countries that only have imported cases or introduced cases resulting from imported cases are not included in this data. Thus, “missing values” for malaria were recoded into zeros.

As prior research demonstrates that the severity of disease and life expectancy scores vary significantly by region, several regional interaction terms and regional dummy predictors will also be tested. Dummy variables were created for the macro regions of Sub-Saharan Africa.

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8 HIV prevalence data is only for adults aged 15-49; this is the standard measurement technique as HIV is primarily a sexually transmitted disease, with primary transmission occurring during the sexually-active years (UNAIDS 2006).
Southeast Asia, and Latin America, where observations for nations in a specified region are coded as one and all others are coded as zero.\textsuperscript{9} Interaction terms are created by multiplying these regional dummies with the disease prevalence data. Inclusion of regional dummy predictors tests whether life expectancy rates differ significantly by region, and interaction terms test if the effects of disease on life expectancy are more pronounced in a given region in comparison to others.

**Additional Control Variables.** As an important indicator of level of economic development, GDP per capita will be included as a predictor in all models, in addition to the year term, as a baseline. I employ a measure of GDP per capita PPP, which represents the total annual output of a country’s economy, in current international dollars using Purchasing Power Parity (PPP) rates, per person (World Bank Data 2011). GDP per capita is the total market value of all final goods and services produced in a country in a given year, equal to total consumer, investment, and government spending, divided by the mid-year population. I utilize purchasing power parity rates as they account for the relative effective domestic purchasing power of the average consumer within an economy and compensates for the weakness of local currencies in international markets. Many macro-sociologists argue that PPP estimates of GDP are more appropriate than dollar estimates based on exchange rates, as PPP rates provide a standard measure allowing comparisons of real price levels between countries (e.g. Firebaugh 2003).\textsuperscript{10}

\textsuperscript{9} Interaction terms for remaining regions of the Middle East and Eastern Europe were not included as these diseases tend to have low levels of prevalence in these regions.

\textsuperscript{10} Some argue that PPP rates tend to inflate GDP per capita estimates for poor nations, as PPP data have sometimes been derived from statistical extrapolations rather than by direct observation (e.g. Korzeniewicz and Moran 2000). However, GDP per capita estimates based on exchange rates were also tested across all models included in this dissertation, and the results were consistent across the two measures.
Many studies emphasize education effects on life expectancy (e.g. Soares 2007; Brady et al. 2007; Wickrama and Lorenz 2002). Thus, I employ a measure of secondary school enrollment (World Bank Data 2011). This measure is a gross enrollment ratio, which represents the ratio of total enrollment, regardless of age, to the population of the age group that officially corresponds to secondary level education. Improvements in life expectancy have also been linked to higher levels of urbanization, as urban areas tend to increase people’s access to medical resources and employment with higher incomes (e.g. Brady et al. 2007). Urbanization is measured as the proportion of a country’s total national population that resides in urban areas (World Bank Data 2011).

Insights from demography demonstrate that fertility and access to medical resources are important influences on cross-national life expectancy rates (e.g. Soares 2007; Riley 2005; Wickrama and Lorenz 2002). High rates of fertility can hamper life expectancy improvements, through elevated rates of population growth and increased potential for infant and maternal mortality. I include a measure of the fertility rate, which is an estimate of the number of children an average woman would have if current age-specific fertility rates remained constant during her reproductive years (World Bank Data 2011). In addition, advances in medicine and increasing access to medical resources improve life expectancy rates in poor nations (e.g. Soares 2007; Riley 2005). I thus include number of physicians per 100,000, as an important indicator of health resource availability. This measure includes physicians who are generalists or specialists, and was log-transformed to reduce the influence of extreme cases.
The structure of national economies has been linked to changes in life expectancy, as it is argued by world-system theorists that strong specialization in agriculture can hamper development and well-being gains (e.g. Amin 1976; Bunker 1985). **Agriculture as a percent of GDP** measures the value added to the economy due to activities related to forestry, farming, hunting, and fishing (World Bank Data 2011). In addition to agriculture, high levels of foreign debt can impair well-being improvements as nations devote fiscal resources towards interest and repayments, and also adopt conditionality policies that reduce or privatize social services that are relevant to human health (e.g. Stiglitz 2007; McMichael 2004). I therefore include **total debt as a percent of GNI**, which measures the total external debt as a percent of gross national income (World Bank Data 2011). This measure was log-transformed to reduce the influence of extreme outliers.

More contested within the macro-sociological literatures are indicators of exports and foreign direct investment. While increasing integration into international trade and financial flows can spur development and well-being gains in less-developed nations as these countries gain export revenues and foreign finances help to establish businesses (e.g. Firebaugh and Beck 1994), these forms of development can also increase dependency, as domestic markets become vulnerable to the vicissitudes and fluctuations of global markets (e.g. Chase-Dunn 1998; McMichael 2004). **Exports as a percent of GDP** represents the value of all goods and services provided by a country to the rest of the world, as a percentage of its gross domestic product (World Bank Data 2011). **Inward FDI as a percent of GDP** is measured as inward foreign
investment stock as a percent of GDP (World Bank Data 2011). This measure was log-transformed to reduce the influence of extreme outliers.\textsuperscript{11}

### 2.6 Results

The correlation matrix and univariate statistics for all continuous predictors are presented in Table 2.2. Close inspection of the correlation matrix reveals that many of the variables are highly correlated, heightening concerns of multicollinearity. In particular, the diseases are highly correlated with one another, and the correlation coefficients for many of the development and demographic indicators, such as GDP per capita, schooling, fertility, and physicians suggest major limitations due to multicollinearity. These findings point to the need to investigate the diseases in separate models, and to only include a limited number of predictors at once in a model.\textsuperscript{12}

Table 2.3 reports the findings for the random effects analyses including HIV prevalence. TB and malaria prevalence are examined separately in Tables 4 and 5, respectively. Model 1 of Tables 2.3-2.5 represents a simple baseline, including predictors for disease, GDP per capita, and year. Models 2-6 of Tables 2.3-2.5 add important sociological and demographic controls in a stepwise fashion to help prevent issues of multicollinearity. The final model in Tables 2.3-2.5, Model 7, represents a saturated model, including all statistically significant predictors from prior

\textsuperscript{11} Following dependency themes, I also tested models including the GINI coefficient to capture the influence of income inequality. This predictor did not produce statistically significant results and thus was not included in the models presented here. This is consistent with fixed effects regression results by Beckfield (2006), and also could be attributed to the limited time span of independent variables (1990-2005).

\textsuperscript{12} Evidence of multicollinearity was further established by examining the variance inflation factors (VIFs) for each independent variable regressed on the other predictor variables. In more saturated models and in the regressions that included multiple disease measures, the VIFs ranged from 4-6; thus more conservative models that only include one disease indicator at a time and a limited number of predictors are presented here.
models. The findings are remarkably consistent across Tables 2.3-2.5, so the results will be discussed in tandem.

Table 2.3 demonstrates that HIV prevalence is associated with declines in life expectancy rates over time in less-developed nations, net of the effects of common development and dependency predictors. In addition, Tables 2.4 and 2.5 illustrate that TB prevalence and malaria prevalence are also negatively associated with life expectancy rates in less-developed nations over time, respectively, net of the influence of other indicators. These findings hold even in the most saturated models; although many of the control predictors in Model 7 across Tables 2.3-2.5 become non-significant, most likely due to issues of multicollinearity, the fact that the disease indicators retain their significance in the presence of all relevant controls suggests that the correlation between life expectancy and disease is more robust than the correlation between life expectancy and the other indicators.

Turning attention to the other predictors, GDP per capita is positively associated with life expectancy rates over time in the majority of models; although it does not attain statistical significance in a few models, this is likely due to issues of multicollinearity, as the correlation matrix presented in Table 2.2 illustrates that GDP per capita is highly correlated with life expectancy, as well as many of the other indicators. In addition, the findings for the year term are fairly consistent across the models, demonstrating that life expectancy is increasing over time in less-developed nations, taking into account the effects of the included predictors. This finding is of substantive importance; a recent analysis of life expectancy by Brady and colleagues (2007) finds a negative association between year and life expectancy, suggesting that life
expectancy is declining over time, net of the effects of the included development and dependency predictors (which are consistent with those examined here). However, that study did not include any predictors for disease. The results reported here therefore suggest that malaria, TB, and HIV rates in less-developed nations at least partly contribute to the declines in life expectancy over time that some poor nations have experienced. This is likely to be especially relevant for HIV, as year in Model 7 of Table 2.3 retains significance, while it does not in Tables 2.4 and 2.5.\textsuperscript{13} Year also dips out of conventional significance thresholds in Model 4 which includes the fertility rate across Tables 2.3-2.5. This suggests that fertility levels and disease are relevant to explaining whether there are life expectancy gains or losses from 1990-2009 in less-developed nations.\textsuperscript{14}

Model 2 of Tables 2.3-2.5 include predictors for education and urbanization; both of these factors have robust positive influences on life expectancy rates, net of the effects of disease, GDP per capita, and year. Models 3-4 include the fertility rate and number of physicians. The results illustrate that physicians tend to be associated with improvements life expectancy rates in less-developed nations over time, but this indicator is highly correlated with fertility and GDP per capita, causing significance to be compromised in some of the fuller models. Given the value of the regression coefficient and level of significance, the fertility rate has very robust negative influences on life expectancy rates in less-developed nations over time.

\textsuperscript{13} Non-significance of year in Model 7 of Tables 4-5 could also be the result of multicollinearity, as many of the included predictors are increasing in a linear fashion over time; so this point is to be interpreted with caution.\textsuperscript{14} Indeed, further tests demonstrate that when the disease predictors are eliminated from the models, the year terms are generally not significant. This provides further evidence that HIV, TB, and malaria contribute to significant life expectancy declines over time.
Models 5-6 include other important economic and structural indicators and the results demonstrate that specialization in agriculture and percent GDP from exports have notable negative associations on life expectancy rates over time in less-developed nations, net of the influence of the baseline predictors. However, predictors for debt and FDI do not have statistically significant associations with life expectancy rates. The final model, Model 7, includes all relevant predictors from prior models, and looking across Tables 2.3-2.5, it is evident that disease and fertility have the most consistent and robust effects on life expectancy scores over time in less-developed nations.

Taken together, the findings presented in Tables 2.3-2.5 demonstrate that HIV, TB, and malaria have robust, pernicious influences on life expectancy rates in less-developed nations over time. In fact, in the majority of the models, the disease indicator is the most powerful predictor of life expectancy. Additionally, while GDP per capita is often associated with increasing life expectancy rates, education, urbanization, physicians, and fertility produced stronger associations with life expectancy scores. This finding confirms prior work which argues that expanding education, increasing women’s social standing, and reliable access to medical care are perhaps more important in producing physical well-being gains over time in poor nations than an exclusive focus on economic growth (e.g. Brady et al. 2007; Soares 2007; Wickrama and Lorenz 2002).

The results presented in Tables 2.3-2.5 confirm the first hypothesis, demonstrating that HIV, TB, and malaria all have negative influences in life expectancy rates in less-developed nations over time, net of the influence of pertinent development and dependency predictors.
Prior research suggests that disease may have a more profound impact on life expectancy in certain regions. Table 2.6 presents the results of random effects models including contextual indicators for region and region-disease interactions. In each model, GDP per capita and year are included as a relevant controls, and Models 1-4 of Table 2.6 depict that GDP per capita and year have consistent positive influences on life expectancy rates over time in less-developed nations, net of the effects of the disease and region predictors. Models 1-3 examine disease effects in Sub-Saharan Africa for HIV, TB, and malaria respectively. These models illustrate that Sub-Saharan African nations tend to have lower levels of life expectancy. In addition, the results demonstrate that the harmful effects of HIV, TB, and malaria on life expectancy rates over time are much more pronounced in Sub-Saharan Africa, in comparison to other nations included in the analyses.

Notably, Models 2 and 3 demonstrate that TB and malaria still have pernicious effects on life expectancy rates for less-developed nations over time, as the main effect for disease retains statistical significance when the Sub-Saharan Africa terms are included in the model. However, in Model 1, the main effect for HIV prevalence is not statistically significant, suggesting that the harmful effects of HIV on life expectancy over time are largely a feature of Sub-Saharan African nations, while the negative influence of malaria and TB on life expectancy rates in less-developed nations have profound influences both within Africa and across other regions as well. In addition, the final model, Model 4, reports significant findings for TB and Southeast Asian nations. Although Southeast Asian nations tend to have higher levels of life expectancy than other less-developed nations, the harmful effects of TB on life expectancy over
time are especially pronounced in this region in relation to others. Again, the main effect for TB retains significance when the Southeast Asian terms are introduced into the model, once again providing evidence that the negative effects of TB on life expectancy over time are robust across all less-developed nations, but are especially strong in the regions of Sub-Saharan Africa and Southeast Asia.

The models reported previously provided some crude evidence that each of these diseases have comparable influences on life expectancy rates in less-developed nations over time, given the similar values of the standardized regression coefficients for HIV, TB, and malaria in Tables 2.3-2.5. However, the samples analyzed are not consistent, so in order to investigate this further, I created a uniform sample through listwise deletion techniques. The results are presented in Table 2.7, which again includes the baseline indicators of GDP per capita and year. Although the diseases are highly related with one another and could not be analyzed in the same model, they are measured with identical units and thus these models allow for comparisons in the magnitude of the effect of the different diseases on life expectancy rates over time in less-developed nations. Results presented in Models 1-3 of Table 2.7 suggest that TB has the most pernicious effects on life expectancy rates in less-developed nations from 1990-2009, as the size of the standardized coefficient is notably larger than for HIV prevalence. The coefficient for malaria is smaller than the other two diseases, suggesting that this disease is the least harmful for life expectancy rates from 1990-2009 in less-developed nations.

Thus, results presented in Table 2.6 confirm the second hypothesis, demonstrating that Sub-Saharan African nations have significantly lower life expectancy rates, and that the effects of
HIV, TB, and malaria prevalence on life expectancy are especially pronounced in this region. However, differences in significance for the main effect for disease suggest that the damaging influences of HIV on life expectancy are largely attributed to conditions in Sub-Saharan Africa, while the harmful effects of TB and malaria on life expectancy rates are more widespread. For TB and malaria, the significant regional interaction is of substantive relevance; these models demonstrate that TB and malaria are more likely to result in premature death in Sub-Saharan African nations, and in Southeast Asian nations in the case of TB, in comparison to other less-developed countries. It is likely that differences in access to health resources or treatment is important in explaining why the effects of TB and malaria on life expectancy are generally more pronounced in Sub-Saharan African nations than in other regions. Explaining these regional patterns in the case of HIV requires a slightly different interpretation. HIV cannot fundamentally be “more deadly” in any particular region, as there is so cure for HIV and it eventually results in death in any locale, or with any level of treatment. However, the significant interaction for HIV and Sub-Saharan Africa suggests that perhaps people die more quickly from HIV in this region than in others, or that simply the overall level of HIV infection is high enough to alter life expectancy gains, while in other nations it is not. Future research that is able to control for the use of life-lengthening ART drugs for HIV patients may be especially useful in further assessing the causes of these important regional trends.

Although there is overwhelming focus on HIV, the evidence garnered across all the models suggests that TB and malaria also have profound influences on life expectancy rates in less-developed nations over time, and that TB may be more harmful overall (given the values of
the standardized regression coefficients and the regional findings). Although HIV also has detrimental influences on life expectancy, this association is largely a feature of Sub-Saharan African nations, while the effects of TB and malaria on premature death appear to be much more widespread.

2.7 Conclusion

Death from communicable disease is comparatively quite rare in developed nations; however, infectious diseases continue to plague the people of less-developed nations. Malaria, tuberculosis, and HIV represent three of the most prominent deadly illnesses that afflict poor regions, and the results demonstrate that each of these diseases are contributing significantly to life expectancy rates in less-developed nations over time, even when taking into account relevant demographic, development, and dependency factors. In addition, the effects of malaria, TB, and HIV on life expectancy are especially pronounced in Sub-Saharan African nations, and TB also has especially harmful influences in Southeast Asian nations. These regional patterns are likely to be the result of heightened poverty and low-quality health care that characterize these regions (e.g. Farmer 2001), leading to limited efficacy of treatments and increased progression of illness into death.

Although there is extensive scholarly and public health consideration given to HIV, the results suggest that attention to TB and malaria is relatively understated. Even though this study does not analyze inequality in life expectancy directly, the findings speak to prior assessments which have concluded that elevated HIV rates in Sub-Saharan Africa are responsible for trends
of increasing cross-national life expectancy divergence. Evidence for this assertion is
demonstrated in the models that tested for differential regional effects and in the patterns of
significance for the year term. However, it is evident that TB and malaria are also contributing
to altered life expectancy trajectories across nations over time, and especially so in Sub-Saharan
Africa. Therefore TB and malaria at least partly contribute to the diverging life expectancy
trends evidenced among Sub-Saharan African nations, and TB and malaria also represent key
threats to well-being, alongside HIV, in poor nations. Consequently, sociological research and
international organizations alike could benefit by expanding attention of disease in less-
developed nations to the “neglected” infections of TB and malaria. This is especially relevant to
the issue of public health funding, as international assistance for diseases such as TB and malaria
has declined over time (MacKellar 2005). In addition, the advent of antibiotic resistant strains
necessitates a renewed commitment to providing appropriate treatments to these infections,
before they become completely untreatable and further heighten mortality.

The regional interactions also suggest that the influences of HIV prevalence on life
expectancy may not be very widespread to other regions, and TB prevalence produced
consistently larger effects on cross-national life expectancy rates, in both the general models and
in the regional assessments. It is possible that this finding can be somewhat attributed to use of
anti-retroviral therapies (ARTs), which prolong the life of HIV patients. Indeed, it is likely that
HIV will have less of a negative influence on life expectancies as ARTs become more widely
available and HIV-infected individuals live longer.
As the health and well-being of people is central to achieving economic growth (e.g. Acemoglu and Johnson 2007; Packard 2009), combating disease and improving life expectancies in poor nations represents at once both a means and an outcome of development. Although some processes of development are contested within the modernization and world-systems/dependency schools, the results presented here provide some support for both perspectives. Modernization, by means of economic growth, educational expansion, and increased urbanization improves life expectancy scores in the face of disease. However, increased exports and specialization in agriculture depress life expectancy gains in less-developed nations, suggesting that focusing on comparative advantages in primary sector products and increased integration into international trade are not the most appropriate means for successful development. In addition, reducing fertility and expanding access to doctors have robust influences on life expectancy. Although neoliberal policies that relegate social provisions may spur short term financial gains in poor nations, they compromise the health of the people, thus likely contributing to long-term developmental declines (e.g. Harvey 2006).

Malaria, TB, and HIV are overlapping and combining with one another to produce especially damaging patterns of morality in poor nations, particularly among Sub-Saharan African nations. Indeed, measures of HIV, TB, and malaria are so highly correlated with one another that it was impossible to include them in a model together. This point speaks more broadly to a key limitation of this analysis; potential issues of multicollinearity greatly compromised the number of indicators that could be included in the models, as well as the significance of many well-established predictors of life expectancy in the more saturated models.
While bias due to multicollinearity is a key concern for this cross-national analysis, the same implications apply to a wide range of studies in the research area. Additionally, the intense concentration of HIV in Sub-Saharan Africa suggests that it may be appropriate to look at HIV dynamics specifically within this region, as well as in less-developed nations on the whole.

Another limitation of the analytical technique includes the inability to examine both direct and indirect effects. Although fertility, education, and urbanization are often seen as competing with GDP per capita for influences on life expectancy (e.g. Brady et al. 2007), GDP gains also serve to enhance these non-economic factors, thus likely having both direct and indirect influences on health measures. In the context of this analysis, it is very probable that many of the common predictors of life expectancy (e.g. GDP per capita, schooling, fertility, and physicians) not only influence rates of disease, but in addition have important causal relationships among each other.\textsuperscript{15} These critiques necessitate the examination of the causes of disease using structural equation modeling, as this technique allows for the construction of composite indicators (latent constructs) and the specification of direct and indirect relationships.\textsuperscript{16} Thus, based on the findings which demonstrate the importance of malaria, TB, and HIV in less-developed nations, along with the methodological concerns encountered here,

\textsuperscript{15} In addition, Bollen and Brand (2008) have critiqued panel models for assuming that coefficient effects and the unexplained variance remains equal across all waves of data. Although these are important critiques, addressing them through SEM methods is not possible here given the structure of the data (see footnote below). Additionally, this study uses a fairly limited time span (15 years) in comparison to other assessments which analyze data across multiple decades, lessening these concerns.

\textsuperscript{16} Although random effects models can be estimated within a SEM framework, this technique requires that each indicator be included for each time point, producing major compromises to degrees of freedom and the number of indicators that can be included in the model for small-N samples. Thus, even though there are notable weaknesses to the traditional panel techniques employed here, this is the most appropriate strategy to examine longitudinal trends, given the limitations of the data. For a full discussion of the limitations of random effects panel models and the characteristics of random effects SEMs, see Bollen and Brand (2008).
the following empirical chapters will utilize structural equation modeling to more
compactly and appropriately examine the causes of these diseases.

Many are unaware that malaria and TB afflict people in impoverished regions at rates
that surpass the scope of the HIV pandemic. Part of this bias comes not only from the focus of
research and public discourse, but also from the knowledge that malaria and TB are treatable
infections. Thus, the fact that these two diseases are negatively associated with life expectancy
rates at a comparable level to HIV is especially discouraging and in some ways quite surprising.
However, the ability to cure malaria and TB is declining with the emergence of antibiotic
resistant strains of both infections. This may partly explain the findings demonstrated here, and
also illustrates the need to further examine the causes of disease in less-developed nations. As
disease dynamics are changing over time with the provisions of anti-retroviral therapies (ARTs)
for HIV and increasing antibiotic resistance for malaria and TB, it will also be important to
monitor rates of all three diseases and their contributions to life expectancy in future projects.
At minimum, the results of this study demonstrate that public health policy could only benefit
from a renewed effort to provide increased TB and malaria prevention and care, especially given
the most recent trends in international health funding.
Table 2.1: Nations Included in the Life Expectancy Analyses

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<tr>
<th>Albania</th>
<th>Dominican Republic</th>
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Table 2.2: Correlation Matrix and Univariate Statistics for Life Expectancy Analyses

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### Table 2.3: Random Effects Regression of HIV Prevalence on Life Expectancy

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Notes: *p<.10, *p<.05, **p<.01, ***p<.001 (one-tailed tests); standardized regression coefficients flagged for statistical significance, unstandardized regression coefficients reported in italics, robust standard errors reported in parentheses.
## Table 2.4: Random Effects Regression of TB Prevalence on Life Expectancy

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Notes: * p<.10, * p<.05, ** p<.01, *** p<.001 (one-tailed tests); standardized regression coefficients flagged for statistical significance, unstandardized regression coefficients reported in italics, robust standard errors reported in parentheses.
Table 2.5: Random Effects Regression of Malaria Prevalence on Life Expectancy

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<tr>
<td></td>
<td>(0.037)</td>
<td>(0.037)</td>
<td>(0.340)</td>
<td>(3.00)</td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.035)</td>
</tr>
<tr>
<td>Number of Physicians</td>
<td>0.140**</td>
<td>1.01</td>
<td>-0.005</td>
<td>0.034</td>
<td>-0.061</td>
<td>-0.061</td>
<td>0.088*</td>
</tr>
<tr>
<td></td>
<td>(0.340)</td>
<td>(3.00)</td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.026)</td>
<td>(0.026)</td>
<td>(0.426)</td>
</tr>
<tr>
<td>Fertility Rate</td>
<td>-0.512***</td>
<td>-2.83</td>
<td>0.046*</td>
<td>-0.028</td>
<td>-0.376</td>
<td>-0.376</td>
<td>-0.448***</td>
</tr>
<tr>
<td></td>
<td>(0.314)</td>
<td>(3.14)</td>
<td>(0.014)</td>
<td>(0.128)</td>
<td>(4.11)</td>
<td>(4.11)</td>
<td>(3.82)</td>
</tr>
<tr>
<td>Agriculture as % GDP</td>
<td>-0.090**</td>
<td>-0.061</td>
<td>-0.061</td>
<td>-0.042</td>
<td>-0.042</td>
<td>-0.042</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.26)</td>
<td>(0.026)</td>
<td>(0.026)</td>
<td>(0.038)</td>
<td>(0.038)</td>
<td>(0.038)</td>
<td></td>
</tr>
<tr>
<td>Exports as % of GDP</td>
<td>-0.046*</td>
<td>-0.028</td>
<td>-0.028</td>
<td>-0.025</td>
<td>-0.025</td>
<td>-0.025</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.014)</td>
<td>(0.014)</td>
<td>(0.014)</td>
<td>(0.128)</td>
<td>(0.128)</td>
<td>(0.128)</td>
<td></td>
</tr>
<tr>
<td>Debt as % GNI</td>
<td>-0.031</td>
<td>-0.376</td>
<td>-0.376</td>
<td>-0.004</td>
<td>-0.004</td>
<td>-0.004</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.411)</td>
<td>(4.11)</td>
<td>(4.11)</td>
<td>(1.28)</td>
<td>(1.28)</td>
<td>(1.28)</td>
<td></td>
</tr>
<tr>
<td>FDI Stocks as % GDP</td>
<td>-0.004</td>
<td>-0.025</td>
<td>-0.025</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.128)</td>
<td>(0.128)</td>
<td>(0.128)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>61.37***</td>
<td>53.90***</td>
<td>60.85***</td>
<td>74.80***</td>
<td>63.94***</td>
<td>62.05***</td>
<td>71.34***</td>
</tr>
<tr>
<td></td>
<td>471</td>
<td>348</td>
<td>380</td>
<td>380</td>
<td>445</td>
<td>380</td>
<td>271</td>
</tr>
<tr>
<td>R²</td>
<td>.4448</td>
<td>.5753</td>
<td>.4340</td>
<td>.6223</td>
<td>.4747</td>
<td>.4897</td>
<td>.6503</td>
</tr>
</tbody>
</table>

Notes: * p<.10, * p<.05, ** p<.01, *** p<.001 (one-tailed tests); standardized regression coefficients flagged for statistical significance, unstandardized regression coefficients reported in italics, robust standard errors reported in parentheses.
Table 2.6: Random Effects Regression of Disease on Life Expectancy over Time - Including Regional Interaction Terms

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>GDP per capita</td>
<td>.090***</td>
<td>.048*</td>
<td>.070*</td>
<td>.065*</td>
</tr>
<tr>
<td></td>
<td>(.001)</td>
<td>(.001)</td>
<td>(.001)</td>
<td>(.001)</td>
</tr>
<tr>
<td>Year</td>
<td>.146***</td>
<td>.129***</td>
<td>.123***</td>
<td>.107***</td>
</tr>
<tr>
<td></td>
<td>(1.41)</td>
<td>(1.12)</td>
<td>(1.06)</td>
<td>(.930)</td>
</tr>
<tr>
<td>Sub-Saharan Africa</td>
<td>-.670***</td>
<td>-.654***</td>
<td>-.798***</td>
<td>-.206***</td>
</tr>
<tr>
<td></td>
<td>(-14.09)</td>
<td>(-13.24)</td>
<td>(-16.16)</td>
<td>(13.99)</td>
</tr>
<tr>
<td></td>
<td>(.26)</td>
<td>(.25)</td>
<td>(.27)</td>
<td>(3.98)</td>
</tr>
<tr>
<td>Southeast Asia</td>
<td></td>
<td></td>
<td>.206***</td>
<td>13.99</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(3.98)</td>
</tr>
<tr>
<td>HIV Prevalence</td>
<td>-.041</td>
<td>-.264***</td>
<td>-.395***</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.314)</td>
<td>(.314)</td>
<td>(.410)</td>
<td></td>
</tr>
<tr>
<td>TB Prevalence</td>
<td></td>
<td>-.172***</td>
<td>-.305***</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-1.53)</td>
<td>(-3.50)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.385)</td>
<td>(.410)</td>
<td></td>
</tr>
<tr>
<td>Malaria Prevalence</td>
<td></td>
<td></td>
<td>-.096**</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(-1.81)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(.058)</td>
<td></td>
</tr>
<tr>
<td>HIV * Sub-Saharan Africa</td>
<td>-.185***</td>
<td>-.253***</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(-1.72)</td>
<td>(-2.33)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(.457)</td>
<td>(.196)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TB * Sub-Saharan Africa</td>
<td></td>
<td></td>
<td>-.196***</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(-1.16)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(.145)</td>
<td></td>
</tr>
<tr>
<td>Malaria * Sub-Saharan Africa</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TB * Southeast Asia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>63.60***</td>
<td>72.58***</td>
<td>62.94***</td>
<td>77.14***</td>
</tr>
<tr>
<td>N</td>
<td>311</td>
<td>467</td>
<td>471</td>
<td>467</td>
</tr>
<tr>
<td>R²</td>
<td>.7643</td>
<td>.7679</td>
<td>.6783</td>
<td>.6063</td>
</tr>
</tbody>
</table>

Notes: * p<.10, * p<.05, ** p<.01, *** p<.001 (one-tailed tests); standardized regression coefficients flagged for statistical significance, unstandardized regression coefficients reported in italics, robust standard errors reported in parentheses.
Table 2.7: Random Effects Regression of Disease on Life Expectancy Over Time - Allowing for Comparisons in the Effects of Disease

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>GDP per capita</td>
<td>.147* (.000)</td>
<td>.091* (.000)</td>
<td>.095* (.000)</td>
</tr>
<tr>
<td>Year</td>
<td>.196*** (.257)</td>
<td>.086*** (.196)</td>
<td>.094*** (.203)</td>
</tr>
<tr>
<td>HIV Prevalence</td>
<td>-.308*** (.340)</td>
<td>-.398*** (.594)</td>
<td></td>
</tr>
<tr>
<td>TB Prevalence</td>
<td></td>
<td>-.398***</td>
<td>-.279*** (.594)</td>
</tr>
<tr>
<td>Malaria Prevalence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>53.97***</td>
<td>80.72***</td>
<td>63.04***</td>
</tr>
<tr>
<td>N</td>
<td>311</td>
<td>311</td>
<td>311</td>
</tr>
<tr>
<td>R²</td>
<td>.5728</td>
<td>.5842</td>
<td>.4662</td>
</tr>
</tbody>
</table>

Notes: * p<.10, * p<.05, ** p<0.01, *** p<0.001 (one-tailed tests); standardized regression coefficients flagged for statistical significance, unstandardized regression coefficients reported in italics, robust standard errors reported in parentheses.
CHAPTER 3

HIV, URBANIZATION, AND DIFFERING IMPACTS OF FEMALE EMPOWERMENT IN SUB-SAHARAN AFRICA

3.1 Introduction

HIV represents one of the most widely studied and talked about infections in medical science and public discourse alike (e.g. Farmer 2001; MacIntosh and Thomas 2004; Shircliff and Shandra 2011; UN2005; UNAIDS 2009). Most efforts to examine HIV focus on the psychological and behavioral factors associated with transmission, and public policy largely aims to change the individual actions of people at most at risk (e.g. Allen et al. 2006; Atlani et al. 2000; Gaillard 2006; UNAIDS 2009). Cross-national statistics reveal that there is amazing inequality in HIV prevalence rates across nations, even among less-developed countries (WHO 2011a). Thus conditions of risk or vulnerability are relevant across multiple contexts, including national levels.

Although a new body of research is beginning to emerge on the cross-national determinants of HIV (McIntosh and Thomas 2004; Burroway 2010; Grey 2004; Shircliff and Shandra 2011), these examinations are limited by their methodological approaches which fail to take into account pertinent mediating relationships, as well as a lack of integration from case-study research, which links HIV rates to high levels of migration to urban areas in Sub-Saharan Africa (Gushulak and MacPherson 2004; Gutmann 2007; Dyson 2003). As environmental changes associated with global warming represents a premier cause of rural-to-urban migration in poor
societies (Centre on the Epidemiology of Disasters 2011; Dyson 2003; Hunter et al. 2011), we need to examine the larger environmental and social contexts in which HIV transmission occurs.

Sub-Saharan Africa represents a locus of the HIV pandemic, where rates can vary from 15-30% in some nations (WHO 2011a). Furthermore, women’s share of the infection is increasing in this region and globally, where there were around 12 million women living with HIV in 2009 in Sub-Saharan Africa, compared to about 8.2 million men (WHO 2011a). The Caribbean has also seen an alarming rise in the number of HIV-infected women, and much of this increase is attributed to the commercial sex industry. A study of HIV prevalence among female sex workers in Georgetown, Guyana, showed that around 40% were infected with HIV (Allen et al. 2006). Contraceptive use and related dimensions of female empowerment thus represent important factors in the epidemiology of this sexually-transmitted infection.17 This can be seen by public policy efforts, which most often direct resources to education on contraceptives and other safe sex practices, especially among women, to prevent the spread of HIV in poor societies (e.g. UNAIDS 2009).

Although increasing the social standing of women is generally touted as a key mechanism to curbing the HIV pandemic (e.g. Shircliff and Sandra 2011; Burroway 2010; Heimer 2007; Scanlan 2010), some studies conducted within Sub-Saharan Africa illustrate contradictory evidence. For example, research demonstrates that increased levels of female empowerment also spur movement into cities, which afford greater diversification and expansion of social relations (e.g. Rushing 1995; Farmer 2001). In this way, female empowerment may indirectly

17 Thus although HIV shares considerable overlap with TB and malaria cross-nationally, the proximate causes of these diseases differ substantially due to the characteristics of transmission that make gender equality a paramount concern for HIV transmission.
promote HIV in certain regions. Indeed, urban areas have historically served as nodal points for disease, and many note that HIV rates are much higher in urban settings than in rural areas (Dyson 2003; Gushulak and MacPherson 2004). These issues point to the need for comparative assessments that carefully test for indirect and direct relationships among economic growth, female empowerment, urbanization, environmental change, and HIV. Additionally, the concentration of this disease within Sub-Saharan Africa, as well as the divergent case-study findings, point to the need to examine the structural factors that promote HIV rates across two samples: less-developed nations and Sub-Saharan African nations.

The emerging themes regarding urbanization and disease fit uniquely into the comparative sociology literature, as urbanization can be both a symptom of development and dependency. Whether or not urbanization improves well-being likely depends on the distribution and types of resources that urban areas provide. Although urban settings tend to have higher levels of health infrastructure in terms of access to schooling and doctors, very high levels of urban growth are likely to strain those resources (Moore et al. 2003). Researchers are also picking up on fundamental processes that relate to population patterns and demographic flows, where the mass movement of people into a concentrated area necessarily increases rates of disease transmission, especially in regions that have high levels of prevalence initially (Dyson 2003; Gushulak and MacPherson 2004; Gutmann 2007). I thus now turn to taking a closer look at HIV dynamics within less-developed nations, including a focus on the processes surrounding urbanization within Sub-Saharan Africa.
3.2 Characteristics of HIV in Less-Developed Nations

The UN general assembly has stated that the AIDS pandemic “will be the single biggest obstacle for reaching the Millennium Development Goals.” This disease no doubt has severe implications for economic development and societal well-being as it is a leading cause of death among young and working-age adults worldwide (Lorentzen et al. 2008; Ukpolo 2004; Garnett et al. 2001). Although anti-retroviral therapies (ARTs) now exist which can prolong the life of infected HIV patients, these drugs still have much lower rates of use in poor nations, due to their high cost and the limited availability of cheaper, generic varieties. For example, in less-developed nations, the average time to death after initial HIV infection ranges from 3-8 years (Heimer 2007; Barnett 2004), while in the United States, where ARTs are widely used, HIV patients can expect to live as long as 24 years after initial diagnosis (Walensky et al. 2006). In Sub-Saharan Africa, where over 60% of the world’s HIV cases are found, less than a third of the people who currently need ARTs are getting them (UNAIDS 2009).

The spread of HIV/AIDS represents a major threat to development and well-being as it spurs increased pressure on medical resources, elevates the number of orphaned children, creates prolonged absences from work, causes decreases in household incomes, and stimulates higher household expenses (Barnett 2004; Heimer 2007; Lozentzen et al. 2008). In fact, a case study of Tanzania finds that if a household contains at least one HIV/AIDS patient, 29% of the household labor supply was spent on AIDS-related matters (Dixon et al. 2001a). Another assessment conducted in Uganda finds that if the key breadwinner becomes infected with HIV,

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18 The Millennium Development Goals are centered on ending poverty and hunger, providing universal education, achieving gender equality, and promoting environmental sustainability.
household incomes drop by around 80% (UN 2001). Indeed, while most of the literature on HIV/AIDS tends to be sub-regional, qualitative case-studies, or descriptive in nature, the cross-national research on HIV/AIDS that does exist most predominantly focuses on the impact of this disease on rates of economic development (e.g. Garnett et al. 2001; Dixon et al. 2001b; Ukpolo 2004; Lozentzen et al. 2008; Acemoglu and Johnson 2007). This body of research broadly concludes that HIV/AIDS has harmful impacts on rates of economic growth (Garnett et al. 2001; Ukpolo 2004; Clapham 2006; Lozentzen et al. 2008; Acemoglu and Johnson 2007).

The impairment of infected individuals’ labor power during what is otherwise considered the most productive stage in the life course undeniably impedes national trajectories of growth and development. Although assessing the impacts of disease on economic development is crucial as high prevalence is likely to reproduce conditions of underdevelopment in less-developed nations, we also need to look beyond the economic ramifications of disease in cross-national assessments to study of the structural causes of disease for themselves. Indeed, the need for more comparative assessments of the determinants of HIV is echoed by demographers, such as Clapham and colleagues (2006: 106) who proclaim that, “Although a vast amount has now been written about the subject [AIDS], the majority consists in localized and limited studies, from which it is difficult to draw broader conclusions.”

The few cross-national examinations of HIV determinants that do exist point to secondary education and female empowerment as the most robust predictors of national HIV rates (e.g. Burroway 2010, McIntosh and Thomas 2004). Although education is often treated as 19 Exceptions to this include cross-national assessments by McIntosh and Thomas (2004), Burroway (2010), and Shircliff and Shandra (2011).
a human capital component to development, especially within classic modernization approaches, demographic and epidemiological literatures also emphasize that education has beneficial influences on health outcomes, outside of economic interpretations (e.g. Wickrama and Lorenz 2002; Soares 2007; Elo 2009; Burroway 2010). This is especially relevant to HIV, as many studies demonstrate that schools in less-developed nations often disseminate information about sexual and reproductive health, such that more people know how to protect themselves from HIV infection (e.g. Burroway 2010; Vandemoortele and Delamonica 2002). Those with higher levels of education tend to know more about HIV (Vandemoortele and Delamonica 2002). Schools are even beginning to introduce specific HIV prevention curriculums. In the Kagera region of Tanzania, for example, school-based risk reduction programs focus on the symptoms of HIV/AIDS, how it spreads, how to protect against infection, and how to provide care to HIV/AIDS patients (Lugalla et al. 2004). Such programs tend to improve knowledge about HIV/AIDS, foster attitudes favorable to risk reduction, and contribute to changed behaviors, including delayed sexual debut, decreased number of sexual partners, or increased condom use (Gallant and Maticka-Tyndale 2004). More educated populations are better prepared to protect themselves, and more likely to change their HIV risk-related behaviors faster than the uneducated (Vandemoortele and Delamonica 2002).

Many studies also find that measures of female schooling or female literacy are especially influential in reducing prevalence (Burroway 2010; Shircliff and Shandra 2011). Female participation in schooling greatly reduces HIV rates not only for the reasons echoed above, but also by enhancing women’s social standing and economic opportunities (Wickrama and Lorenz
2002). Issues of female empowerment are particularly relevant as women face a higher HIV burden cross-nationally, representing nearly 60% of global HIV cases (Heimer 2007). This is likely because women are more vulnerable to HIV infection both socially and biologically. Women tend to have less access to health care and preventative treatment, fewer educational opportunities, and strong ties to traditional gender roles, all of which subordinate their health status (Wickrama and Lorenz 2002). Medically, HIV cells also infect women more easily than men (UNAIDS 2009). In fact, male-to-female transmission is estimated to be about 2 or 3 times more “efficient” as female-to-male transmission (Heimer 2007, UNAIDS 2009).

Female empowerment is also particularly relevant to the use of contraceptives, and contraceptive use is shown to be a major factor reducing HIV rates across nations (e.g. Shircliff and Shandra 2011). This is consistent with the case-study literature on the female sex industry. Female sex workers are at a higher risk of HIV exposure as they are often not in a position to insist that their customers wear condoms (Allen et al. 2006). Alarmingly, men will still pay more money for unprotected sex with a sex worker (Gaillard et al. 2006). This means that female sex workers are not only at risk of becoming infected with HIV, but that if they are already infected, they can pass the virus on to their other clients. In recent years some countries have documented a decline in the rate of new HIV infections among sex workers. In Haiti, for example, HIV prevalence among female sex workers attending HIV voluntary counseling and testing declined from 63 percent in 1987 to 22 percent in 2003 (Gaillard et al. 2006). The decline is attributed to better education and knowledge about HIV prevention. Indeed, these strategies are most common among international public policy efforts, which direct resources to
education on contraceptives and other safe sex practices to prevent the spread of HIV in poor societies (e.g. UNAIDS 2009; Global Fund to Fight HIV, TB and Malaria 2011; WHO 2005; World Bank 2010).

While these ideas about education and female empowerment are consistent with prior research and theory on modernization and health (e.g. Brady et al. 2007; Bates et al. 2004; Princhtet and Summers 1996), some sub-regional studies conducted within Sub-Saharan Africa find that increasing education and economic opportunities, especially for women, promotes movement into cities and the erosion of traditional sexual norms, heightening possibilities for HIV transmission (e.g. Heimer 2007; Rushing 1999). Indeed, many studies find higher rates of HIV in urban areas, contending that urban populations tend to be more mobile and have a higher number of sexual contacts than rural people (e.g. Dyson 2003). Work migration patterns to urban centers can also further HIV rates in rural villages (Gushulak and MacPherson 2004; Gutmann 2007). For example, many Sub-Saharan African men leave their isolated rural villages for temporary work in the cities or in other regions. Absence from home inflates opportunities to engage in unsafe sex practices, possibly resulting in the contraction of sexually-transmitted infections which are then passed on when the husband returns home (Heimer 2007; Hunt 1989; Gregson et al. 2001; Gushulak and MacPherson 2004).

Demographic assessments also point out that high rates of urban growth can strain public health resources, indirectly encouraging people to engage in risky sexual behaviors (e.g. Moore et al. 2003). Thus while economic growth, increasing women’s social standing, and the expansion of education are generally seen as indicators of modernization that are good for health
(e.g. Brady et al. 2007; Firebaugh and Beck 1994), these factors may also promote high levels of urban migration, which could serve to increase HIV rates (e.g. Dyson 2003). The very limited body of cross-national work on HIV already supports this line of thinking, despite that these assertions have not been fully tested or properly considered. For example, Burroway (2010) finds that schooling is especially effective at reducing HIV rates in Latin America, but not in Sub-Saharan Africa. Additionally, Shircliff and Shandra (2011) find that GDP growth is associated with an increase in HIV rates in less-developed nations. Other research also demonstrates that within Sub-Saharan Africa, the relationship between economic development and HIV prevalence is extremely weak (e.g. Heimer 2007; Gregson et al. 2001). Based on this evidence, it is likely that urban growth serves as a mediating variable in each of these contexts, particularly for Sub-Saharan African nations.

Related to these trends in urbanization are new ideas about the role of environmental change in contributing to heightened rates of urban migration. Migration is a particular adaptation strategy used by rural households under environmental strain (e.g. Nunam 2010; Comim, Kumar, and Sirven 2009), and world-systems/dependency theorization emphasizes that over-urbanization in less-developed nations often results from changes in the agricultural sector or the distribution of land ownership, which in turn spur massive rural-to-urban migration flows. As rural households depend upon the natural environment for their livelihoods, changes in weather and other conditions related to climate change can also threaten well-being and encourage migration (Comim et al. 2009). In particular, climate change dynamics have been linked to changing rainfall patterns, where many areas, especially in Sub-Saharan Africa, are
becoming dryer (Collier, Conway and Venables 2008). Even these patterns represent broader global development dynamics, as affluent nations are most responsible for the issue of global warming, yet people in less-developed nations are generally much more vulnerable to the effects of climate change, including susceptibilities to natural disasters, sea level rise, and altered weather patterns (e.g. Roberts and Parks 2008; Collier et al. 2008).

Drought represents a major threat to agricultural production for rural households, especially as many poor regions lack irrigation. A new study by Hunter and colleagues (2011) finds that drought is a significant predictor of migration from rural Mexico. Changes in rainfall have also been linked to emigration trends in Burkina Faso, Mali, and Indonesia (e.g. Henry et al. 2004; Moller et al. 2001; Findley 1994). These studies have generally linked changing environmental conditions to international migration, despite that the majority of environmental migrants migrate internally to urban areas (Centre for Research on the Epidemiology of Disasters 2011). Thus, exploring the link between drought and urban growth represents an emerging area of research, and demonstrates the underlying importance of environmental conditions in contributing to disease trends.

The emerging findings regarding the roles of female empowerment, schooling, economic growth, and urbanization in contributing to HIV prevalence draw on important aspects of modernization and dependency theorizations. Many studies have found that education, female empowerment, and affluence reduce HIV rates across nations, consistent with a modernization approach to health. However, emerging patterns in urbanization are deeply informed by dependency/world-system traditions that emphasize issues of over-urbanization in less-
developed nations, which often result from changes in the environment or access to natural resources. There may also be some support for aspects of modernization theory which highlight that development is often accompanied by a shift away from traditional value systems (e.g. Learner 1958). In this way, modernization may disrupt sexual norms and promote more liberal sexual behavior, including more risky sexual behavior. Thus, while modernization does improve health generally, there may be some specific aspects of modernization that lead to higher levels of sexually-transmitted infections in less-developed societies.

Cultural elements are likely to have particular relevance in the case of HIV (and other sexually transmitted infections), as sexual norms can vary greatly across cultural or religious groups. Huntington (1996) notes that the world remains fractured along cultural or religious lines, with each civilization having a distinct value system. Some of these value systems are characterized by strict sexual norms that buffer against HIV transmission. For example, many note that Muslim nations tend to have much lower rates of HIV prevalence (e.g. Grey 2004; MacIntosh and Thomas 2004; Burroway 2010), and thus represents another important factor to be taken into account in the analyses.

Based on these assessments, it is clear that HIV/AIDS has higher rates of occurrence in the most underdeveloped of nations and for the most vulnerable populations within these nations (such as women). Indeed poverty is often cited as the key cause of HIV transmission, as poor nations have a higher percentage of people who engage in risky sexual behavior (Heimer 2007). This proves to be especially true for women, who may be forced to engage in sex for money or due to their social positions. Although GDP per capita is generally used to capture
“poverty” (e.g. MacIntosh and Thomas 2004; Burrowary 2010), it is clear from the case study literature that poverty largely operates through the propensity for unsafe sexual practices, largely for women, as they are more efficient transmitters of the virus and less likely to receive diagnosis and care. Additionally, proxies for health care, such as access to doctors, are most likely influenced by economic growth and other development characteristics, as the theorizations discussed in the prior chapters emphasized. Even though the causes of HIV prevalence are expected to follow both direct and indirect paths, current modeling strategies have not appropriately accounted for this. Furthermore, it is likely that some of the structural dynamics operate differently in Sub-Saharan African nations, yet cross-national researchers have only analyzed samples of all less-developed nations (e.g. Burrowary 2010; Shircliff and Shandra 2011; MacIntosh and Thomas 2004). Although regional interaction terms are often employed, these do not fully capture the differential effects that exist across multiple variables, or the unique effects that might only occur for a single group. I address these concerns by analyzing two samples, a sample of less-developed nations and a sample of Sub-Saharan African nations, and by appropriately modeling both direct and indirect effects on HIV rates.

3.3 Hypotheses

Review of the modernization and world-systems literatures, as well as the emerging evidence on urban migration leads to the construction of several hypotheses. First, (H1) I predict that female empowerment, captured through dimensions of female schooling, use of contraceptives, and reduced fertility, has a direct negative influence on HIV rates. Additionally,
(H2) I predict that social-health capabilities (such as access to doctors) also have a direct negative influence on HIV rates and an indirect influence through enhancing female empowerment. Also following modernization themes, (H3) I predict that GDP capita has direct negative influences on HIV rates, as well as indirect influences through the beneficial effects of economic development on social-health capabilities and female empowerment.

Another important set of predictions surrounds processes of urbanization. Consistent with ideas from modernization theory, (H4) I predict that level of urbanization increases access to health resources; however, I also postulate that (H5) high levels of urban growth reduce access to social-health capabilities. Additionally, (H6) I predict that high levels of urban growth increase HIV rates. Engaging dependency/world-systems theorizations, (H7) I also postulate that environmental changes associated with climate change, particularly drought, increase rates of urban growth, thus having important indirect influences on HIV prevalence.

Lastly, many studies have demonstrated that cultural context at least partly contributes to HIV trends (e.g. Gray 2004; MacIntosh and Thomas 2004), where Muslim nations have much lower rates of prevalence, given their strict norms regarding sex behavior. Thus (H8) I predict that percent Muslim will have a direct negative influence on HIV prevalence, and also an indirect association by reducing female empowerment. As Sub-Saharan African nations may exhibit some unique characteristics, I will test these hypotheses across two samples.
3.4 Methods

Samples. Given the high rates of prevalence of HIV in Sub-Saharan Africa, as well as the sub-regional evidence that suggests differential relationships surrounding female empowerment and urban growth, it is necessary to utilize two samples. Both samples are restricted to less-developed nations as the structural processes causing HIV prevalence differ greatly across more- and less-developed nations. In addition, HIV is comparatively very rare in affluent nations. Less-developed nations can defined as nations in the lower three quartiles of the income classification of countries (World Bank 2010). The first sample is comprised of 101 less-developed nations. It includes nations from Latin America, Southeast Asia, Sub-Saharan Africa, and other developing regions. The second sample is restricted to 41 Sub-Saharan African nations. For a complete list of nations included in the samples, see Table 3.1.

20 Although there were some missing data points, there appeared to be no pattern to the missing values that would bias results. Utilizing the strengths of the analytic technique, I treat missing values by using full information maximum likelihood, where all available data is used to generate maximum likelihood based statistics. I also conducted the analyses using listwise deletion strategies, and achieved consistent results; however the sample size was greatly reduced, and thus I prefer to report the model utilizing data from as many cases as possible here.

21 The samples used in the dissertation differ across the chapters due to the different dependent variables utilized and in order to maximize sample size across each analysis. Conducting the analyses across uniform samples did not alter any of the substantive findings reported here, however I prefer to report the results that are based on as much information (data) as possible.

22 I also excluded Sub-Saharan African nations from this sample to confirm that their inclusion was not leading to erroneous conclusions. Although the model was consistent across all less-developed nations and non-African less-developed nations, I choose to present the results for the full sample of less-developed nations here as it produced better fit statistics based on the larger sample size. In addition, Sub-Saharan African nations represent an important sub-set of less-developed nations and although they do warrant their own investigation, Sub-Saharan African nations also contribute to dynamics across less-developed nations that should be accounted for. Sub-Saharan African nations are thus retained in the full sample of less-developed nations for important empirical and theoretical reasons.

23 Although there were some missing data points, there appeared to be no pattern to the missing values that would bias results. Utilizing the strengths of the analytic technique, I treat missing values by using full information maximum likelihood, where all available data are used to generate maximum likelihood based statistics. I also conducted the analyses using listwise deletion strategies, and achieved consistent results; however the sample size was greatly reduced, and thus I prefer to report the model utilizing data from as many cases as possible here.
Analytic Strategy. I utilize structural equation modeling (SEM) to assess the cross-national predictors of HIV prevalence. SEM is a powerful extension of multiple regression, and for a number of reasons is particularly well-suited to the analysis. First, SEM allows for the inclusion of latent, or composite, variables. This feature in principle utilizes the strengths of both factor analysis and predictive modeling. This is especially relevant when measuring factors, such as “female empowerment,” which may be comprised of a number of indicators, which are technically justified by the confirmatory factor analysis incorporated in SEM (Bollen 1989). Secondly, although the theorization on development and disease specifies a bundle of direct and indirect effects, the dominant methodologies used in comparative assessments of well-being rely on direct effects only models (e.g., OLS regression, panel regression). However, SEM techniques allow for the specification of direct and indirect effects, which more accurately represent the complex theorizations discussed earlier and the hypotheses I endeavor to test. Third, these characteristics allow the estimation of unbiased coefficients even in the presence of highly correlated independent variables, which is often considered to be the “bane of cross-national analyses,” and can contribute to non-significance or non-inclusion of highly-relevant factors. Next, SEM explicitly models measurement error, lessening any bias in estimates that could result from a failure to specify those disturbances (Bollen 1989). SEM also provides fit statistics for the model as a whole, allowing the researcher to judge the fit of the model specified to the data provided (Bollen 1989). Taken as a whole, these features make SEM the most appropriate methodological approach for this line of research.
Dependent Variable. The key dependent variable is HIV Prevalence for the year 2006. The data were obtained from the World Health Organization Statistical Information System (2011). This measure represents a rate, capturing the number of people aged 15-49 infected per 100,000. This measure was log-transformed to reduce the influence of extreme outliers.24

Independent Variables. As the issue of gender inequality is especially relevant to the HIV pandemic, measures that capture female empowerment are central to the analysis. I will measure female empowerment with a latent or composite construct drawing on variables of contraceptive prevalence, female participation in secondary education, and the fertility rate for the year 2000. Together, these represent the indicators that are most commonly used to capture female empowerment (e.g. Shircliff and Shandra 2011; Wickrama and Lorenz 2002). Data were acquired from the World Bank (2011). The fertility rate is an estimate of the number of children an average woman would have if current age-specific fertility rates remained constant during her reproductive years. Contraceptive prevalence represents the percentage of women ages 15-49 who are practicing, or whose sexual partners are practicing, any form of contraception. I include female participation in education with ratio of female to male secondary school enrollment. This measure represents the ratio of the female to male gross enrollment rates that correspond to secondary level education.

In addition to gender equality, access to social-health capabilities that promote more healthful behaviors or knowledge of HIV issues is likely to be a significant factor associated with

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24 Although negative binomial regression can be used when the dependent variable is highly skewed, this form of regression is not available through SEM. To ensure that skewness is not biasing the results, I tested models with the natural log of HIV rates and with raw HIV rates. Both models produced consistent covariate effects; however the model with the natural log of malaria rates achieved better model fit statistics, and thus is the model presented here.
HIV prevalence in less-developed nations. Specifically, secondary-level education has been consistently shown in prior research to directly reduce HIV rates (e.g. Burroway 2010). I thus include the secondary school enrollment, measured as a gross enrollment ratio, which calculates the ratio of total enrollment, regardless of age, to the population of the age group that officially corresponds to secondary level education for the year 2000. In addition, access to physicians is another major health resource that greatly increases people’s knowledge of HIV and access to condoms or other preventative strategies (e.g. Burroway 2010; Soares 2007). I thus include number of physicians, which measures the number of physicians in a nation per 100,000 people for the year 2000. This measure was log-transformed to reduce the influence of extreme outliers.

I measure level of economic development using GDP per capita PPP, which is the total annual output of a country’s economy, in current international dollars, per person, for the year 2000 (World Bank 2010). GDP per capita is the total market value of all final goods and services produced in a country in a given year, equal to total consumer, investment, and government spending, divided by the mid-year population. It is converted into current international dollars using Purchasing Power Parity (PPP) rates, which provides a standard measure allowing for comparisons of real price levels between countries.

In order to examine the debates in the literature surrounding urbanization and HIV, I include level of urbanization and the urban growth rate for the year 2000. Percent urban refers to the proportion of the population living in urban areas as defined by national statistical offices. Urban growth represents the annual growth rate in the population living in urban areas.
Some studies point to environmental change as a cause of rural-to-urban migration, and therefore contributing to high levels of urban growth. I follow prior studies examining environmental drivers of migration and include percent affected by drought. To create this measure, I weighted data on the total number of people affected by drought from the years 1990-1999 by the total population level. The drought data are taken from the EM-DAT International Disaster Database and were measured across the years 1990-1999 in order to avoid idiosyncrasies that result from extreme droughts for a certain year, and to take into account the cumulative impact on migration that prolonged drought could have over several growing seasons. In EM-DAT, the number affected by drought reflects the number of people that have required immediate assistance (i.e. requiring basic survival needs such as food, water, shelter, sanitation and immediate medical assistance), been injured, and/or left homeless due to drought.

Lastly, prior examinations of HIV have demonstrated that cultural or religious context can have a major influence on prevalence rates (e.g. Burroway 2010, MacIntosh and Thomas 2004; Gray 2004). In particular, Muslim populations tend to have lower rates of HIV transmission, due to strict cultural norms regarding sexual behavior. I thus include percent Muslim in the analyses. These data were taken from the CIA Factbook (2011).

3.5 Results

Table 3.2 provides the means, standard deviations, and bivariate correlations for all variables included in the analysis. These are provided as a preliminary test of multicollinearity among central predictors. Although it can be difficult to confirm the existence of
multicollinearity based solely on correlations among variables, the magnitude of the relationships among the variables demonstrates a high probability that multicollinearity is an issue for the data used, especially among the development and gender equality indicators. This further warrants the use of the SEM analytical technique given its superior handling of correlated independent variables through the creation of latent constructs and direct and indirect pathways that circumvents the tendency to bias coefficient estimates.

Figure 3.1 is a graphical representation for the sample of less-developed nations of the model derived from the theoretically specified relationships outlined above and the model building and trimming techniques as discussed by Byrne (2009).\textsuperscript{25} Before examining the relationships displayed in the model, it is essential that the model fit statistics are deemed to be in an acceptable range. Inspection of the model fit statistics for Figure 3.1 reveals that the chi-square (40.036, df = 36, not significant), RMSEA (0.021), IFI (.995), TLI (.989), and CFI (.995) demonstrate a model well within the desired ranges for a “good” fit to the data. Attaining satisfactory model fit statistics permit interpretation of the pathway coefficients. With the exception of the paths from percent Muslim to social-social-health capabilities and from percent Muslim to female empowerment, all of the paths are statistically significant at the .01 level. The paths from percent Muslim to social-health capabilities and female empowerment are only statistically significant at the .10 level; however, given the limited sample size, their substantive

\textsuperscript{25} Following the advice offered by Byrne (2009), each plausible causal relationship was examined in the model. Virtually all non-significant paths are deleted simply because they show no statistical relationship to the dependent variable of interest. Among a few indicators, coefficients were moderately large, but inclusion of the predictor suggested poor model fit. Thus, the model presented here is comprehensive in that it includes all possibly significant paths, as well as parsimonious, as any non-relevant paths or factors are appropriately trimmed from the model.
contributions, and the improvement in model fit when included, these causal pathways are retained in the final model. Standardized regression coefficients are reported.

Results presented in Figure 3.1 depict that percent Muslim (-.41) and female empowerment (-.63) have robust negative direct effects on HIV prevalence in less-developed nations. Somewhat surprisingly, these factors represent the only variables to have a direct influence on HIV rates; all other predictors largely operate through female empowerment. In particular, social-health capabilities, captured specifically with participation in secondary schooling and access to physicians, are shown to greatly increase female empowerment (.92). Indeed, despite the near identification between these two factors, model fit statistics confirm the representation of social-health capabilities and female empowerment as two distinct components, where schooling and doctors only reduce HIV prevalence through their effects on increasing female access to contraceptives, higher education, and reduced fertility. GDP per capita also has no direct effect on HIV prevalence (or female empowerment), but largely only contributes through promoting social-health capabilities (.30). Additionally, while percent Muslim has strong direct effects on reducing HIV prevalence (-.41), Muslim societies are also shown to have lower levels of health capability (-.12) and female empowerment (-.07). Thus, percent Muslim also has modest positive indirect effects on HIV rates in less-developed nations.

GDP per capita also has beneficial indirect effects on HIV by increasing the level of urbanization (.63). Interestingly, level of urbanization is associated with increased access to educational and medical resources (.26); however, high rates of urban growth serve to depress

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26 In addition, excluding these pathways did not alter the relationships evidenced among the other variables, further warranting their inclusion.
access to educational and medial resources (-.36). The negative relationship between percent urban and urban growth (-.49) demonstrates that the least urbanized less-developed nations are those that are experiencing the fastest rates of growth. These findings help to clarify debates that surround urbanization and health in less-developed nations; while more urbanized societies overall tend to have increased access to social-health capabilities, consistent with modernization approaches, very high rates of urban growth reduce access to schooling and physicians, most likely as population inflows strain public resources. Furthermore, high levels of urban growth are spurred by environmental decline associated with climate change (.33), thus also providing evidence for world-systems/dependency approaches to over-urbanization in less-developed nations.

Although a number of important structural relationships were demonstrated in Figure 3.1 for less-developed nations, it is also necessary to examine these dynamics within Sub-Saharan Africa, as many studies suggest unique effects of urban growth and female empowerment within this region (e.g. Rushing 1999; Dyson 2003). Figure 3.2 presents the results for the sub-sample of Sub-Saharan African nations. It is first important to note that the model fit statistics are in an acceptable range, and in fact, illustrate an excellent fit between the model presented and the data analyzed. Indeed, the chi-square (15.55, df = 16, not significant), RMSEA (0.000), IFI (1.04), TLI (1.01), and CFI (1.00) fit statistics demonstrate a model with exceptional fit to the data. All of the paths presented in Figure 3.2 are statistically significant at the .001 level, with standardized regression coefficients reported.
The results presented in Figure 3.2 depict that some of the relationships identified for the sample of less-developed nations operate in different ways for Sub-Saharan African nations. Despite that the diagram looks significantly altered, there are also a number of consistencies across the two models, namely concerning the effects of percent Muslim, GDP per capita, and environmental change. Taking a look at the results presented in Figure 3.2, what is most interesting to note are the unique effects of urban growth. The results demonstrate that urban growth has a robust direct effect on HIV prevalence in Sub-Saharan African nations, where high rates of urbanization are associated with furthering HIV rates (.46). Consistent with the case-study evidence, female empowerment increases urban growth in Sub-Saharan African nations (.31). Additionally, there is now no direct effect of female empowerment on HIV rates; although the path did approach statistical significance, it appears that the beneficial effects of female empowerment are largely cancelled out by the movement to urban areas in Sub-Saharan Africa. Consistent with the earlier model, percent affected by drought has a strong effect on urban growth (.53). Additionally, Muslim directly reduces HIV rates (.44), but also indirectly influences HIV prevalence by greatly reducing female empowerment (-.33) in Sub-Saharan African nations. Curiously, this may be somewhat beneficial in this context, as female empowerment is linked to higher rates of urbanization and increased HIV. Lastly, GDP is once again found to have no direct bearing on HIV rates, but does significantly contribute to

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27 Unfortunately, social-health capabilities, measured through access to doctors and participation in schooling, could not be included in this model due to poor model fit. The small sample size could be a contributing to this, as well as substantive conclusions that these factors don’t matter as much as the other aspects of health resources that are included in the model (such as female schooling, contraceptive prevalence, and fertility) for Sub-Saharan African nations. Additionally, percent urban is not included in this model; although the model fit statistics were not impaired with the inclusion of this variable, it was found to be non-significant. Thus, high levels of urban growth are likely to be taking place across the majority of Sub-Saharan African nations, not just the ones that are least urbanized.
increasing women’s empowerment in Sub-Saharan African nations. In this way, level of economic development is positively associated with HIV prevalence in Sub-Saharan Africa.

Taken together, the results illustrate that elevated rates of urban growth play a unique role in contributing to HIV prevalence across less-developed nations and within the region of Sub-Saharan Africa. Environmental change proves to be a significant cause of urban growth, and urban growth itself directly contributes to HIV rates in Sub-Saharan African nations. In less-developed nations, urban growth is also shown to reduce access to health-promoting resources, thus having indirect positive effects on HIV prevalence. Additionally, while female empowerment is generally linked to lowering HIV rates in less-developed nations, it serves to foster urban migration particularly among Sub-Saharan African nations, thus having notable indirect harmful effects on HIV prevalence in this specific context. Cultural or religious underpinnings are also very important determinants of HIV, as Muslim nations have much lower rates of prevalence, and also indirectly contribute through reducing female empowerment.

3.6 Conclusion

HIV is unique from the other diseases studied here as it is a sexually transmitted virus. Thus, issues of female empowerment become paramount, especially as reductions in fertility and increased use of contraceptives should directly translate into lower levels of HIV transmission. While this is generally true, and evidenced here in the sample of less-developed nations, female empowerment is also shown to be a significant factor spurring urban growth in Sub-Saharan African nations. The movement and concentration of people itself is a demographic or
epidemiological phenomenon that fundamentally increases possibilities for disease transmission, especially in nations characterized by a high level of prevalence (e.g., Dyson 2003). Consequently, while factors such as using contraceptives inevitably reduce HIV transmission, processes of female empowerment are likely to spur population flows to cities, where urban environments afford more diversity in sex partners, cancelling out the beneficial effects of increased female empowerment that would prevent against HIV infection.

These remarkable findings for the sample of Sub-Saharan African nations get at the heart of debates within the limited branch of comparative analysis on HIV, as one study has found economic growth to be positively associated with HIV rates in less-developed nations (e.g., Shircliff and Shandra 2011), and another concluded that schooling isn’t as effective in reducing disease in Sub-Saharan Africa as in other regions (e.g., Burroway 2010). Although the links between female empowerment, urbanization, and HIV prevalence have garnered some attention from researchers within specific regions of Sub-Saharan Africa (e.g., Rushing 1999; Dyson 2003; Gushulak and MacPherson 2004; Gutmann 2007), this study demonstrates that these findings can be generalized to Sub-Saharan African nations on the whole, despite that these processes may be unique to this region. The differential effects for urbanization growth on HIV within Sub-Saharan Africa could be partially due to the level of infection; given that Sub-Saharan Africa nations have such high rates of HIV to begin with, population flows will have a much more profound influence on disease rates than in nations with lower levels of prevalence. In this way, issues of urban growth could be relevant to other nations that have specifically high rates of urbanization and/or HIV prevalence, such as those in the Caribbean. Thus, whether or not
there is some kind of “threshold effect” taking place across these factors is one avenue for future research. Additionally, Sub-Saharan African nations tend to be more traditional and rural than other nations, perhaps causing social changes related to enhanced female empowerment and high levels of urban growth to have especially profound effects on sexual behavior.

Indeed, many less-developed nations are rapidly urbanizing and analyses such as these provide keen insight into the reasons behind intense urbanization, namely, environmental alterations associated with climate change. In fact, the region of Sub-Saharan Africa has the highest growth in urban population in the world, and it is here that we also see the relationship between drought and urban growth to be exceptionally strong (.53). Although prior studies of urban growth have largely centered on the economic motivations for migration to urban areas, it is clear to see that patterns of urban growth are fundamentally tied the natural environment, where rural people who are dependent on the land may be forced to move as a coping strategy in the face of environmental decline. While researchers have identified environmental changes to be a factor spurring migration in localized studies, this chapter represents the first to demonstrate this phenomenon on a cross-national scale. Indeed, the very fact that these trends can be evidenced at the national levels signifies the importance of environmental conditions in contributing to harmful patterns of urban development, and therefore infectious disease.

It is also noteworthy that the findings evidenced among less-developed nations for urbanization effects on health-promoting resources greatly speaks to important theorizations on modernization and dependency. While modernization theorists have long touted urbanization as a sign of development and a means to improve health, world-systems/dependency scholars are
cautious to note that patterns of neoliberal economic development and environmental change often lead to over-urbanization in less-developed societies. There is strong evidence for both lines of thinking, which maybe are not completely incompatible. The results demonstrate that more urbanized nations tend to have greater access to health resources, but that very high levels of urban growth strain health resources. Also, the direct link between urban growth and HIV prevalence among Sub-Saharan African nations cannot be understated. Thus, we see signs of both modernization and dependency in explaining urbanization-effects on health.

Although there is support for modernization theory in this regard, this perspective falls short in a few other areas. Most prominently, economic development or GDP per capita, does not directly translate into improved health. Rather, it only reduces HIV rates through its effects on social-health capabilities among less-developed nations. The Sub-Saharan Africa models also depict an indirect, positive effect of GDP per capita on HIV through female empowerment. Thus, GDP per capita gains do not necessarily or inherently trickle-down to benefit the masses. Furthermore, both models demonstrate that it is dangerous to pursue a blind focus on economic growth, urbanization, or female empowerment as means to improve human welfare. While these factors may be associated with improving health and well-being in certain settings or at certain points in history; politicians, activists, and researchers alike need to have greater awareness of the local or regional context, and how altering these characteristics might impact other realms (such as migration flows or sexual norms) in ways that compromise health. Thus, neoliberal approaches that place unduly emphasis on economic growth and urban-industrial expansion could be sacrificing long-term potential for successful development.
These analyses most significantly illustrate that the factors explaining HIV prevalence across nations are not clear-cut, and at times, do not fit neatly into modernization or world-systems perspectives on development and health. There are complex relationships among economic indicators, urban growth, and female empowerment that play out differently in Sub-Saharan Africa than in other regions. Indeed, the common-sense recommendations for curbing HIV do not work in the ways that we expect them to in Sub-Saharan Africa nations. This not only questions a blind faith in these forms of modernization and public policy intervention, but demonstrates that we need to be careful to not take a one-sided approach to this pandemic.

While sex education and contraceptives seem to be helpful in reducing HIV rates in Latin American nations (e.g. Burroway 2010; Gaillard 2006), these strategies cannot be extended to Sub-Saharan African populations with similar success. This region is historically much more rural and traditional than other developing zones. Thus, the opening up of villages and tribes that occurs with modernization and increased gender equality will potentially have different ramifications for disease than elsewhere. Indeed, tracking the trends in urban development will likely be of upmost importance to reducing the burden of this deadly infection, for Sub-Saharan African nations and other less-developed nations alike. The pernicious effects of urban growth on social-health capabilities speaks to important relationships that are likely to be underlying factors in the transmission of TB and malaria as well. Indeed, not only are less-developed regions rapidly urbanizing, but many cities are characterized by the proliferation of urban slums. This represents an extreme form of urbanization that is likely to have major ramifications for human health.
Table 3.1: Nations Included in the HIV Analyses

<table>
<thead>
<tr>
<th>Nation</th>
<th>Country</th>
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<tbody>
<tr>
<td>Algeria</td>
<td>Djibouti</td>
<td>Lesotho*</td>
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<td>Estonia</td>
<td>Mali*</td>
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<td>Gambia, The*</td>
<td>Mauritania*</td>
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<td>Cuba</td>
<td>Lebanon</td>
<td>Poland</td>
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Notes: Nations that are included in the Sub-Saharan Africa sample are denoted with an asterisk (N=41).
# Table 3.2: Univariate Statistics and Correlation Matrix for HIV Prevalence

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<thead>
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<th>9.</th>
<th>10.</th>
<th>11.</th>
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<tbody>
<tr>
<td>1. HIV Prevalence (ln)</td>
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<tr>
<td>2. Fertility Rate</td>
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<td>3. Female Tertiary Schooling</td>
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<td>-.686</td>
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<td>4. Contraceptive Prevalence</td>
<td>-.354</td>
<td>-.892</td>
<td>.761</td>
<td>1.00</td>
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<td>5. Secondary Schooling</td>
<td>-.159</td>
<td>-.875</td>
<td>.743</td>
<td>.877</td>
<td>1.00</td>
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<td>6. Physician Rate (ln)</td>
<td>-.244</td>
<td>-.669</td>
<td>.618</td>
<td>.692</td>
<td>.780</td>
<td>1.00</td>
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<td>7. Percent Urban</td>
<td>-.085</td>
<td>-.456</td>
<td>.673</td>
<td>.439</td>
<td>.574</td>
<td>.666</td>
<td>1.00</td>
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<td>8. Urban Growth Rate</td>
<td>.224</td>
<td>-.201</td>
<td>.189</td>
<td>.198</td>
<td>.220</td>
<td>.087</td>
<td>-.757</td>
<td>1.00</td>
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<td>9. Percent Affected by Drought (ln)</td>
<td>.419</td>
<td>.451</td>
<td>-.523</td>
<td>-.561</td>
<td>-.621</td>
<td>-.573</td>
<td>-.558</td>
<td>.483</td>
<td>1.00</td>
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<td>10. GDP per capita</td>
<td>-.021</td>
<td>-.549</td>
<td>.579</td>
<td>.568</td>
<td>.718</td>
<td>.502</td>
<td>.679</td>
<td>-.470</td>
<td>-.467</td>
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<td>11. Percent Muslim</td>
<td>-.243</td>
<td>.725</td>
<td>-.362</td>
<td>-.707</td>
<td>-.665</td>
<td>-.398</td>
<td>-.002</td>
<td>.021</td>
<td>.183</td>
<td>-.446</td>
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<td>Mean</td>
<td>-.197</td>
<td>3.77</td>
<td>86.67</td>
<td>42.36</td>
<td>52.88</td>
<td>1.43</td>
<td>43.45</td>
<td>2.44</td>
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<td>3779</td>
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<td>Standard Deviation</td>
<td>1.56</td>
<td>1.72</td>
<td>46.01</td>
<td>24.59</td>
<td>29.11</td>
<td>1.49</td>
<td>20.11</td>
<td>1.03</td>
<td>.723</td>
<td>3279</td>
<td>37.38</td>
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Figure 3.1: SEM Predicting HIV Prevalence for Less-Developed Nations
Figure 3.2: SEM Predicting HIV Prevalence for Sub-Saharan African Nations
CHAPTER 4
ENVIRONMENTAL CHANGE, URBAN SLUMS,
AND THE FORGOTTEN PLAGUES: TUBERCULOSIS AND MALARIA

4.1 Introduction

Tuberculosis and malaria not only represent a primary cause of death among poor societies today, but they also represent leading killers in world history. This is true for TB in particular, which was responsible for about one in four deaths throughout the 19th century (Farmer 2000; Gandy and Zamula 2002). Although TB and malaria’s victims during this historical period included members of all nations, these diseases have always disproportionately afflicted disadvantaged groups. For example, in the United States in 1900 the annual TB death rate among whites was 200 per 100,000, while among African Americans the TB death rate was 400 per 100,000. Poverty continues to be cited as the key risk factor for acquiring and dying from these diseases (e.g. Bates et al. 2004; Farmer 2000, 2001), and this is clearly evidenced in current cross-national trends, where the overwhelming majority of global TB and malaria cases occur in Sub-Saharan Africa, Southeast Asia, and the Pacific.

In fact, TB and malaria prevalence rates are so highly characterized by international development trends that these diseases largely fail to afflict people in affluent nations today. Malaria was eradicated from the United States in the 1960s, and TB rates diminished with the development of antibiotics and a vaccine in the 1940s. However, many note that despite major medical interventions, TB and malaria didn’t disappear; these diseases continue as major threats
to development and well-being in poor nations. In fact, TB and malaria now afflict more people than at any previous period of human history (Gandy and Zamula 2002; WHO 2011b). The ‘forgotten plagues’ have been forgotten in large part because they have ceased to bother the wealthy. As Paul Farmer (2000: 185) writes, “One place for diseases like TB to hide is among poor people.”

Although malaria and TB are often thought of as a “forgotten” or “old” diseases (e.g. Packard 2009, Farmer 2000, 2001), cross-national data illustrate that many parts of the world-system are experiencing a resurgence of these infections. In 2010 there were over 9 million new cases of TB diagnosed, and malaria rates have increased by around 30% since 1990 in many Sub-Saharan African and Southeast Asian nations (WHO 2011a). A leading factor in explaining the reemergence of TB and malaria is the advent of antibiotic resistant strains of both infections (Bates et al. 2004; Kim et al. 2005). Resistance to antibiotics typically develops when the parasite or virus is exposed to sub-optimum concentrations of drugs, which may result from inappropriate prescribing or non-adherence to treatment (e.g. Bates et al. 2004). While the majority of studies blame patients for antibiotic resistance, due to their failure to observe treatments, this research does not adequately take into account the costs and inequalities associated with health care for people in less-developed nations (Bates et al. 2004; Farmer 1997, 2000, 2001; Gandy and Zamula 2002). Indeed, although most focus on the biological pathways through which antibiotic resistance to malaria and TB develops, it is clear to see that issues of access to high-quality health resources are fundamentally tied to global inequality dynamics.
As current developmental strategies prioritize market liberalization over provisions for public resources for health (e.g. Stiglitz 2002, 2007), the resurgence of TB and malaria is likely to be intimately connected to current cross-national development dynamics. The prior chapter demonstrated that there are likely to be unique relationships among GDP per capita, health resources, and disease. This is especially relevant for TB and malaria, as the epidemiology of these diseases places greater importance on basic issues of sanitation and infrastructure. Given the extremely high degree of overlap among these two infections, it is plausible to model TB and malaria together.  

Doing so would illuminate a common set of factors that predispose poor nations to communicable infections. Additionally, urbanization is predicted to be a consistent theme from the prior chapter; many of the most current studies note the encroachment of malaria into urban areas, for similar reasons that would also promote TB transmission, including slum expansion characterized by inadequate housing, crowding, and issues of sanitation and sewage (e.g. Davis 2006; Moore et al. 2003). Urban slums represent a powerful manifestation of underdevelopment that we see taking shape in many rapidly urbanizing poor nations, and this is likely linked to environmental conditions and changing climates that threaten rural livelihoods. Although rural people seek urban areas as a coping strategy in the face of major environmental changes, the linkages between urban growth and disease make this strategy one that is not likely to improve well-being.  

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28 Indeed, although this strategy was pursued for HIV as well, the fact that HIV is a sexually transmitted infection made the proximate causes of HIV differ from TB and malaria, which, although different diseases themselves, prove to be much more similar in their proximate causes than HIV.
4.2 The Forgotten Plagues and Patterns of Underdevelopment

TB and malaria epidemiologically represent two very different infections. The tubercle bacillus, or the TB virus, is a contagious respiratory infection transmitted through intimate contact, body fluids including saliva, or in the air by coughing (WHO 2011b; Kim et al. 2005). In contrast, malaria is a parasitic disease transmitted by mosquitoes (WHO 2011c; Norris 2004). Despite the fact that the biological mechanics of these diseases contrasts greatly, both infections are highly correlated with poverty (e.g. Bates et al. 2004; Packard 2009; Farmer 2001). Although issues of poverty in the prior chapter included relevant social dimensions of gender equality, absolute deprivation in basic infrastructure seem more proximate to the infections of TB and malaria. For example, issues of sanitation and housing have been identified in prior studies as especially important factors related to TB and malaria transmission in poor nations (Corbett et al. 2003; Williams 2009; Antunes and Waldman 2001; Norris 2004). Additionally, the development of antibiotic resistance points to the robust influence of access to health resources in curbing disease rates (e.g. Bates et al. 2004).

TB is caused by various strains of mycobacteria. Over one-third of people globally have Mycobacterium tuberculosis (M-TB), the latent organism that causes the TB virus (WHO 2011b; Kim et al. 2005). Tuberculosis usually attacks the lungs but can also affect other parts of the body (WHO 2011b). It is spread through the air when people who have an active MTB infection cough, sneeze, or otherwise transmit their saliva to others. Most infections in humans result in an asymptomatic, latent infection, but about one in ten latent infections eventually
progress to active disease, which, if left untreated, kills more than 50% of those infected (WHO 2011b).

The classic symptoms of TB are a chronic cough with blood-tinged sputum, fever, night sweats, and severe weight loss, which gave rise to the formerly prevalent colloquial term for TB, “consumption” (WHO 2011b; Farmer 2000). TB is truly an old disease as skeletal remains show prehistoric humans likely had TB, due to evidence of tubercular decay found in the spines of Egyptian mummies from 3000-2400 BC. Tuberculosis caused the most widespread public concern in the 19th and early 20th centuries as an endemic disease of the urban poor. It was not until 1940s with the development of a vaccine and the antibiotic streptomycin that effective prevention and treatment became possible (WHO 2011b). However, new trends in antibiotic resistance threaten possibilities for curing this deadly virus (Bates et al. 2004).

Many studies reveal that rates of TB are increasing in many less-developed nations (WHO 2011a, Gandy and Zamula 2002; Antunes and Waldman 2001). Medical explanations describe that increasing prevalence of antibiotic resistant TB and interactions with the HIV infection explain trends of increased incidence (Gandy and Zamula 2002; Farmer 2000; Bates et al 2004; Kim et al. 2005). Although infected Mycobacterium tuberculosis (M-TB) has effective medical interventions, they are rather intensive. Treatment is difficult due to the unusual structure and chemical composition of the mycobacterial cell wall, which makes many antibiotics ineffective and hinders the entry of drugs (WHO 2011b). Additionally, M-TB itself has a propensity to acquire drug-resistance to a single agent and must be used in tandem with multiple antibiotics for a significant amount of time to be completely effective. Standard “short-course”
treatment for TB consists of 6 months of therapy using four different first-line anti-TB drugs (Kim et al. 2005).

However, a growing proportion of cases are diagnosed as multidrug-resistant tuberculosis (MDR-TB), which is highly resistant to the gamut of first-strain antibiotics and some secondary interventions, and thus much more expensive to treat, with costs mounting to around $30 per day (Kim et al. 2005). Since a little more than a dozen effective anti-TB agents are presently in use, the emergence of super-strains resistant to all known drugs is a serious threat (Kim et al. 2005). Although it is not the only source, M-TB can become MDR-TB if people receive intermittent and/or poorly executed therapy (Bates et al. 2004; Kim et al. 2005). As poor nations have inadequate health systems that promote unsatisfactory interventions or lack ample supplies of drugs, it is likely that the emergence of MDR-TB is not solely a medical phenomenon, but linked to structural inequalities and issues of persistent poverty in less-developed nations. In fact, some studies report that only around 15 percent of patients worldwide receive adequately supervised TB treatments (Gandy and Zumla 2002). However, development of MDR-TB in patients is often portrayed as their failure to respond to therapy. The patient’s agency – their ability to comply with costly and difficult regimens – is exaggerated in many medical reports (Farmer 1997, 2000, 2001; Gandy and Zamula 2002).

Malaria shares a similar history; it is also a very old infection, and poses new challenges as parallel issues with antibiotic resistance threatens the ability to successfully treat it. It was once thought that malaria came from fetid marshes, hence the name ‘mal-aria,’ which means ‘bad air’ (WHO 2011c). However, in 1880 scientists discovered the real cause of malaria: a one-
cell parasite called plasmodium. Later they found that the parasite is transmitted from person to person through the bite of a female Anopheles mosquito, which requires blood to nurture her eggs (Norris 2004). Today approximately 3 billion people are at risk of acquiring malaria (WHO 2011c). The disease was once more widespread, but it was successfully eliminated from many countries with temperate climates during the mid-20th century.

The malaria parasite enters the human host when an infected Anopheles mosquito takes a blood meal (WHO 2011c). Inside the human host, the parasite undergoes a series of changes as part of its complex life-cycle. The various stages allow the plasmodia parasite to evade the immune system, infect the liver and red blood cells, and finally develop into a form that is able to infect a mosquito again when it bites an infected person. Inside the mosquito, the parasite matures until it reaches the sexual stage where it can again infect a human host when the mosquito takes her next blood meal, 10 to 14 days later (WHO 2011c; Sachs and Malaney 2002).

Malaria symptoms appear about 9 to 14 days after the infectious mosquito bite. Typically, malaria produces fever, headache, vomiting and other flu-like symptoms (WHO 2011c). If drugs are not available for treatment or the parasites are resistant to them, the infection can progress rapidly to become life-threatening. Malaria kills by infecting and destroying red blood cells (anemia) and by clogging the capillaries that carry blood to the brain or other vital organs (WHO 2011c). Thus, many people who survive an episode of severe malaria may suffer from neurological disorders, including learning disabilities, reduced fine motor functions, executive thinking impairment, or other forms of brain damage (Sachs and Malaney 2002; Pattanayak et al. 2006). Children are at heightened risk for malaria infection; it is
estimated that malaria kills an African child every 30 seconds (Breman et al. 2004). Pregnant
women and their unborn children are also particularly vulnerable to malaria, which is a major
cause of prenatal mortality and low birth weight among newborns (Sachs and Malaney 2002;
Pattanayak et al. 2006).

The most common preventative measures for malaria include the use of insecticide-
treated mosquito bed nets and the application of indoor insecticides to reduce the likelihood of
mosquito bites (Breman et al. 2007; Pattanayak et al. 2006; Sachs and Malaney 2002; Williams et
al. 2009). Once infected with malaria, people can often be successfully treated with antibiotics.
However, many insecticides are no longer useful against mosquitoes transmitting the disease and
malaria parasites are developing dangerous levels of resistance to antibiotics (Breman et al. 2007;
Pattanayak et al. 2006; Bates et al. 2004). Like TB, antibiotic resistance of the malaria parasite
develops when the parasite is exposed to sub-optimum concentrations of drugs or from
inappropriate prescribing or non-adherence to treatment schedules, which is common in less-
developed nations with inadequate health care systems. In general, the malaria virus has already
developed strong resistance to the cheapest interventions available, so successful treatment
requires more expensive and intensive antibiotic regimens (Bates et al. 2004; Pattanayak et al.
2006).

While vaccine research for malaria is on-going, research by Trouiller and colleagues
(2002) points out that the pharmaceutical industry is more concerned with developing profitable
vaccines and treatments for cancer and central nervous system disorders for afflictions that
characterize affluent nations; “Research and development is too costly and risky to invest in low-
return neglected diseases like malaria for poor nations” (Trouiller et al. 2002: 2188). So while years of under-financed vaccine research have produced few hopeful candidates, an effective vaccine for malaria is at best years away (WHO 2011c). Developed nations have been able to successfully eradicate the disease, largely due to the influence of favorable ecological factors (Sachs and Malaney 2002). The time period required for necessary life-cycle change in the mosquito increases as temperature declines, and given the life span of the mosquito, transmission becomes much less likely below a temperature of 64 degrees, and malaria parasites cease development completely at a temperature of 61 degrees. Thus, the base case reproduction rate of malaria is considerably lower in temperate regions than in the tropics, such that even moderately intensive efforts at vector control can lead to the elimination of the parasite (Sachs and Malaney 2002).

The advent of antibiotic resistant strains of TB and malaria are routinely used in reference to the ‘new face’ of infectious disease, yet they constitute only part of the changing picture (Farmer 2000). Rather, some continue to emphasize that the most critical factor contributing towards the re-emergence of these diseases is persistent poverty (e.g. Packard 2009; Gandy and Zamula 2002; Farmer 1999, 2000, 2001). “Poverty is the real reason why these infections are treatable in the U.S. and ‘untreatable’ in poor nations” (Farmer 2000: 209).

Poverty has been linked to prevalence of TB and malaria for several reasons, such as issues with overcrowding, high fertility, a lack of knowledge on disease transmission, and reduced access to health services, all of which characterize poor nations (Bates et al. 2004; Corbett et al. 2003; Gandy and Zamula 2002; Antunes and Waldman 2001). Gandy and Zamula
(2002) and Navarro (2002, 2007) point out that health care systems in less-developed nations may be increasingly inadequate as neoliberal economic policies and integration into foreign markets often leads to the reduction of public health provisions. Public health resources, such as improved sanitation and clean water access are especially important to consider given the epidemiological characteristics of TB and malaria, and access to physicians and education is especially relevant to the issue of antibiotic resistance, as proper knowledge of disease vectors and methods of Western medicine reduce prevalence and increase the success of treatments (e.g. Kim et al. 2005).

In addition to poor health resources, a second component of poverty in less-developed nations concerns expansion of urban poverty. Urban areas have historically been TB hotspots, as urban settings tend to concentrate people, and this virus is very easily transmitted through the air when coughing and by any type of fluid exchange (Antunes and Waldman 2001; Gandy and Zamula 2002). In poor nations and historically, sanitation and sewage can pose major problems in urban areas, contributing to disease prevalence (Davis 2006). An extensive study on urban TB prevalence conducted in Sao Paulo, Brazil by Antunes and Waldman (2001) illustrates that urban poverty is a continuing issue, as socio-economic position is a robust predictor, with much higher rates of morality in the most underprivileged areas of the city. Additionally, overcrowding at the household and neighborhood levels and immigration were highly predictive of TB mortality (Antunes and Waldman 2001).

A growing body of research also identifies the encroachment of malaria into urban environments (e.g. Donnelly et al. 2005; Pattanayek et al 2006; Norris 2004; Breman et al. 2004).
Given that malaria has clear ecological underpinnings as mosquitos serve as the key vector, this disease has in the past been considered a plague for rural populations who inherently live closer to mosquito habits, such as ponds and other slow-moving water bodies. Indeed, many contemporary studies continue to maintain that urban habitats do not adequately support mosquito populations (e.g. Hay et al. 2005; Pimentel et al. 2007), although the relationship between urbanization and malaria prevalence is becoming a topic of debate in the literature. While studies continue to illustrate that rates of malaria in urban areas are lower relative to rates of malaria in rural regions (e.g. Hay et al. 2005), some researchers point out that malaria incidence among urban populations is increasing in many nations (e.g. Donnelly et al. 2005; Pattanayek et al. 2006; Norris 2004; Breman et al. 2004).

Urban settings can be ripe environments for mosquito populations, especially in impoverished areas that have open water sources and sanitation streams or pits (Breman et al. 2004; Donnelly et al. 2005; Pattanayek et al. 2006). Poor water runoff and inadequate storm water handling systems can also be appropriate mosquito habitats in urban areas. In addition, artificial containers, such as bottle caps, discarded tires, and other forms of garbage are popular vectors (Norris 2004). Virtually any container or structure that can hold water has the potential to serve as mosquito habitat and be the source of large mosquito populations.

Thus, the proliferation of urban slums is likely to be a key factor associated with both TB and malaria rates. Urban slums are bourgeoning in less-developed nations, where 95% of the global slum population resides (Davis 2006; UN-HABITAT 2010). In fact, approximately one billion people, nearly one-third of the world’s urban population, live in slum conditions.
Slum conditions are characterized by substandard housing, crowding, inadequate access to sanitation and clean water, and tenure insecurity (Davis 2006; UN-HABITAT 2010; Jorgenson and Rice 2010), and living in such conditions greatly increases possibilities for disease transmission.

Urban slum proliferation and poor access to health-promoting resources in less-developed nations represent prominent symptoms of underdevelopment. While neoliberal modernization perspectives emphasize that increased economic growth through integration and specialization as mechanisms to enhance development and reduce poverty and disease, world-systems/dependency approaches emphasize that these forms of growth de-prioritize health resources and promote urban poverty, heightening disease rates in poor nations. In this way, environmental conditions become a re-occurring theme. Not only is the threat of rural livelihoods due to environmental change once again likely to be a factor in promoting urban slums (which is perhaps the most extreme form of urban growth), but urban slum conditions relate to the built environment, an important context within which the reproduction of disproportionate disease burden is enacted.

4.4 Hypotheses

One key explanation regarding heightened TB and malaria rates concerns the emergence of antibiotic resistant infections. Thus, (H1) I predict that social-health capabilities, specifically captured with components related to the availability of physicians, access to improved sanitation, participation in schooling, and reduced fertility have a robust negative influence on TB and
malaria rates in less-developed countries, as these factors have been shown to increase knowledge of disease vectors, use of preventative treatments (like bed nets), and reduce possibilities for transmission (e.g. Bates et al. 2004; Elo 2009). Additionally, (H2) I predict that the proportion of the urban population living in slum conditions represents an important element contributing to disease, both directly (through issues of crowding and inadequate housing) and indirectly by reducing access to social-health capabilities (such as schooling). Urban slums represent an extreme form of urban growth; thus it is likely that changing environmental conditions once again promote this form of urbanization. (H3) I therefore predict that drought is an important indirect influence on TB and malaria rates, where drought increases the proportion of people living in urban slums in less-developed nations.

I also postulate that a number of pertinent development and dependency variables influence the relationships outlined above, including characteristics such as world-system position, specialization in agriculture, and foreign debt. (H4) I also predict that GDP per capita has an indirect influence on disease through social-health capabilities, as was evidenced for HIV in the previous chapter. I will therefore also test for direct and indirect effects related to these variables and the key measures of interest.

4.5 Methods

Sample. The sample is restricted to 135 less-developed nations, reported in Table 4.1. As these diseases are extremely rare in affluent nations, the sample is naturally restricted based on data availability on TB and malaria to nations that fall within the lower three quartiles of the
income classification of countries, as defined by the World Bank (2010). Data on malaria cases is only provided for 102 malaria endemic nations. Malaria endemic can be defined as nations in which there is a constant measurable incidence both of cases of malaria and of natural transmission in an area (WHO Statistical Information System 2011). Countries that only have imported cases or introduced cases resulting from imported cases are not included in this data. Thus, any “missing values” for malaria prevalence for a case that was included in the sample based on TB prevalence data were recoded into zeros.

**Analytic Strategy.** I utilize structural equation modeling (SEM) to assess the cross-national predictors of TB and malaria prevalence, given the relevance of accounting for indirect and direct effects and the need to model highly inter-correlated predictors. SEM is also fundamentally required given the modeling of TB and malaria together as the key dependent variable. Indeed, limitations with multicollinearity in Chapter 2 demonstrated the need to model these diseases together in technically feasible ways.

**Dependent Variables.** The key dependent variable is represented in a latent construct derived from malaria prevalence and tuberculosis prevalence for the year 2006. Data on the number of TB and malaria cases per country were taken from the World Health Organization Statistical Information System (2011). These measures were transformed into a rate to capture the number of people infected per 100,000. This was done using total population data taken

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29 A sub-sample of Sub-Saharan African nations was also tested; the results were consistent across the two samples, indicating that there are not unique effects for Sub-Saharan Africa that need to be taken into account.
from the World Bank (2011) to control for the absolute population level of the reporting nation. The disease data are log-transformed to address issues of extreme skew.30

**Independent Variables.** Social-health capabilities, such as access to doctors or improved sanitation, are important factors that are likely to greatly influence TB and malaria rates in less-developed nations. I therefore measure social-health capabilities using a latent construct that is comprised of the following indicators: number of physicians, the fertility rate, percent of the total population with access to improved sanitation, and participation in secondary schooling. Each of these factors represent non-economic components of development that are demonstrated to reduce people’s risk of disease, largely by improving knowledge of disease vectors, reducing access to harmful vectors, and increasing use of preventative strategies (such as bed-nets) and methods of Western medicine (e.g. Soares 2007; Norris 2004; Farmer 2000; Elo 2009). Data were acquired from the World Bank (2011) and measured for the year 2005, except the sanitation variable, which was measured in 2006 to avoid confounding with the urban slums variable described below. **Number of physicians** measures the number of physicians in a nation per 100,000 people. This measure was log-transformed to reduce the influence of extreme outliers. **The fertility rate** is an estimate of the number of children an average woman would have if current age-specific fertility rates remained constant during her reproductive years.31 **Access to improved sanitation** measures the percentage of the

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30 Although negative binomial regression can be used when the dependent variable is highly skewed, this form of regression is not available through SEM. To ensure that skewness is not biasing the results, I tested models with the natural log of TB/malaria rates and with raw TB/malaria rates. Both models produced consistent covariate effects; however the model with the natural log of TB and malaria rates achieved better model fit statistics, and thus is the model presented here.

31 The fertility rate is represented in the following models as a component of social-health capabilities, although it was included in the female empowerment construct in the preceding chapter. Issues of female empowerment were
population with at least adequate access to disposal facilities that can effectively prevent human, animal, and insect contact with excreta. **Secondary school enrollment** represents a gross enrollment ratio, which calculates the ratio of total enrollment, regardless of age, to the population of the age group that officially corresponds to secondary level education.

The proliferation of urban slums is potentially relevant to issues of infectious disease in less-developed nations. **Percent of the urban population living in urban slums** is a relatively new measure available from the UN-HABITAT for the year 2005.32 An urban household is classified as a slum dwelling if it lacks one or more of the following four components: (1) sufficient living per person, where there must be no more than 3 people per habitable room; (2) durability of construction, where it must be built in a non-hazardous location, and exhibit structural qualities adequate to protect inhabitants from the outdoors; (3) access to improved water, which includes piped connection to house, public pipe for no more than 5 households, or protected well, spring, or rainwater collection; and (4) or access to improved sanitation, which consists of direct

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32 In this model, I choose to look specifically as urban slums as these settings have been a common site of current TB and malaria studies, given that dimensions of crowding, poor-quality housing, and dirty water represent key risk factors for these infections given their biological characteristics (e.g. Antunes and Waldman 2001; Donnelly et al. 2005; Pattanayek et al 2006). Although the urban growth variable could have been used, urban slums represent a more appropriate way to capture the type of urbanization that would be most associated with promoting TB and malaria rates. For HIV, the key element is the mixing of people and change in sex behaviors (e.g. number of sexual contacts) that comes with movement to an urban area more generally, making urban growth more relevant for the preceding analysis.
connection to public sewer, direct connection to a septic tank, pour flush latrine, or a ventilated pit latrine (UN-HABITAT 2010).

Environmental change is a key cause of rural-to-urban migration, and likely also contributes to urban slum growth. Percent affected by drought measures the percent of the population that have required immediate assistance (i.e. requiring basic survival needs such as food, water, shelter, sanitation and immediate medical assistance), been injured, and/or left homeless due to drought from the years 2000-2005. To create this measure, I weighted data on the total number of people affected by drought from the years 2000-2005 by the total population level. The drought data are taken from the EM-DAT International Disaster Database (2011) and were measured across the years 2000-2005 in order to avoid idiosyncrasies that result from extreme droughts for a certain year.

World-system position and level of economic development are key indicators that could have independent effects on disease prevalence, given postulated trickle-down effects of national economic growth to individuals/households (e.g. Friedman 2005; Princhett and Summers 1996; Firebaugh and Beck 1994). However, HIV analyses in the prior chapter suggested that GDP only operates indirectly though social-health capabilities in reducing disease. I measure world-system position using an index developed by Kick and colleagues (2011), based on prior assessment by Snyder and Kick (1979). The world-system position index is based on a multiple-network analysis of four nation-to-nation linkages, specifically: military and armament transfers, co-membership in non-governmental organizations, trade, and sponsoring an embassy for the years 1995-1999. This measure therefore captures many of the forms of economic, military, and
political dependency that world-systems scholars argue leads to persistently low levels of physical well-being in less-developed nations.

I measure level of economic development using GDP per capita, which is the total annual output of a country’s economy, in current international dollars, per person, for the year 2000 (World Bank 2010). GDP per capita is the total market value of all final goods and services produced in a country in a given year, equal to total consumer, investment, and government spending, divided by the mid-year population. It is converted into current international dollars using Purchasing Power Parity (PPP) rates, which provides a standard measure allowing for comparisons of real price levels between countries.

The literature on development and urbanization in poor societies identifies that dependency may be a key factor depressing levels of economic growth, facilitating unhealthy forms of urbanization, and perhaps also producing direct effects on disease rates. The acceptance of foreign debt also often requires the adoption of conditionality policies that reduce or privatize social services that are relevant to human health (e.g. Stiglitz 2007; McMichael 2004). I thus also include percent GDP from agriculture and level of foreign debt as indicators of dependency. These measures were obtained from the World Bank (2011) and measured for the year 2000. Percent GDP from agriculture measures the value added to the economy due to activities related to forestry, farming, hunting, and fishing. Total debt measures the total external debt as a percent of gross national income. This measure was log-transformed to reduce the influence of extreme outliers.
4.6 Results

Table 4.2 provides the means, standard deviations, and bivariate correlations for all variables included in the analysis. It appears that the magnitude of the bivariate relationships among the variables once again demonstrates a strong potential that multicollinearity could bias the analyses, especially among the development and health capability indicators. This confirms the use of SEM given its ability to address these issues through the creation of latent constructs and direct and indirect pathways.

Figure 4.1 is a graphical representation of the model analyzed. The model is derived from the theoretically specified relationships outlined above and the model building and trimming techniques as discussed by Byrne (2009). Before examining the relationships displayed in the model, it is essential that the model fit statistics are deemed to be in an acceptable range. Inspection of the model presented in Figure 4.1 reveals that the chi-square (44.675, df = 41, not significant), RMSEA (0.026), IFI (.996), TLI (.994), and CFI (.996) fit statistics demonstrate a model with excellent fit to the data. Attaining satisfactory model fit statistics permit interpretation of the pathway coefficients, all of which are highly statistically significant at the .001 level.

The results presented in figure 4.1 reveal that the cross-national determinants of TB and malaria are very similar, given the extremely high factor loadings for TB and malaria (.80 and .89, respectively), even when taking into account the effects of the other variables included in the analysis.

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33 Following the advice offered by Byrne (2009), each plausible causal relationship was examined in the model. All non-significant paths are deleted simply because they show no statistical relationship to the dependent variable of interest. Among a few indicators, coefficients were moderately large, but inclusion of the predictor suggested poor model fit. Thus, the model presented here is comprehensive in that it includes all possibly significant paths, as well as parsimonious, as any non-relevant paths or factors are appropriately trimmed from the model.
In looking at the structural determinants of disease, social-health resources, measured by schooling, sanitation, physicians, and fertility, have an extremely robust, negative influence on malaria and TB rates (-.97). In fact, social-health capabilities represent the only set of predictors that have a direct relationship to TB and malaria prevalence; all other predictors of disease in the model work indirectly through social-health capabilities. This includes GDP per capita, which has indirect linkages to disease through fostering social-health capabilities (.40). In addition, urban slums have a powerful, indirect relationship to TB and malaria through reducing social-health capabilities (-.59).

Similar to insights gleaned in the prior chapter, urban slum proliferation is driven by drought, or those affected by this form of environmental change (.24). Additionally, GDP per capita reduces the percent of the population affected by drought (-.35). In combination with environmental factors, urban slum growth is furthered by states of dependency, including economic specialization in agriculture and level of foreign debt (.63). Dependency also greatly reduces level of economic growth (-.74), and world-system position greatly lowers levels of dependency (-.46), thus having notable indirect effects on GDP per capita and the other components in the model. In other words, nations that occupy more dependent positions in the world-system have lower levels of economic development, and therefore reduced social-health capabilities and increased disease.

Overall, the results presented in Figure 4.1 reveal that social-health capabilities are paramount in reducing the rates of malaria and TB infection in poor nations. Urban slums are a major impediment to accessing health resources, and this form of urban development is driven
by environmental changes and characteristics of dependency. Indeed, world-systems position, specialization in agriculture, and foreign debt represent important factors that set the stage for low levels of economic development, increased environmental vulnerabilities, urban slum development, and decreased social-health capabilities. It is also important to emphasize that GDP per capita once again operates indirectly, only reducing TB and malaria rates through its effects on social-health capabilities.

5.7 Conclusions

Despite the fact that TB and malaria fundamentally represent two very different infections, the analysis reveals that these diseases are extraordinarily similar in terms of their underlying structural causes. These diseases truly represent afflictions of the poor, as basic indicators of schooling, sanitation, doctors, and fertility predicted prevalence rates at a near-perfect or deterministic relationship (-.97). This finding explains that very straightforward improvements in health infrastructure could solve the TB and malaria disease burden. Frankly, the longer that we go without addressing these issues in poor nations, the greater the possibility that these diseases will not be treatable or curable with these comparatively simple interventions.

Indeed, although addressing these components of social-health capabilities does seem very simple or straightforward, there is overwhelming disagreement on how to do this most effectively. Neoliberal approaches to modernization contend that we should put (just about all of) our faith in economic growth, but once again, the results imply a slightly different story. Similar to the HIV models, GDP per capita has no direct bearing on TB and malaria rates. So
while economic growth is associated with improved health, this is a relationship that must be fostered carefully among states through provisions in health-promoting infrastructure and interventions.

Urban slums also represent a stronger predictor of social-health capabilities, and it is important to note that level of economic development does not protect against the proliferation of urban slums in less-developed nations. Thus, issues of dependency (economic specialization in agriculture and foreign debt) become much more relevant in explaining this very harmful form of urbanization. In addition to dependency, environmental conditions also predict urban slums. In some ways this finding is especially frightening as the literature on environmental migration describes the movement to urban areas as coping mechanism, often the last resort for poor rural households that can no longer support their livelihoods off their land. In other words, this variable captures a destitute group of people, more or less coerced into leaving their homelands, only to move into an urban slum environment.

Somewhat surprisingly, urban slums have no direct relationship to disease prevalence. As this indicator largely takes into account issues of crowding, durability of housing, and access to clean water, it would be expected that these factors have independent effects on TB and malaria. However, the absence of a relationship may be telling in-of-itself, suggesting that issues of crowding and infrastructure in urban settings which serve as major vulnerabilities to disease could be alleviated simply by also providing doctors, schools, etc. in these areas. This therefore represents a hopeful sign. Although many governments have attempted to deal with the slum issue through eviction efforts, for example as in the favelas of Rio de Janeiro, Brazil, perhaps we
should direct resources to simply providing basic services within these locations to improve the conditions in these settings.

The results demonstrate that while there is some consistency with ideas from modernization theory with regards to the relationships among world-system position, GDP per capita, social-health capabilities, and disease, where more integrated nations will have higher levels of economic development and therefore improved health and health resources, there also are some notable discrepancies. Specifically, GDP per capita has not direct links to disease – calling into question the assertion that the relationship between economic development and improvements in health are inherent or natural. Additionally, specialization in agriculture and some forms of investment dependence do not seem to encourage successful development (across economic or social realms), and in fact, the results show that it does just the opposite. These ideas are most consistent with world-systems and dependency themes, which question the assumed relationship between economic growth and human well-being, as well as clearly illustrate how international patterns in production and finance relegate less-developed nations. The world-systems/dependency school is also articulate in explaining how these methods of economic integration create issues of over-urbanization, which is captured here in its most extreme form – urban slums.

The prior HIV analyses demonstrated that environmental degradation associated with climate change represents a premier cause of urban growth. This chapter reaches a similar conclusion in the context of urban slums, argued to be the most harmful type of urban growth taking place in poor nations today (e.g. Davis 2006). Not only does this once again signify the
importance of considering the role of the natural and physical environment in contributing to disease dynamics, but also points to issues of degradation that may be especially relevant for certain diseases, such as malaria, where certain ecological conditions are essential to the disease vector. Indeed, it is likely that environmental changes could be a major factor contributing to malaria prevalence more directly, if these changes lead to the proliferation of mosquito habitats. The next chapter looks at these dynamics more closely, exposing the links between poverty, environmental degradation, and disease most obviously.
Table 4.1: Nations Included in the TB/Malaria Analyses

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Table 4.2: Univariate Statistics and Correlation Matrix for TB/Malaria Prevalence

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<td>1. Malaria Prevalence (ln)</td>
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<td>2. TB Prevalence (ln)</td>
<td>0.778</td>
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<td>3. Percent Access to Sanitation</td>
<td>-0.785</td>
<td>-0.683</td>
<td>1.00</td>
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<td>4. Fertility Rate</td>
<td>0.775</td>
<td>0.543</td>
<td>-0.678</td>
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<td>5. Physician Rate (ln)</td>
<td>-0.773</td>
<td>-0.781</td>
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<td>6. Secondary Schooling</td>
<td>-0.777</td>
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<td>0.774</td>
<td>-0.824</td>
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<td>7. % Urban Slums</td>
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<td>0.637</td>
<td>-0.736</td>
<td>0.707</td>
<td>-0.601</td>
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<td>8. % Afft. Drought (ln)</td>
<td>0.530</td>
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<td>-0.323</td>
<td>0.386</td>
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<td>9. GDP per capita</td>
<td>-0.722</td>
<td>-0.645</td>
<td>0.717</td>
<td>-0.694</td>
<td>0.807</td>
<td>0.804</td>
<td>-0.644</td>
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<td>10. Debt % of GNI (ln)</td>
<td>0.494</td>
<td>0.299</td>
<td>-0.339</td>
<td>0.537</td>
<td>-0.482</td>
<td>-0.541</td>
<td>0.474</td>
<td>0.277</td>
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<td>11. Ag. % of GDP</td>
<td>0.626</td>
<td>0.555</td>
<td>-0.689</td>
<td>0.612</td>
<td>-0.651</td>
<td>-0.734</td>
<td>0.762</td>
<td>0.348</td>
<td>-0.711</td>
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<td>12. World System Position</td>
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<td>-0.311</td>
<td>0.549</td>
<td>-0.695</td>
<td>0.402</td>
<td>0.651</td>
<td>-0.614</td>
<td>-0.224</td>
<td>0.497</td>
<td>-0.532</td>
<td>-0.576</td>
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<td>Mean</td>
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<td>4.97</td>
<td>60.83</td>
<td>3.47</td>
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<td>Standard Deviation</td>
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<td>1.22</td>
<td>29.78</td>
<td>1.61</td>
<td>1.29</td>
<td>27.69</td>
<td>24.13</td>
<td>0.697</td>
<td>33.33</td>
<td>0.751</td>
<td>14.75</td>
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Figure 4.1: SEM Predicting Tuberculosis and Malaria Prevalence
CHAPTER 5

EXPORT AGRICULTURE IS FEEDING MALARIA RATES IN POOR NATIONS

5.1 Introduction

Malaria, a preventable and treatable disease, continues to afflict more than 1 billion people in 102 countries, resulting in around 2 million deaths annually (WHO 2011a). Although rates of malaria declined globally throughout the majority of the 20th century, incidence of and mortality from malaria has increased in many less-developed nations since around the 1990s (WHO 2011a; Breman et al. 2007; Breman 2009). As described in the previous chapters, several studies in the field of epidemiology and demography emphasize that malaria rates are on the rise due to the advent of anti-biotic resistant strains of the disease (e.g. Bates et al. 2004; Breman 2009; Pattanayak et al. 2006). However, there is also a second explanation that comes from a different branch of science, the environmental sciences, which emphasizes the role of human alternations to the natural environment in fueling malaria rates. Although these explanations draw on different proximate causal mechanisms, issues of environmental degradation and depressed levels of health resources in poor nations both stem from larger global inequalities.

Malaria is a parasitic disease transmitted through the bite of a mosquito, and many forms of environmental degradation can spur the proliferation of mosquito habitats. In particular, deforestation associated with agriculture in regions of the Amazon is linked to increasing mosquito populations in case-study research (e.g. Norris 2004; Pimentel et al. 2007). Furthermore, biodiversity loss, especially in bird species which eat mosquitos, is likely to be a
significant precursor to heightened malaria transmission. Like deforestation, biodiversity loss also has clear connections to export dependence on agriculture and other primary sector products (e.g. Shandra et al. 2009). Although associations between environmental degradation and human health have been well examined in the environmental justice literature (e.g. Wilson et al. 2002, Schell and Denham 2003), relatively little attention has been devoted to the role of the environment in promoting infectious diseases (Liu et al. 2007).

As environmental externalities from agriculture and inequalities in social-health capabilities are tied to structural inequalities across nations, it is appropriate to explore the causes of malaria from a comparative, political-economy perspective. While much of the current literature on malaria is restricted to case-studies and sub-regional assessments of the behaviors or individual-level factors that predispose people to the disease, I seek to draw attention to the developmental, infrastructural, and environmental factors that underlie increased malaria prevalence. Prior chapters have already demonstrated how neoliberal approaches to modernization and world-systems/dependency perspectives provide keen insights into many factors that cause infectious diseases remain concentrated in poor nations, including level of economic development, availability of doctors and other social-health capabilities, and urban slums. In addition to this, these bodies of literature explain how the organization of the world economy promotes agricultural export enterprises and heightened levels of environmental degradation in less-developed regions relative to affluent nations (e.g. Chase-Dunn 1998; McMichael 2004; Rice 2007). Thus, the socio-economic development characteristics also
influence patterns in ecological degradation across nations, leading to a more comprehensive assessment of the causes of malaria in less-developed regions.

5.2 Malaria Resurgence and Environmental Degradation

Malaria is a mosquito-borne infectious disease caused by eukaryotic protists of the genus Plasmodium. The disease results from the multiplication of Plasmodium parasites within the red blood cells, causing symptoms that typically include fever and headache, and can progress in severe cases to coma or death. Today approximately 3 billion people who live in tropical and sub-tropical zones are at risk of acquiring malaria (WHO 2011b). The disease was once more widespread, but it was successfully eradicated from many countries with temperate climates during the mid-20th century. Thus, given the success of treatments and interventions among affluent nations, its continued impact on people in less-developed societies is often neglected or forgotten (Packard 2009). However global rates of malaria prevalence are now increasing, due to rising incidence among some nations, particularly those in Sub-Saharan Africa and Southeast Asia (WHO 2011a).

Malaria is highly associated with poverty; and poverty is concentrated in the tropical and subtropical zones, the same geographical boundaries that most closely frame malaria transmission (Sachs and Malaney 2002; Pattanayak et al. 2006). Demographic assessments reveal that about 60% of malaria cases occur in the poorest 20% of the world’s population (Breman et al. 2004). Malaria-endemic countries are not only poorer than non-malarious countries, but they also have significantly lower rates of economic growth (Acemoglu and Johnson 2007; Packard
There are multiple channels by which malaria impedes economic development, including effects on fertility, population growth, savings and investments, worker productivity, absenteeism, premature mortality, and medical costs (Sachs and Malaney 2002). At the household level, poor rural families have higher rates of malaria mortality as they are much less likely to be able to seek effective treatment and also tend to lack adequate sanitation (Pattanayak et al. 2006). Additionally, the advent of malaria infection further impoverishes households; a recent study conducted in rural Uganda finds that households with a malaria-infected member spent about 35% of their average monthly income on malaria treatments (Williams et al. 2009). This study also finds that in families where more than one member falls ill, a third of households spend nearly all of their monthly income on malaria treatments (Williams et al. 2009). Clearly, gains in human capital and economic growth are compromised for nations that have high rates of malaria prevalence, which is only going to reproduce conditions of underdevelopment (Packard 2009; Acemoglu and Johnson 2007).

While the most common preventative measure for malaria includes the use of insecticide-treated mosquito bed nets, malaria parasites are developing dangerous levels of resistance to many insecticides (Breman et al. 2007; Pattanayak et al. 2006; Sachs and Malaney 2002; Williams et al. 2009). As was discussed in prior chapters, antibiotic resistance is also becoming a pervasive factor promoting prevalence in poor nations (Breman et al. 2007; Breman 2009; Pattanayak et al. 2006; Bates et al. 2004). In general, the malaria virus has already developed strong resistance to the cheapest interventions available, so successful treatment requires more expensive and intensive solutions.
Although the last century witnessed many successful programs to eradicate the parasite among more-developed nations with favorable ecological conditions, the world is now facing a rapidly increasing malaria disease burden (Sachs and Malaney 2002; Bates et al. 2004; Pattanayak et al. 2006; Breman 2009). It is likely that malaria prevalence and mortality have increased not only due to increasing anti-biotic resistance, but also from human-induced changes to the natural environment. A burgeoning area of research finds that increased levels of environmental degradation are contributing to upsurges in malaria prevalence in certain areas (e.g. Norris 2004; Pattanayak et al. 2006; Vittor et al. 2009, 2006; Bates et al. 2004). In fact, some studies attribute 70-90% of the risk of malaria to environmental factors alone (e.g. Smith et al. 1999).

There is a wide body of research which recognizes that mosquitoes fundamentally depend upon water and warm temperatures for their breeding and life-cycle, explaining why malaria is largely a rural, tropical disease (e.g. Norris 2004; Vittor et al. 2006, 2009; Pattanayak et al. 2006; Pimentel et al. 2007). Human alteration of the environment often exacerbates existing mosquito-associated problems by expanding habitats, creating new habitats, or altering habitats in such a way that limited mosquito populations may explode (Norris 2004; Pattanayak et al. 2006; Pimentel et al. 2007). Habits may include stagnant pools, slow-moving streams, geothermal pools, tree holes, and water pooling in artificial containers or shallow depressions (Norris 2004). Many human alterations to the environment serve to increase potential mosquito habitats by increasing the temperature of water bodies, creating new mosquito habitats by introducing water into regions where naturally occurring surface water might be rare or limited,
such as by establishing reservoirs, irrigating fields, or creating open waste and water systems, or by reducing natural mosquito predators, such as birds (Norris 2004).

One of the most common sources of degradation that is linked to the expansion of mosquito populations is export agriculture (Norris 2004; Pattanayak et al. 2006; Vittor et al. 2006, 2009). Establishing large areas for crops and plantations leads to increased rates of deforestation, and the connections between deforestation and mosquito proliferation are becoming more evident in epidemiological case-studies (e.g. Pattanayak et al. 2006; Norris 2004; Vittor et al. 2006, 2009; Pimentel et al. 2007). Deforestation can be linked to malaria transmission in several ways. Deforestation increases the available mosquito habitats by exposing shallow pools and streams of water to sunlight. Additionally, leaving rotten stumps behind creates “tree holes” which are prime mosquito habitats as rain water fills the stump like a bowl, creating a warm, protected reservoir. Deforestation can also affect local and regional climates as the felling of trees is associated with increasing temperatures and reducing rainfall in some areas. Generally, deforestation is often the first step in a chain of land-use changes that propagate mosquito populations, including irrigation and road construction (Norris 2004; Pattanayak et al. 2006).

These propositions are well-supported in the case-study literature. For example, Vittor and colleagues (2006, 2009) have conducted several analyses in regions of the Peruvian and Brazilian Amazon, where they find much higher levels of mosquito larvae at sites with significant loss of forest cover. In fact, deforestation sites had a biting rate that was more than 278 times the rate determined for areas that were predominately forested (Vittor et al. 2006). Similarly,
Yasuoka and Levins (2007) conduct a content analysis of case-studies centered on changes in ecology and malaria incidence in sub-regions of Thailand, Nepal, India, China, Guyana, Uganda, and other topical areas. They find 60 examples which demonstrate that deforestation related to agricultural development is linked to an increase in mosquito populations. In particular, agricultural development is especially favorable for sun-loving mosquito species, allowing them to increase in or invade deforested areas where water bodies become exposed to sunlight (Yasuoka and Levins 2007).

In addition to deforestation, the expansion of agricultural enterprises has also been linked to increasing biodiversity loss (e.g. Shandra et al. 2009), and epidemiologists are beginning to take note that changes in species diversity and richness can be a significant precursor to infectious disease. A relevant example comes from a study of hemorrhagic fever in South America, which is transmitted by rodent populations (Mills 2006). Researchers are finding that biodiversity loss greatly increases the efficiency of infectious disease transmission due to declining predator populations that lead to increases in the number of host species (e.g. Mills 2006). These ideas have also been applied to the malaria virus, but only in limited contexts. Many descriptive and policy-orientated articles make deductive connections between biodiversity loss and malaria transmission (e.g. Daszek and Cunningham 2003; Chapin et al. 2000), although this has not yet been empirically tested. These studies point to biodiversity loss in bird species in particular, as birds represent the species-group that faces some of the highest rates of threat and extinction, as well as the implicit connection of bird population decline to mosquito proliferation as insects represent a key source of food for most avian species.
Given the clear ecological underpinnings of the malaria virus, this disease has often been considered a plague primarily for rural populations, as rural people inherently live closer to mosquito vectors. Although the prior chapter provided insight into the encroachment of malaria in urban areas, it is important to recognize that malaria is still more widespread in rural areas, and issues of rural sanitation and clean water access are paramount in explaining current prevalence patterns, as open pits and streams represent prime mosquito habitats (e.g. Pattanayek et al 2006; Norris 2004).

Issues of environmental degradation associated with agriculture, access to improved sanitation, and the availability or effectiveness of other forms of health infrastructure, such as doctors, are deeply tied to global development dynamics and the structure of the world-economy. Although much of the relationship between factors related to development, including health infrastructure, have already been explored in prior chapters, the theoretical implications for the environment deserve further attention. I thus now turn to a discussion of modernization and world-systems/dependency perspectives to the environment, in order to more appropriately assess the direct and indirect linkages among development dynamics, environmental degradation, and malaria prevalence.

5.3 Development, Environmental Degradation, and Human Well-Being

Neoliberal approaches to modernization and world-systems/dependency scholars not only differ in their theorizations on societal development, but also in their ideas regarding society and environment relationships. Recall that a central theme of modernization is the
incorporation into a complex division of labor; at the international scale, it is promoted that nations encourage development by producing according to comparative advantages (Ricardo 1817; Friedman 2005). As many poor nations are located in tropical areas, it is often argued by agencies, such as the World Bank, that these nations specialize in the production and export of agricultural commodities and raw materials (see for example a report published by the World Bank in 2008 titled, Agriculture for Development). As these nations have been heavily dependent upon primary sector exports since colonization in the 15th century, continuing on this developmental route is almost compulsory given path dependencies in infrastructure, organization, and labor (Bunker and Ciccan tell 2006), despite that this form of economic integration and specialization has historically never led to consistent developmental gains.

Indeed, while the modernization school sees the international division of labor as being economically efficient and beneficial to all participation nations, world-systems/dependency scholars point out that the international division of labor does not benefit all nations equally, and that this type of structure allows the gains of core or affluent nations to outpace all others. The harmful effects of primary sector export dependency in less-developed nations are specifically articulated by dependency/world-systems theorizations on unequal exchange. The phrase, “unequal exchange” was coined by Arghiri Emmanuel in the 1970s, to highlight the stark inequalities that characterize international trade. The exchange of relatively higher-value core products for less-valuable peripheral commodities involves transfers in surplus value up the world-system from less-developed nations to more-developed nations (Emmanuel 1972; Hornborg 2001; Wallerstein 1974, 2004). Unequal exchange is an inherent component in the
capitalist world-economy as this system is predicated on the endless accumulation of capital; unfair exchanges that systematically undervalue periphery products relative to core products enhance the profitability and concentration of capital in affluent nations, reproducing international inequality (Emmanuel 1972; Hornborg 2001; Wallerstein 1974; 2004).

Theorization on unequal exchange also has particular relevance for ecological conditions and environmental outcomes. Referred to as “unequal ecological exchange,” this concept explains that the global organization of production concentrates environmental degradation in less-developed nations, as primary sector production causes many environmental problems, including deforestation and biodiversity loss (e.g. Shandra et al. 2009; Jorgenson 2010; Jorgenson et al. 2010; Rice 2007, 2009; Austin 2010a, 2010b). Thus, the international division of labor allows powerful nations to gain disproportionate access to natural resources and environmental space in less-developed nations, which is used to further their own interests and maintain advantaged positions in the world-system (Bunker 1985; Rice 2007, 2009; Hornborg 2001). Bunker’s (1985) work in the Amazon represents one of the foundations of this line of research, as he argued that unequal exchange includes a process of energy and material loss in less-developed nations, given their focus on extractive and agricultural exports. He concluded that this structural feature of the world-system increases international inequality as materials extracted in the periphery are then sent to productive economies of more-developed nations for use in highly-profitable core production processes (Bunker 1985, Rice 2009). Thus while consumption largely takes place in affluent nations, the environmental externalities are concentrated in poor regions (e.g. Hornborg 2001; Jorgenson et al. 2010).
In short, neoliberal approaches to modernization have an overwhelming focus on achieving economic growth, thus treatment of the natural environment is a secondary concern. Perhaps environmental sustainability in the pursuit of comparative advantage and economic integration is largely ignored as it is assumed that nations will become more ecologically efficient as they modernize (e.g. Mol 2001). Although there is much counter-evidence to this line of thinking when the overall costs of consumption and waste are taken into account (e.g. Jorgenson and Clark 2011; Jorgenson et al. 2010), theorizations by Bunker (1985) within the world-systems school also emphasize that the natural environment may be a nation’s most vital resource base. Thus, sustainable production and management of natural resources are much more likely to translate into long-term developmental success, even if there are short-term economic drawbacks (Bunker 1985).

The unequal ecological exchange arguments about how the structure of primary sector production in the world-system causes an unfair amount of environmental degradation in poor regions has direct links to the epidemiological case-studies on environmental change and mosquito proliferation discussed previously, where primary sector export flows could be seen as an indirect cause of increasing malaria prevalence. Additionally, the wide array of comparative research on life expectancy and infant mortality has associated agriculture and raw material export dependence with lower levels of physical well-being attainment in less-developed nations (e.g. Wimberley and Bello 1992; Ragin and Bradshaw 1992; Shandra et al. 2004); this therefore represents a specific context in which agricultural exports impair physical well-being. I now turn to specifying the key hypotheses.
5.4 Hypotheses

In addition to the importance of access to health-promoting capabilities emphasized in the prior chapter, there is an alternative explanation regarding heightened malaria rates in less-developed nations. It highlights the role of human alterations to the environment which are likely to promote mosquito habitats. I thus articulate the following hypothesis: (H1) I predict that environmental degradation, in the forms of deforestation and biodiversity loss in bird species, associated with export dependence on agriculture is fueling malaria prevalence in less-developed nations (net of the influence of other relevant predictors). In other words, primary sector export flows will have an indirect impact in malaria rates in less-developed nations, through their direct influence on deforestation and biodiversity loss.

I also predict (H2) that social-health capabilities, captured specifically with measures of rural access to improved sanitation, availability of physicians, fertility, and participation in secondary schooling, greatly reduce malaria rates in less-developed nations (as was demonstrated in the previous chapter). In addition, I postulate that a number of pertinent developmental and ecological factors influence the proposed relationships, including latitude, world-system position, and level of economic development. Of particular relevance, (H3) I postulate that GDP per capita has an indirect influence on malaria rates through social-health capabilities, as was evidenced in the prior chapter.
5.5 Methods

Sample. The sample is restricted to 102 malaria-endemic nations. Malaria endemic nations can be defined as nations in which there is a constant measurable incidence both of cases of malaria and of natural transmission in an area (WHO Statistical Information System 2011). Countries that only have imported cases or introduced cases resulting from imported cases are not included in the analyses. All malaria endemic nations have GDP per capita estimates that are in the lower three quartiles of the income classification of countries (World Bank 2010), thus broadly representing a sample of less-developed nations. For a complete list of nations included in the sample, see Table 5.1.

Analytic Strategy. I utilize structural equation modeling (SEM) to assess the cross-national predictors of malaria prevalence. As described in the prior chapters, SEM is a powerful extension of multiple regression, and is especially appropriate for this analysis given the construction of latent variables, such as the concept of “social-health capabilities.” In addition, the key hypothesis concerns the specification of direct and indirect effects. When these features are combined with those discussed earlier, it is clear that SEM is the most appropriate methodological approach for this line of research.

34 A sub-sample of Sub-Saharan African nations was also tested; the results were consistent across the two samples, indicating that there are not unique effects for Sub-Saharan Africa that need to be taken into account.
35 As this study is concerned with the factors that cause malaria, it is appropriate to exclude non-malarious nations from the sample.
36 Although there were some missing data points, there appeared to be no pattern to the missing values that would bias results. Utilizing the strengths of the analytic technique, I treat missing values by using full information maximum likelihood, where all available data is used to generate maximum likelihood based statistics. I also conducted the analyses using listwise deletion strategies, and achieved the same substantive conclusions; however the sample size was greatly reduced, and thus I prefer to report the model utilizing data from as many cases as possible here.
**Dependent Variable.** The key dependent variable is malaria prevalence for the year 2006. Data on the number of malaria cases per country were taken from the World Health Organization Statistical Information System (2011). This measure was transformed into a rate to capture the number of people infected per 100,000. This was done using total population data taken from the World Bank (2011) to control for the absolute population level of the reporting nation. This measure was log-transformed to reduce the influence of extreme outliers.37

**Independent Variables.** As the malaria parasite requires particular climate conditions, latitude represents an important predictor. Data on latitude were taken from the CIA World Fact Book (2010), which provides average latitude for each nation in the world. The latitude scores were transformed into absolute values to capture distance from the equator. Thus, it is expected that a negative relationship be evidenced between latitude and malaria, as malaria rates are likely to decrease with distance from tropical zones.

World-system position and level of economic development are key indicators that are likely to have independent effects on malaria prevalence, given postulated trickle-down effects of national economic growth to individuals/households (e.g. Friedman 2005; Princhett and Summers 1996; Firebaugh and Beck 1994). In addition, world-system position and level of economic development are also linked to various dimensions of social-health capabilities and environmental degradation (e.g. Soares 2007; Burns et al. 2003). I measure world-system position using an index developed by Kick and colleagues (2011), based on prior assessment by

37 Although negative binomial regression can be used when the dependent variable is highly skewed, this form of regression is not available through SEM. To ensure that skewness is not biasing the results, I tested models with the natural log of malaria rates and with raw malaria rates. Both models produced consistent covariate effects; however the model with the natural log of malaria rates achieved better model fit statistics, and thus is the model presented here.
Snyder and Kick (1979). The world-system position index is based on a multiple-network analysis of four nation-to-nation linkages, specifically: military and armament transfers, co-membership in non-governmental organizations, trade, and sponsoring an embassy for the years 1995-1999. This measure therefore captures many of the forms of economic, military, and political dependency that world-systems scholars argue leads to persistently low levels of physical well-being in less-developed nations.

I measure level of economic development using GDP per capita, which is the total annual output of a country’s economy, in current international dollars, per person, for the year 2000 (World Bank 2010). GDP per capita is the total market value of all final goods and services produced in a country in a given year, equal to total consumer, investment, and government spending, divided by the mid-year population. It is converted into current international dollars using Purchasing Power Parity (PPP) rates, which provides a standard measure allowing for comparisons of real price levels between countries.

Social-health capabilities are important non-economic factors that are likely to greatly influence malaria rates in less-developed nations. I measure social-health capabilities using a latent construct that is comprised of the following indicators: number of physicians, the fertility rate, rural population access to improved sanitation, and secondary schooling for the year 2005. Data were acquired from the World Bank (2011). Number of physicians measures the number of physicians in a nation per 100,000 people. This measure was log-transformed to address issues of skewness. The fertility rate is an estimate of the number of children an average woman would have if current age-specific fertility rates remained constant during her reproductive years.
Rural access to improved sanitation measures the percentage of the rural population with at least adequate access to disposal facilities that can effectively prevent human, animal, and insect contact with excreta.\footnote{In this chapter, I prefer to include rural access to sanitation as this analysis largely concerns rural factors associated with malaria prevalence (I also tested this model with total access to sanitation, and the substantive results were consistent). It is clear however based on the results of the prior chapter, that sanitation issues in urban areas can have notable influences on malaria rates as well.} Secondary school enrollment represents a gross enrollment ratio, which calculates the ratio of total enrollment, regardless of age, to the population of the age group that officially corresponds to secondary level education.

In addition to social-health capabilities, a central proposition is that environmental degradation, namely deforestation and biodiversity loss, associated with agriculture and raw material production is fueling malaria prevalence. I thus employ a measure of primary sector export flows to capture export dependence on primary sector productions. This indicator was originally developed by Jorgenson (2006), and it quantifies the relative extent to which primary sector exports are sent to more-developed countries from a less-developed exporting country for the year 2000. Export data were acquired from the UN Commodity Trade Database (2005). To create this measure, export values for prominent primary sector products are first converted from dollar amounts to proportional scores for each sending nation.\footnote{These categories include pulp and paper, wood, lumber and cork, live animals, meat and meat preparations, dairy products and eggs, cereals and cereal preparations, fruit and vegetables, sugar, sugar preparations, and honey, coffee, tea, cocoa, and spices, feed stuff for animals excluding unmilled cereals, miscellaneous food preparations, unmanufactured tobacco, and crude animal and vegetable materials.} This score reflects the total proportion of primary sector products that are sent to a specific receiving country from a sending country. Each proportion score is then multiplied by the receiving nation’s GDP per capita (which is taken from the World Bank and measured in constant 2000 U.S. dollars). These
products are then summed, and this final measure quantifies a nation’s relative level of primary sector exports that are sent to more-developed nations.

I employ measures of deforestation and biodiversity loss as prominent forms of environmental degradation that are likely to be directly associated with increasing malaria prevalence. The natural deforestation rate is measured as an annual percent change score, calculated using FAO estimates of natural forest area, from 2000-2005 (FAO 2008). These data were collected for the Global Forest Resource Assessment (GFRA) and represent point estimates for natural forest stock measured in thousand square hectares for 2000 and 2005. The natural forest area measure includes land area that is more than 0.5 hectares which contains trees higher than 5 meters and a canopy cover of more than 10 percent. The forest cover change score is multiplied by -1 to appropriately capture rate of forest loss.

Biodiversity loss is measured as the number of threatened bird species for the year 2002. These data are based on the World Conservation Union’s Red List of Threatened Animals, and are available through the World Bank database (2011). The “Red List,” a comprehensive inventory of threatened species, is the most widely used source on endangered animals (e.g. Shandra et al. 2009). It is based on information provided by a network of more than 7000 conservation experts and their associated organizations from almost every nation on the planet. This measure was logged to reduce the influence of extreme outliers.

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40 The natural deforestation rate reflects the annual percent change in natural forest cover, which does not include forest plantations or areas used for forestry and other related purposes, only native vegetation.
41 Although alternative measures, such as number of extinct species, are also available, the number of threatened species best captures declines in bird populations and species diversity that would have the most direct influence on mosquito proliferation.
5.6 Results

Table 5.2 provides the means, standard deviations, and bivariate correlations for all variables included in the analysis. These are provided as a preliminary test of multicollinearity among central predictors. Although it can be difficult to confirm the existence of multicollinearity based solely on correlations among variables, the magnitude of the relationships among the variables demonstrates a high probability that multicollinearity is problematic for the data analyzed, especially among the development and health capability indicators. This further warrants the use of the SEM analytical techniques given its superior handling of correlated independent variables through the creation of latent constructs and direct and indirect pathways that circumvents the tendency to bias coefficient estimates.

Figure 5.1 is a graphical representation of the model analyzed. The model is derived from the theoretically specified relationships outlined above and the model building and trimming techniques as discussed by Byrne (2009). Before examining the relationships displayed in the model, it is essential that the model fit statistics are deemed to be in an acceptable range. Inspection of the model presented in Figure 5.1 reveals that the chi-square (41.606, df = 38, not significant), RMSEA (0.031), IFI (.993), TLI (.987), and CFI (.992) fit statistics demonstrate a model well within the desired ranges for a “good” fit to the data.

42 Following the advice offered by Byrne (2009), each plausible causal relationship was examined in the model. Virtually all non-significant paths are deleted simply because they show no statistical relationship to the dependent variable of interest. Among a few indicators, coefficients were moderately large, but inclusion of the predictor suggested poor model fit. Thus, the model presented here is comprehensive in that it includes all possibly significant paths, as well as parsimonious, as any non-relevant paths or factors are appropriately trimmed from the model.
Attaining satisfactory model fit statistics permit interpretation of the pathway coefficients, all of which are statistically significant at the .05 level.

Results presented in Figure 5.1 depict that world-system position is positively associated with GDP per capita (.45) and health capability (.24). GDP also has a positive influence on health capability in less-developed nations (.51). Of notable importance, world-system position and GDP per capita do not have direct influences on malaria prevalence, but rather only significantly influence malaria rates through their effects on improving health capability. Health capability in turn, has direct negative influences on malaria prevalence (-.46), indicating that access to doctors, the fertility rate, availability of improved sanitation in rural areas, and participation in schooling are extremely important in reducing malaria transmission in less-developed nations.

Latitude also has strong associations with social-health capabilities (.46); the positive score indicates that social-health capabilities improve in nations located further from the equator. This finding implies that social-health capabilities are lowest in the nations that are most likely to have ecological predispositions to infectious diseases. Somewhat surprisingly, latitude has no direct effect on malaria prevalence; this suggests that within the tropical and sub-tropical zones (or malaria-endemic regions) there is little variation in malaria prevalence.\textsuperscript{43} Latitude also has direct linkages to primary sector export flows (-.33) and the deforestation rate (-.33). This suggests that nations that are closer to the equator have increased dependence on

\textsuperscript{43} This is consistent with epidemiological research that shows that the malaria parasite ceases development at temperatures below 61 degrees; nations that are considered to be “malaria endemic” do not have temperatures that consistently fall below this threshold for the majority of the year, thus equalizing possibilities for transmission for nations within tropical and sub-tropical zones.
primary sector exports, and also experience heightened rates of deforestation relative to nations located further away from the equator. In addition, GDP per capita is negatively associated with deforestation (-.26), suggesting that higher levels of economic development reduce forest loss. These relationships were not found for number of threatened in bird species, suggesting that this form of environmental degradation is fairly even across tropical and sub-tropical zones, and that economic development does not necessarily buffer against loss in bird species.44

Figure 5.1 also demonstrates that increasing deforestation and biodiversity loss in birds results from high levels of primary sector export dependence (.22 and .25, respectively). These forms of environmental degradation, in turn, further malaria prevalence rates (.18 and .25, respectively). Thus, primary sector export flows have indirect links to heightened malaria prevalence through their influence on deforestation and biodiversity loss in avian species, which is consistent with the case-study evidence.45

Taken together, the results depict that less-developed nations that are in advantaged world-system positions of power have higher levels of economic development and improved social-health capabilities. Social-health capabilities in the form of schooling, reduced fertility, access to doctors, and availability of improved sanitation in rural areas greatly reduce malaria rates, and, although not able to be directly tested, are likely to reduce the chance of developing an antibiotic resistant strain of the infection. This finding provides robust support for hypothesis 2. Additionally, the results illustrate that the first hypothesis is also well-supported;

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44 This is consistent with prior cross-national work that has linked biodiversity loss in bird species to manufacturing expansion, which is positively associated with economic growth (e.g. McKinney, Kick and Fulkerson 2010).
45 I also tested whether deforestation increases biodiversity loss; this relationship was not evidenced here and this is likely due to limitations of the data/time points, where there is not an appropriate lag between the deforestation rate (2000-2005) and the biodiversity loss indicator (2002).
export dependence on primary sector products has notable indirect effects on malaria prevalence through its positive influence on deforestation and biodiversity loss in bird species. Processes of environmental degradation associated with agriculture and social-health capabilities represent robust predictors of malaria rates in less-developed nations. Thus, while aspects of modernization reduce malaria rates, the overall structure of the world-economy facilitates increased resource degradation in poor nations, which is shown to directly influence malaria rates. This represents an important contribution of this research; although case studies have linked environmental degradation to malaria rates in certain locales, the broader connections of resource use to international development dynamics aptly demonstrates that this epidemiological phenomenon is fundamentally tied to processes of international inequality.

5.7 Conclusion

Malaria standouts out from the other diseases under investigation as this infection has direct links to environmental conditions, given the mosquito as the disease vector. Although most studies of environmental degradation treat deforestation, biodiversity loss, and other ecological measures as outcomes to examine, this analysis demonstrates that environmental decline is an important predictor of disease. In particular, the analyses demonstrate that deforestation and biodiversity loss in bird species associated with primary sector export dependence increases malaria rates in endemic nations. In this way, the results provide robust evidence for political-economy or world-system interpretations of the relationships between international development and environmental degradation. The primary sector export flows
indicator captures the relative amount of agricultural exports that are sent to more-developed nations; thus, consumption levels in affluent areas are prominently linked to furthering the malaria virus in poor nations. Although many studies of unequal exchange dynamics link agricultural production in less-developed nations to environmental outcomes, this study also illuminates the avenues by which inequality in the international organization of production promotes deadly infectious disease.

In addition, the findings confirm that global positions of power promote domestic economic welfare and social-health capabilities. This finding is in line with both bodies of theory, and therefore illuminates that elevated malaria rates are likely to persist among less-developed nations. The results also illustrate that economic development does not necessarily trickle-down to human well-being gains, as there is no direct path from GDP per capita to malaria prevalence. This finding now represents the most consistent across all of the models predicting disease. As previously mentioned, a lack of a direct effect from GDP per capita to disease challenges some of the most central ideas expressed by modernization theorists and neoliberal economic perspectives. This school of thought also falls short in so far that harnessing a “comparative advantage” in primary sector production does not promote successful development given the associations between export dependence, ecological degradation, and disease. However, there is some support for modernization perspectives as economically advanced societies tend to have higher levels of health capability and therefore reduced rates of malaria. But the relationship between development gains and social-health capabilities is a
condition that must be fostered among nations; a laissez-faire approach will likely not fully address this important link.

It is also relevant to point out that while the effects of social-health capabilities on malaria were comparatively more robust than the factors related to environmental degradation, we do see a notable decline in the value of the health capability coefficient from the malaria/TB model presented in Chapter 4, where the coefficient from health capability to disease was -.97, while here it is -.46. This suggests that while social-health capabilities or resources are paramount in explaining malaria prevalence across nations, the environmental factors also matter greatly, as their inclusion in the model reduced the effects of social-health capabilities significantly. From a public policy standpoint, these results demonstrate that anti-malaria efforts should be especially directed towards nations that have high dependence on agriculture, and for populations within these nations that work in or live near agricultural production sites.

Export agriculture, environmental degradation, and low levels of health infrastructure are the product of structural inequalities across countries, which essentially allow some nations to promote public health provisions and become less reliant on agricultural production at the expense of others. As inequalities in the organization of the world-economy have remained fairly constant over time (e.g. Snyder and Kick 1979; Kick et al. 2011), it is likely that the underlying causes of malaria among the least developed of nations will only reproduce conditions of underdevelopment and further transmission of this devastating virus. Malaria is currently a preventable and treatable disease – but this in itself is changing with the emergence

46 Although the models are distinct and therefore direct comparisons are not completely empirically accurate (as identical samples and data were not analyzed), it is still possible to make rough comparisons across the analyses as the samples and measures employed are similar.
of antibiotic resistant strains of the virus. It is essential to address the structural and environmental conditions that promote transmission and antibiotic resistance in less-developed nations, or this could quickly become an epidemic that threatens the livelihood of over 3 billion people. I now turn to a discussion of theory and public policy, as addressing the underlying causes of infectious disease is of paramount concern.
Table 5.1: Nations Included in the Malaria Analyses

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<th>Afghanistan</th>
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Table 5.2: Correlation Matrix and Univariate Statistics for Malaria Prevalence

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<td>3. GDP per capita</td>
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</tr>
<tr>
<td>4. Physicians (ln)</td>
<td>0.507</td>
<td>0.333</td>
<td>0.606</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5. Fertility Rate</td>
<td>-0.456</td>
<td>-0.472</td>
<td>-0.587</td>
<td>-0.821</td>
<td>1.00</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>6. Access to Rural Sanitation</td>
<td>0.444</td>
<td>0.338</td>
<td>0.555</td>
<td>0.679</td>
<td>-0.659</td>
<td>1.00</td>
<td></td>
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<tr>
<td>7. Secondary Schooling</td>
<td>0.424</td>
<td>0.428</td>
<td>0.652</td>
<td>0.852</td>
<td>-0.85</td>
<td>0.655</td>
<td>1.00</td>
<td></td>
<td></td>
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<tr>
<td>8. Primary Export Flows</td>
<td>-0.382</td>
<td>0.231</td>
<td>0.215</td>
<td>-0.12</td>
<td>0.009</td>
<td>-0.061</td>
<td>-0.053</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Deforestation Rate</td>
<td>-0.507</td>
<td>-0.103</td>
<td>-0.291</td>
<td>-0.493</td>
<td>0.529</td>
<td>-0.394</td>
<td>-0.439</td>
<td>0.336</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Threatened Bird Species (ln)</td>
<td>-0.141</td>
<td>-0.027</td>
<td>-0.149</td>
<td>-0.279</td>
<td>0.179</td>
<td>-0.221</td>
<td>-0.274</td>
<td>0.38</td>
<td>0.021</td>
<td>1.00</td>
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</tr>
<tr>
<td>11. Malaria Prev (ln)</td>
<td>-0.332</td>
<td>-0.335</td>
<td>-0.429</td>
<td>-0.639</td>
<td>0.634</td>
<td>-0.363</td>
<td>-0.559</td>
<td>0.314</td>
<td>0.421</td>
<td>0.404</td>
<td>1.00</td>
</tr>
<tr>
<td>Mean</td>
<td>17.37</td>
<td>3.04</td>
<td>31.85</td>
<td>-1.16</td>
<td>4.21</td>
<td>40.79</td>
<td>45.32</td>
<td>15249</td>
<td>5.3</td>
<td>2.59</td>
<td>5.8</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>12.04</td>
<td>1.65</td>
<td>28.94</td>
<td>1.49</td>
<td>1.60</td>
<td>28.06</td>
<td>25.46</td>
<td>7735</td>
<td>6.93</td>
<td>1.13</td>
<td>3.73</td>
</tr>
</tbody>
</table>
Figure 5.1: SEM Predicting Malaria Prevalence
6.1 General Conclusions

At the beginning of this dissertation, I made the broad prediction that the underlying risks or vulnerabilities associated with these diseases would overlap greatly. This hypothesis is ultimately supported as remarkable consistencies are found with regards to the role of environmental degradation, urbanization, economic development, and social-health capabilities in being major contributors to disease dynamics. Thus it is clear that HIV, TB, and malaria are similar in their social, economic, infrastructural, and environmental determinants. While international development agencies continue to treat poverty, environmental degradation, and disease and stand-alone threats to development (e.g. UN 2005), these dimensions overlap, combine, and influence one another to reproduce conditions of underdevelopment in poor nations.

HIV, TB, and malaria are acquired through bio-physical processes where viruses or bacteria upset the functioning of the human body. However, disease represents a significant sociological issue as structural inequalities across nations lead to disparities in economic development, social-health capabilities, and environmental degradation, which in turn have robust influences on disease rates in less-developed nations. Although the current literature on
disease is largely focused on the individual-level and biological factors that promote transmission in certain settings, this dissertation highlights the underlying factors that lead to higher rates of disease in some nations relative to others. Diseases are specifically contracted through coughing, unsafe sex, and insect bites, but a number of structural relationships have preceded and contributed to these precise mechanisms, such as the overall availability of doctors, the level of deforestation, or how safe the drinking water is. Thus in many ways, this analysis brings to light the root causes of health disparities across nations.

Poverty is often singled out as the source of infectious disease in less-developed nations (e.g. Packard 2009; Bates et al. 2004; Farmer 1999, 2000, 2001; MacIntosh and Thomas 2004; Soares 2007), and poverty is most often measured in cross-national analyses using GDP per capita (e.g. MacIntosh and Thomas 2004; Brady et al. 2007). But many consider poverty to be a multi-dimensional concept, as it can include social or infrastructural dimensions as well as economic characteristics (e.g. Sen 1999; Farmer 1999, 2000, 2001; Bates et al. 2004). Key elements of poverty captured in the analyses are GDP per capita, fertility, schooling, availability of doctors, and access to sanitation. Urban slums and environmental degradation also represent other important dimensions of poverty that were shown to indirectly contribute to disease.

Although economic development, or in essence GDP per capita, garners overwhelming attention in cross-national assessments of physical well-being for its robust and direct influence on health outcomes, the modeling strategies employed here illustrate that GDP only operates indirectly to influence disease and that other non-economic factors proved to be much more proximate and robust in explaining infectious disease rates. In particular, social-health
capabilities of access to doctors, participation in schooling, reduced fertility, and (in the
TB/malaria analyses) access to sanitation, were consistent and robust predictors across the
models. It should also be emphasized that the indirect effect of GDP per capita on social-health
capabilities was also notably stable across the disease analyses, which does garner some support
for approaches that emphasize economic growth as a means to improve human welfare. But the
need to understand this as an indirect relationship which largely exists through non-economic,
social resources deserves further emphasis in current research, theorization, and public policy.

Urbanization is also a common theme in each chapter; the results depict that high levels
of urban growth and urban slum development serve to depress social-social-health capabilities,
and urban growth is also directly linked to furthering HIV rates in Sub-Saharan African nations.
Environmental alterations associated with climate change are driving these patterns in
urbanization, thus representing an important underlying cause of infectious disease in less-
developed nations. Environmental changes are also particularly relevant in explaining cross-
national trends in malaria rates, where deforestation and biodiversity loss resulting from export
agriculture exemplifies a significant cause of malaria prevalence in less-developed nations.

The unique findings for HIV in Sub-Saharan Africa deserve further emphasis. While
female empowerment is overall linked to improvements in health, the characteristics of HIV
transmission in Sub-Saharan Africa symbolize an example where this form of social change may
be producing unintended consequences. Improving women’s access to education and lessening
the role of child-rearing, especially in rural settings, combines with deteriorating environmental
conditions to stimulate people to seek alternative opportunities in cities. As women’s social
standing improves, there is increased movement into cities and norms about sexual behavior may also change; many have more freedom in choosing their partners and exchange partners more frequently (e.g. Rushing 1999). Although awareness about HIV/AIDS is increasing, many studies demonstrate that it continues to be major social stigma, including heavy discrimination against people, especially women, who have HIV (e.g. Lee et al. 2005; Paxton et al. 2005). Thus, even if access to contraceptives and other health resources improves, these gains could be cancelled out by more liberal sexual norms that tend to characterize populations in urban areas. As economic development improves female empowerment, we see GDP per capita having a notable positive relationship to HIV prevalence within the region of Sub-Saharan Africa. Thus, achieving GDP per capita gains does not necessarily ensure reductions in disease, pointing to major theoretical implications.

6.2 The Perils and Pitfalls of Neoliberal Development Strategies

Few would argue that development promotes health, but how to define and pursue development is hotly debated. Neoliberal approaches to modernization and world-systems/dependency perspectives have different explanations to disease trends. The former contends that high rates of disease exist in poor nations because they are not yet modernized; they most importantly need economic growth, as well as Western models for education and global economic integration to promote development and reduce disease. The latter argues that high rates of disease in poor nations continue to persist due to capitalist processes of dependency, which are characterized by specialization in low-wage production, over-
urbanization, high levels of foreign debt, increasing environmental degradation, and low levels of health resources in less-developed nations. While many parts of the modernization perspective are supported in this research, much validation is also garnered for world-systems/dependency theories that draw attention to issues of persistent poverty and dependent development in explaining disease trends.

Neoliberal approaches to modernization emphasize the role of economic development in reducing disease, and contend that this should be pursued through market liberalization, integration, and export specialization (e.g. Freidman 2005; World Bank 2008; Rostow 1960). In addition, it is assumed that there is an inherent or automatic relationship between national economic growth and human welfare, where GDP per capita naturally “trickles-down” to benefit the masses. The findings of the present study demonstrate robust counter evidence to some of these claims. Most prominently, GDP per capita has no direct effect on HIV, TB, or malaria rates in less-developed nations; economic development only reduces infectious disease in so far as these resources are channeled to social-health capabilities such as doctors and education.

The fact that an indirect relationship between GDP per capita and disease exists is entirely consistent with the propagations of modernization theory and its attention to non-economic indicators of development, including schooling. The indirect, beneficial relationship was extremely consistent across the models, providing powerful evidence that economic and non-economic modernization factors are extremely important to health outcomes. However, the HIV analyses for Sub-Saharan Africa also reveal areas where GDP per capita and female
empowerment have positive influences on HIV rates, thus demonstrating areas where modernization may not be good for health. Additionally, the absence of a direct relationship from GDP per capita to disease across all models is at odds with a major underlying assumption of the modernization perspective, as well as the current neoliberal development policies that give overwhelming primacy to fostering economic growth. Indeed, Stiglitz (2002, 2007), Harvey (2006), and other authors (e.g. McMichael 2004) demonstrate that the pursuit of GDP growth is often done at the expense of social services for health and other public goods in less-developed nations today, which perhaps could be contributing to these relatively low coefficients. Market-based strategies are often part of conditionality agreements on international funds and support, such as foreign loans. Thus, approaches that deprioritize social services are likely to further disease, as reduced public provisions for schooling, sanitation, and doctors are likely to have profound consequences on disease rates. In this way, development policies that give overwhelming precedence to economic growth over domestic needs and conditions are only compromising developmental gains. The extent to which GDP growth or neoliberal economic integration is being pursued through reductions in health services is certainly an area of inquiry that deserves further attention, in light of the results presented here.

\[47\] Indeed, this would likely take the form of a longitudinal analysis, in order to properly determine if participation in neoliberal development schemes has led to reductions in health or other public services over time. This is one major reason why this particular avenue of research was not pursued in the current study – as cross-sectional SEMs were required to demonstrate pertinent indirect and direct effects. While some information will be lost by employing a longitudinal design, it will be important to examine the extent to which neoliberal development strategies reduce health infrastructure, given the findings of the present study. Many scholars have already found this to be the case in certain regions and locales (e.g. Biggs et al. 2010; Novarro 2002, 2007; Boccia 2009).

In the context of disease, neoliberal modernization strategies gleaned from comparative advantage theory also have harmful ramifications for human health. Specialization in export
agriculture leads to higher rates of deforestation and biodiversity loss, which in turn fuel malaria rates in less-developed nations. Additionally, overwhelming reliance on the land, which is more prominent in nations with large agricultural sectors, is a major vulnerability in a face of climate change. Drought represents one of the foremost effects of altered weather patterns that climatologists see taking shape over Sub-Saharan Africa and most other areas in the Southern Hemisphere, and it represents a major threat to rural livelihoods, spurring movement to cities (e.g. Collier et al. 2008). Harnessing a comparative advantage in agriculture can be therefore linked to human well-being declines, especially given current climatic conditions. Even if this form of economic specialization produces some GDP gains, it will be at the expense of human health, which in the end is likely to be more costly to development (e.g. Acemoglu and Johnson 2007; Packard 2009; Arndt and Lewis 2001; Lorentzen et al. 2008; Ukpolo 2004).

Political-economic interpretations offered by world-systems/dependency theory challenge these methods of development, arguing that economic liberalization and specialization in less-developed nations only promotes underdevelopment due to the nature of capitalist accumulation in the global economy. Indeed, this perspective argues that the world-system hierarchy or inequalities across nations are constantly reproduced, as the structure of international trade and investment benefits core nations at the expense of periphery nations, concentrating low-wage and environmentally-damaging forms of production in poor nations (e.g. Wallerstein 1974, 2004; Hornborg 2001). These structural inequalities lead to the persistence of low levels of social-social-health capabilities in less-developed nations, largely
explaining elevated rates of disease and the persistence of “old” and curable infections in poor regions.

World-systems/dependency perspectives to development also emphasize that unequal patterns in capitalist development will lead to over-urbanization and heightened rates of environmental degradation in less-developed nations (e.g. Bradshaw 1987; Rice 2009). In addition to GDP per capita and social-health capabilities, the influences of over-urbanization and environmental decline were among some of the most robust and consistent across the disease models. Although urban slums represents a new line of inquiry for many scholars (e.g. Davis 2006; Jorgenson and Rice 2010), it is important to point out that the links between capitalist development and urban slum proliferation have been conferred since at least the late 1800s (e.g. Engles 1892).

The findings for environmental change and degradation are especially salient from a world-systems perspective, as core consumption and waste patterns fundamentally contribute to the forms of environmental alteration that are linked to disease in this analysis, including deforestation, biodiversity loss, and drought. Indeed, while affluent nations consume the majority of the world’s food and expel the majority of global greenhouse gas emissions, it is largely people in poor nations who must deal with the environmental effects of core consumption. Although the consequences of climate change have been linked to dimensions of human well-being, especially in the context of natural disasters (e.g. Roberts and Parks 2008), this represents the first study to illustrate how climate change dynamics are also heightening
rural-to-urban migration flows within nations, which in turn have powerful implications for
disease.

Thus, many dependency scholars are quite cautious about the promise of economic
growth and integration to improve the welfare of the people in poor nations. Indeed, the
critiques offered by Stiglitz (2002, 2007), Harvey (2006), and others, while fairly recent, are
largely consistent with arguments made by dependency theorists decades ago (e.g. Amin 1976;
Frank 1969; Wallerstein 1974). The results demonstrate that dependent positions of power in
the world-system, foreign debt, and specialization in agriculture not only truncate GDP per
capita gains, but some models also show these factors reduce social-health capabilities, create
urban slums, and increase certain forms of environmental degradation. Certain classic world-
system indicators that relate to world-system position, debt, and specialization in agriculture,
while influential in the life expectancy, TB, and malaria models, were not evidenced in the HIV
analyses. However, dependency themes do emerge in the significant and robust effects of
environmental decline on urban growth. As HIV is a sexually-transmitted infection, perhaps the
movement of people and issues of female empowerment are more predominant in explaining
prevalence trends across nations. But it is important to keep in mind the dependency factors
that lead to extreme problems with drought and over-urbanization.

Thus a near-exclusive focus on economic development may not be the best way to
improve health and well-being in poor nations. Rather, development should be jointly

48 There were some signs that debt, world-system position, and other dependency indicators had indirect links to
HIV through GDP per capita and the urbanization variables, but issues of model fit precluded their inclusion. This
could also be partly due to data limitations, as the HIV analyses had the smallest sample sizes and increased time-lag
for the independent variables.
promoted through increased attention to social-health capabilities, urbanization, and increased environmental sustainability, alongside economic growth. In particular, doctors, schooling, sanitation, and reduced fertility represent important means to successful development that are likely to intensify future GDP gains, given the well-established links between disease burden and rates of national economic growth (e.g. Acemoglu and Johnson 2007; Packard 2009; Lorentzen et al. 2008). We must take a much broader approach to development for it to be truly successful in poor nations, namely by pursuing development mutually across economic, social, and environmental realms (Brundtland Commission 1987).

6.3 International Health Programs and the Limitations of Ad Hoc Practices

Although treatment of relevant international development policy has been considered in the discussion of modernization and dependency perspectives, public health initiatives and funding deserves more careful attention and critical analysis, in light of the substantive findings gleaned here. As was previously mentioned, supranational health and development agencies have historically played a major role in curbing disease and increasing well-being in poor nations, through the supply of medical technologies and interventions, physicians, and other forms of health resources throughout the 20th century (Soares 2007; WHO 2010). However, new patterns in international health support are emerging, where funding for global health issues apart from HIV/AIDS has decreased substantially over the last three decades (MacKellar 2005). One of the biggest areas of loss includes basic health infrastructure, including provisions for improved sanitation and clean water access. Other studies note an absence of support for the
development of malaria vaccines and new TB and malaria antibiotics, as it is not profitable for core-based pharmaceutical industries to invest in low-return interventions for people of poor societies (Troullier et al. 2002). These points also speak more broadly to a fundamental issue of TB and malaria being neglected or forgotten diseases, given the stark disproportion in health funding and attention on these diseases in comparison to HIV/AIDS. It is no doubt that the re-emergence of these two diseases is at least partly a result of these trends.

Although there is much more support for HIV-related initiatives and research, international agencies are beginning to recognize the “triple threat” of HIV, TB, and malaria, which is a hopeful sign (e.g. UN 2005). As previously mentioned, one of the best and most recent examples of this is the creation of a new agency, The Global Fund to Fight AIDS, Tuberculosis, and Malaria, which is partnered by UNAIDS, the World Bank, and the Bill and Melinda Gates Foundation, among others. The Global Fund to Fight AIDS, Tuberculosis, and Malaria just produced a report, outlining their 5-year plan to address these diseases. The report, titled “The Global Fund Strategy 2012-2016: Investing for Impact”, describes that the fight against the three pandemics is at a crucial juncture in the lead-up to the 2015 Millennium Development Goals deadline. In effort to reduce disease, it will shift to a new model of “Investing for Impact”, which includes “funding in more proactive, more flexible, more predictable and more effective ways.”

The overall health impact goals include 10 million lives saved and 140-180 million new infections averted from 2012 to 2016. The agency plans to achieve these goals by specifically addressing each disease in the following way over the 5-year period: (1) HIV/AIDS: 7.3 million
people alive on ARTs; (2) Tuberculosis: 21 million TB treatments (directly-observed, short-course); and (3) Malaria: 390 million bed nets distributed.

It is clear that these strategies are ad-hoc, individual-level, and at times, post-hoc types of interventions, which are only likely to produce limited and short-term gains. Treatments do not stop or prevent these pandemics from spreading, and the provisions clearly do not address the issue of antibiotic resistance for TB and malaria in any adequate manner. Based on the insights gleaned here, it is likely that investing in broader initiatives that address issues of health infrastructure, such as access and quality of physicians, improved sanitation, increased participation in schooling, would do more to not only prevent these diseases in less-developed nations, but reduce rates of other infectious diseases and health issues as well. Indeed, getting to the underlying causes that stem from issues of persistent poverty is not only going to improve human well-being in a more holistic manner, but also lead to more successful development over the long-term. The fact that support for improved sanitation and clean water is the area of public health funding that has seen the greatest decline over the last 30 years is especially frightening in light of the results presented here. The Global Fund does include one small statement late in the report addressing investments on “strengthening health systems”, but it is clear that this is not a primary area of funding or concern.

Critical demographers note that ad-hoc health interventions in poor nations often only tend to shift the burden to other diseases or afflictions. For example, Soares (2007) points out that less-developed nation’s lack of stable and well-integrated health provisions can mean that vaccination of one disease might simply increase mortality from competing causes of death,
therefore only having a modest impact on overall quality of life. It is also relevant to emphasize that the vast majority of international public health funding is going to providing anti-retroviral therapies (ARTs) to HIV-infected individuals in poor nations. ARTs do not prevent, nor cure HIV. In fact, a most critical point of view acknowledges that ARTs simply lengthen and improve the life of HIV-infected individuals, thereby only prolonging and increasing opportunities for further transmission. Albeit quite costly to the households that need them, TB and malaria treatments and TB vaccines are cheap compared to ARTs. Even if we are going to continue to almost exclusively concentrate on individual-level treatments as the solution, I argue it is crucial that we at least examine if these medical resources are being used in the most effective and beneficial manner, that is, in ways that save the most lives and prevent further disease transmission most successfully.

That being said, increased provisions for drugs are completely useless if there are not adequately trained physicians to administer them safely, or if poor populations struggle to access medical resources. TB and malaria treatments require adherence to rigorous scheduling and complex regimens to be effective, which depends on the existence of high-quality health resources. If not, issues of antibiotic resistance are only going to increase, producing even more profound effects on mortality. Thus, there must be amplified attention to the broader, underlying causes of disease in order for the existing methods of addressing them to be effective. The analyses reveal that this is likely to be especially important for urban areas in developing countries, and that such provisions may even improve health despite being located in the most destitute settings of the city. The very fact that TB and malaria pose continued threats
to mortality in poor nations, despite known treatments and interventions, signals that individual-
level or ad-hoc approaches are not working to curb these diseases.

In extending the focus of health funding to these larger, underlying structural issues, another area that deserves increased international public policy attention are provisions for environmental refugees. The most recent estimates of climate change dynamics suggest that issues of drought and other forms of ecological change or decline are only going to increase over time, and it is the poorest nations that are most vulnerable to these threats, both geographically and socio-economically. The resulting growth in urban areas from these environmental patterns must be managed in some way. Additionally, while support for environmental policies is comparatively harder to garner than for issues of public health, the results of the analyses demonstrate that environmental degradation has a direct bearing on health outcomes, thus making conservation and sustainable management of the environment a human health issue.

The results from the Sub-Saharan Africa disease model also illustrate that policy makers need to be careful to consider the local or regional context when developing disease interventions. Something that is beneficial in one region may serve to jeopardize health in other areas, as was demonstrated here in the case of HIV, for GDP per capita, female empowerment, and urbanization. Thus, changes to one area of society can have profound and unintended effects on other components, so we must be sure when promoting international-level initiatives that they are being carried out in specific locations in ways that takes into account the particular context in which these interventions are being applied.
6.4 Limitations and Future Research

Although there is great reason to examine the cross-national determinants of disease, there are a few limitations which must be acknowledged. One potential issue is that aggregate-level data is often represented by averages or per capita estimates, which tell researchers little about the distribution of these factors within nations. There is some evidence from international studies of income inequality which demonstrate that using data for all people in the world (at the individual-level) can produce different findings or conclusions than when using national-level estimates (e.g. Milanovic 2005). In related vein, national-level data sometimes cannot appropriately capture the specific mechanisms that would help to explain cross-national disparities in disease rates. For example, it would help if we knew more about the patterns and motivations surrounding rural-to-urban migration, or the specific processes that cause urbanization to be linked to HIV in Sub-Saharan Africa but not necessarily elsewhere. While we sacrifice much from solely taking an individual-level approach, there must be more attention given to the ways that cross-national and individual-level factors combine to contribute to disease trends. One possibility to address this involves using hierarchical linear modeling techniques which can take into account both national-level and individual-level attributes.

Even this is prescribed with caution however, as issues with multicollinearity and an exclusive look at direct-effects only can lead to erroneous conclusions. Overall, I argue that there are far too many factors that operate indirectly for comparative researchers who study issues of development and health, that we must be extremely cautious when interpreting these kinds of results in the context of public policy. Indeed, the ability of GDP per capita to
dominate other relevant indicators when modeled as competing predictors has at least partially contributed to a near-exclusive focus on national economic growth as the principle way to improve human welfare in poor countries (e.g. World Bank 2008; Stiglitz 2002, 2007; McMichael 2004; Pritchett and Summers 1996). The very fact that GDP gains are in some nations are being pursued through the reduction of social services (e.g. Stiglitz 2002, 2007; Navarro 2007) should stand out as being contrary to what we know fundamentally about how human health, or human capital more broadly, contributes to successful development (e.g. Sen 1999; Acemoglu and Johnson 2007).

This study also brings to light the utility of taking an interdisciplinary perspective. In trying to explain the underlying causes of disease, we must be attuned to social, economic, infrastructural, and environmental factors. The role of environmental characteristics in particular in influencing infectious disease represents a key area of interdisciplinary research that deserves far more attention. In addition, more cross-collaboration within disciplines is clearly needed, as many findings from case-study or survey research are not being applied to or tested across cross-national assessments (such as the findings for female empowerment, urbanization, and HIV in Africa). Being able to evidence causal processes at multiple scales not only leads to more comprehensive assessments of disease, but can illuminate where to apply what strategies, thus increasing the effectiveness of current interventions.
6.5 Conclusion

The body of research dedicated to exploring infectious disease has not only neglected to consider the forgotten plagues of TB and malaria, but also focuses on the proximate, individual-level predictors of health outcomes. However, it is time to examine more distal factors associated with disease in order to attack the root causes of these three pandemics. Without a structural explanation, we are limited to behavioral explanations, which are not sufficient when disparities in health are patterned according to national-level characteristics, or when we see infectious disease concentrated in certain areas of the world and certain strata of the world-system. Addressing the underlying structural conditions that accelerate transmission and death from disease could potentially help more people in more ways than programs aimed at specific interventions for certain groups of people. Although ending global poverty and inequality is an arduous task, we can at least begin by addressing the most fundamental issues of sanitation, schooling, and doctors in less-developed nations. Indeed, if we continue to neglect the importance of basic health infrastructure, it is likely that evolving patterns in antibiotic resistance could lead to a re-emergence of the forgotten diseases even among populations in affluent nations.

Though issues of poverty, environmental degradation, and disease are often considered to be stand-alone threats to development, it is clear that these ills are combining and influencing one another to threaten human welfare in less-developed nations. In order to pursue development most successfully, there must be even attention to economic, social, and environmental factors, as a one-sided approach will only contribute to developmental declines
over the long-term. TB and malaria have plagued human societies for centuries; thus the very fact that people are still suffering from these diseases, given known cures and interventions, is particularly alarming and telling that the current strategies for development and health are not working. Although HIV is a seemly “new” infection, it also follows long-standing patterns of poverty and inequality, and thus in many ways is not a unique threat, but simply the latest version of unrelenting trends in infectious disease. As the ramifications for the reproduction of international inequality is clear, both the new pandemic and the forgotten diseases are going to be persistent features of underdevelopment in poor nations.
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