MINORITY HIV RATES, INEQUALITY, AND THE POLITICS OF AIDS FUNDING

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Dissertation Prepared for the Degree of

DOCTOR OF PHILOSOPHY

UNIVERSITY OF NORTH TEXAS

August 2012

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Doctor of Philosophy (Political Science), August 2012, 135 pp., 19 tables, bibliography, 180 titles.

Since the 1990s, the HIV/AIDS epidemic has increasingly impacted minority groups in the United States, particularly African Americans. Why is this happening? Comparative studies of developing nations have convincingly established a relationship between concentrated poverty, ethnic boundaries, and lack of effective governmental response as contributing to high levels of infection in those countries. To date, however, no study has sought to apply these insights to the American context. This dissertation endeavors to show that, first, marginalization of U.S. sub-groups most at risk of infection is largely a product of poor health outcomes associated with concentrated urban poverty and economic stratification. Second, this sub-group marginalization is exacerbated by the politics of retrenchment which increasingly privatizes risks onto individuals, states, and non-governmental providers. The net result of these changes is a U.S. health care system too fractured to recognize and respond to changes in HIV/AIDS demographics.
ACKNOWLEDGEMENTS

Previous versions of Chapters 3 and 4 were presented at the annual meetings of the Southwest Social Science Association in Las Vegas, March 16-19, 2011, and the Western Political Science Association in San Antonio, April 21-24, 2011. Thanks to my reviewers for all of the helpful comments received at both meetings, especially Martin Johnson of the University of California, Riverside, and Regina Branton, University of North Texas, for their in-depth critiques of the methods employed therein.

Special thanks also to Jonathan Rothwell of the Brookings Institution and Douglas Massey, Princeton University, for providing their data on density zoning and class segregation. I owe a debt of gratitude, especially, to Sonia Arbona of the Texas State Department of Health Services for her invaluable help in locating data on HIV/AIDS prevalence and socioeconomic indicators for Texas counties and zip codes.
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CHAPTER 1

INTRODUCTION

1.1. Problem Statement

Broadly stated, this dissertation seeks to answer two general questions. The first of these is: what is the relationship between HIV/AIDS and poverty in the United States? The growth of HIV/AIDS infection among minorities, and especially the urban poor, makes answering this question very important to our understanding of disease prevalence and ways to deal with the epidemic. There are firm theoretical reasons, described below, to posit that structural inequalities inherent in the American system are at least indirectly responsible for the disproportionate HIV infection rates among racial and ethnic minorities. These groups are less likely to be economically mobile, have access to medical care, proper nutrition, and healthful living conditions.

Second, how have government policies exacerbated disproportionate infection rates? A growing literature on the politics of retrenchment, defined as the privatization of risk onto individuals, states, and non-governmental providers, demonstrates how this process affects those segments of society which can least afford to bear the costs. The main proponents of this theory, however, admit that specific types of policy need to be examined in order to establish its validity. Since social policy domains vary so widely, the politics of AIDS policy retrenchment must be studied in isolation to determine if the theory holds.

How do poverty, social context, and public opinion interact to drive this public health problem? Additionally, why do governmental responses at all levels, seem incapable of stemming the tide of infection among vulnerable populations? This dissertation seeks to provide
answers to these questions as well. I will examine proximate causes of disproportionate infection rates in minorities and their relation to social and structural inequalities. Then, the connections to public opinion and policy outputs are explored. I conclude that major changes are needed before the problem of HIV/AIDS prevalence in the U.S. can be addressed.

In the course of trying to answer these questions, anomalous findings kept appearing. A letter from Senator Michael Enzi to the Government Accountability Office in 2006 outlines the crux of the problem. The Ryan White Initiative, which provides federal funding for HIV/AIDS prevention and care, has been in place since 1990, yet the estimated number of new HIV infections has not decreased. Over the same period, however, AIDS deaths have remained remarkably constant (Bauer 2007, Culshaw 2007), and have declined since 1985. As detailed below, CDC has noticed this tendency, but cannot offer a solid explanation. These anomalous findings sent me deeper into the literature on HIV and AIDS than I had thought would be necessary.

Culshaw (2007) recounts similar experiences. Only after writing a thesis and dissertation on mathematical models of HIV/AIDS transmission and prevalence did she come to the conclusion that the basis of the epidemiological theory underpinning these models may contain serious inconsistencies. If it were only a small group of scientists and social scientists noting these anomalies, it would be easy to dismiss these voices as small pockets of dissent to the orthodox theory. However, a great many scholars have begun to publish a large corpus of work on the difficulties inherent in trying to equate AIDS risk strictly with sexual behavior.

This dissertation is an attempt to unravel some of these inconsistencies and to offer a partial solution. The addition of poverty to the explanatory framework makes some sense of the oddities surrounding this disease’s prevalence patterns. While poverty defines the contours of this disease - the poor are more likely to die of AIDS defining diseases but public perceptions and governmental responses continue to focus on risk and blame based on sexual assumptions. As Culshaw argues, AIDS is “so mired in emotion, hysteria, and politics that
is no longer primarily a health issue. AIDS has been transported out of the realm of public and personal health and into a strange new world in which pronouncements by powerful government officials and ill-informed celebrities are taken as gospel…” (Culshaw 2007, 4). This represents a fundamental disconnect between the reality of, and response to, one of the major health problems of our time.

Part of the problem with investigating the correlates of HIV disease is the de facto wall of separation between the social and biological sciences. Social scientists are discouraged from looking at the epidemiological evidence behind the disease because this is not what they do. Consequently, the biomedical community is able to effectively “black box” AIDS research and publish in highly specialized journals that only a few read and understand. This phenomenon helps create and preserve, as Sarewitz tells us, a “policy myth” which all of the scientists and engineers sign onto, since it justifies government funding of the research and development budgets (Sarewitz 1996). Moreover, social scientists look at these problems strictly through the lenses of their own carefully circumscribed fields of endeavor. As long as these walls of separation exist, no integration of knowledge is likely to occur. This study is partially an attempt to bring some of these lines of inquiry back together and investigate some of the fundamental claims behind them.

1.2. Background

Congressional funding of AIDS research and prevention efforts began in 1982 after it was discovered that a mysterious disease was afflicting homosexual men in New York and San Francisco. From the beginning, funding decisions involved difficult and often conflicting considerations. Among these are the tradeoffs involved in channeling monies toward research and treatment as opposed to preventative and educational efforts (Stoto, Blumenthal, Durch & Feldman 1988). Further, the decisions of lawmakers became embroiled in cultural debates as political elites argued about the rightness of pledging federal funds to fight a disease
thought, by some, to have been caused by an immoral gay lifestyle (Patton 1990, Cohen 1999).

During the 1990’s and 2000’s, however, the face of AIDS began to change. With increasing frequency, AIDS became a disease affecting the black community. In the years 2001-2005, in a 33 state study, it was found that while blacks were only 13 percent of the population, they accounted for 50.5 percent of all HIV/AIDS diagnoses. By contrast, whites were 72 percent of the population but made up only 29.3 percent of all new diagnoses. Figures show that black men are diagnosed with HIV/AIDS at seven times the rate of white men and black women at twenty times the rate of white women (CDC 2007). Additionally, among women, African Americans and Latinas constitute 80 percent of all infections, with a growing number of infections occurring in girls between the ages of 13 and 19. Given this changing face of AIDS it is essential to ask: has government funding of AIDS prevention and treatment efforts kept pace?

Early work on the relationship between HIV/AIDS rates and poverty found the connection to be tenuous and likely due to other contributing factors such as imbalances between population and aid programs (Caldwell 2000, Butler 2000). Reports presented at the 2010 International AIDS Conference in Vienna, however, confirm the ties between poverty and HIV transmission (Dugger 2010). The International Monetary Fund, World Bank, and World Health Organization gave cash payments in the form of monthly stipends to 3,800 school age girls in Malawi and compared them to girls who did not receive payments. The study finds that those who receive the stipends are half as likely to be infected with herpes or HIV after 18 months. Paid subjects are more likely to delay sexual contact and to report condom use.

Yet, despite these new insights and a handful of small studies, there is little effort to define this important causal link. Much of the research into poverty and HIV/AIDS infection is being done in the international literature (Stillwaggon 2006, Lieberman 2009, Epstein 2007, Poku 2005). More effort is needed to dispel the presumption that since the U.S. is a rich
democracy, it does not have the same dynamics of ethnic boundaries and income inequalities (described below) that drive infection rates in developing countries. To date, few studies of American HIV/AIDS rates has established the relationship between structural inequality, defined as concentrated urban poverty, and risk of infection. Linking these phenomena would provide important insights in restructuring public policy.

Of additional importance, while previous scholarship has noted that government responses inadequately address the problem of differential infection rates among minorities, no study has established the mechanism by which government efforts fail. Previous literature emphasizes the influence of secondary marginalization whereby minority groups further marginalize disadvantaged sub-groups, especially the gay and drug addicted through social stigmatization (Patton 1990, Cohen 1999). However, secondary marginalization occurs on many levels. In this study, I seek to demonstrate that social stigmatization is accompanied by a measurable marginalization in terms of wealth and health care resources by privatizing health risks onto those groups least able to afford them.

Part of the rationale for not funding prevention during the early days of the epidemic was predicated on the idea that HIV transmission is primarily a moral issue (Stoto et al. 1988). If, however, it can be convincingly demonstrated that this is not the case in all situations, and that structural inequalities drive the growth of this epidemic, then my study has potential importance in guiding future policy directions. Finally, in the last chapter, I look at the provisions of the new national health care bill, passed in 2010 to determine if health prospects are likely to improve for the poor and infected.

As mentioned in Section 1.1, most of the evidence linking HIV/AIDS prevalence and ethnic boundaries has been done from the comparative perspective. Lieberman (2009) finds that in countries where strong inter-ethnic divisions are found and there is a history of anti-race policies, such as India and South Africa, HIV infection rates go up and remain high. In
countries with low ethnic boundaries and strong governmental responses, Brazil and Uganda for instance, infection rates go down.

Do strong racial/ethnic boundaries exist in the United States? The literature on American racial politics indicates that they do. Some research finds that in social contexts with high levels of racial diversity and socioeconomic status (SES), individuals exhibit higher levels of support for social programs like welfare and affirmative action. Low SES and high ethnic diversity, on the other hand, decrease support for redistributive policies (Branton & Jones 2005). In a similar vein, Bledsoe, et al, (1995) demonstrate that the levels of racial composition, or neighborhood homogeneity, affect both black and white attitudes toward race relations. Based upon these findings, it is likely that living in a racially bifurcated context in which persons of different races compete for resources and community programs, ameliorative social policy is more likely to be politicized(Hero & Tolbert 1996). Further, Dawson (1994) finds that government policies have exacerbated racial inequalities in income and social status. This study is based theoretically on the idea that these phenomena are reciprocal and mutually reinforcing, serving to further marginalize disadvantaged groups.

1.3. AIDS and Minorities: Two Theories

Analyzing the literature reveals two major strands of explanation. The first emphasizes primarily social behaviors as the underlying driver of minority HIV infection rates (Lieberman 2009, Patton 1990, Cohen 1999). According to these theories, minority groups that live in bifurcated contexts - in which they represent a large subgroup within a dominant majority - minorities will be marginalized in terms of opportunity and outcome. The second set of theories is predicated more upon socioeconomic indicators (Stillwaggon 2006, Butler 2000). In conditions of poverty, minorities simply do not have access to the resources needed to live healthful lives (Oppong & Harold 2010, CSDH 2008). This can increase susceptibility to disease in general while simultaneously failing to curtail high-risk behavior.
The research outlined above reveals a very mixed explanatory picture. It is unlikely that either racial or economic factors alone are the singular predictor of disease prevalence among minority populations; it is likely that they interact. However, if it is determined that racial attitudes are more important then there is little that can be done aside from trying to soften racial intolerance through education and socialization. If the socioeconomic factors are more credible, on the other hand, then structural adjustment as Lieberman (2009) has shown in the case of Brazil and Uganda, is more likely to stem the tide of disproportionate minority infection.

The current literature has not gone far enough in trying to sort this out. Lieberman’s study (2009) includes empirical measures of ethnic stratification by country, but does not compare social and socioeconomic determinants. Providing an answer to the question of why social and socioeconomic factors compare in their explanatory power could have valuable implications. The model presented below endeavors to take a step in that direction using Texas as a case study. Texas has high HIV infection rates, high income inequality and, perhaps most important, a highly bifurcated social structure with Anglo, Latino, and African American populations living together in urban areas.

1.4. The Political Economy of HIV/AIDS

A growing body of literature recognizes that epidemics like AIDS are a product not merely of biological processes, but social ones as well. The realization that “the structure of social relations” grows out of the “system of economic production” has reshaped discussions about the proximate causes of disease prevalence (Singer 1998, 4). Butler argues that poverty is not a sufficient cause, *per se*, for the epidemic. Rather poverty acts as a cofactor both by promoting certain types of behavior, and by restricting knowledge, education, and access to medical resources (Butler 2000). Ignoring aspects of the problem like unequal access may worsen prevalence rates in vulnerable populations.
Part of the problem in identifying the most at-risk groups lies in conceptualizing the underlying processes behind prevalence. Several researchers note the unsatisfactory performance of spatial models in identifying the diffusion of HIV and AIDS in populations (Shannon & Pyle 1989, Palloni 1996). Other research notes that economic and political factors pushing the spread of disease must also be coupled with cultural and social contexts in order to be fully explanatory (Craddock 1996). Cultural “values” like policy priorities, socioeconomic status, and gender must also be considered as powerfully interacting forces driving the epidemiology of disease (Okigbo, Okigbo, Hall & Ziegler 2002).

Importantly, an economic system characterized by high levels of inequality tends to be self-reinforcing. An unequal distribution of income has the unexpected consequence of further reducing the elasticity of monetary resources. This is because inequality reduces the degree to which income is used to address poverty reduction (Fosu 2010, Adams 2004). Consequently, rising levels of inequality tend to increase, rather than decrease poverty at a rate proportional to the mean income level. Not surprisingly, unequal conditions affect groups differently. Where structural conditions disfavor a racial group or groups relative to a more dominant majority, distributional inequity is even more likely to exacerbate the problem. Opportunities to rise out of poverty are further restricted (Fosu 2010).

Sociologists note that the political economy of AIDS that has extended its reach into the African American community can be traced to a couple of major causes. First, the illegal drug trade is a major source of equal opportunity employment in inner-city environments where opportunities for work above minimum wage are few (Carlson 1996, Singer 1998). Second, while there is no evidence that African American women are more promiscuous than other Americans, the inner-city environment of poverty, racism, and classism lead to a loss of “self-efficacy” (Sobo 1998, 98). This loss of efficacy can lead women to tolerate unsafe sexual attitudes of their male partners. Sobo’s survey of inner-city women showed that they were often in a state of risk-denial about their sexual relations. Importantly, women with higher
prospects of economic mobility and stronger “extra-conjugal” networks are more likely to use condoms.

As mentioned above, most of the literature on structural inequality, poverty, and HIV/AIDS infection rates has focused on sub-Saharan Africa and other developing countries. While the data are readily available, few studies examine these relationships in developed or advanced economies. How does the United States rank among developed nations? Figure 1 provides a graphic representation of the correlation between HIV infection rates and the GINI coefficients of countries classified as having “advanced economies” by the International Monetary Fund (IMF) 1.

Figure 1.1. Graph of HIV and GINI Coefficients, IMF Advanced Economies

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1HIV rates are expressed as percentage of the population infected and are taken from World Health Organization estimates obtained through www.globalhealthfacts.org
The GINI Index is a common, well acknowledged measure of income inequality (Firebaugh 1999). It ranges from zero to one with one being complete inequality, so the higher the number, the greater the levels of income disparity between the rich and the poor. Since 1980, the GINI index for U.S. communities has increased 6.5 points. The lowest level recorded was in 1968, when it fell to 38.6, and it climbed to its highest level ever in 2006 at 47.0. By comparison, the estimated GINI for 1929 was 45.0 during the stock market crash that precipitated the Great Depression. As an additional comparison, the average GINI among European Union member nations is 31.

Another significant point can be inferred from the GINI data: it is not strictly poverty, but relative poverty, that correlates with high rates of HIV infection. Previous research on African AIDS has relied on measures like the Human Poverty Index, compiled by UNAIDS, or per capita GDP and found no, and even negative, correlations between poverty and AIDS. Curiously, in the African context, wealthier countries like South Africa bear an unusually high disease burden despite having better infrastructure and ample resources. When we apply the GINI, however, a different picture emerges. A simple analysis of variance test shows the very significant statistical relationship between relative inequality and adult HIV prevalence among 112 countries for which both measures are available.

<table>
<thead>
<tr>
<th>Variables</th>
<th>F-Statistic, df</th>
<th>p &gt; F</th>
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<tbody>
<tr>
<td>Income Inequality and HIV Rates</td>
<td>3.60, 46</td>
<td>.001</td>
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Additionally, since funds allocated by the federal government are administered by states and localities through grants under the Ryan White Act, it is important to look at how sub-governments spend these monies. A plan for detailed analysis of state and urban expenditures is outlined for Chapter Six below. A look at the funding formula for federal grants to states
reveals that aid is largely mismatched in terms of targeting groups most at risk. This is because states and legislators compete for money, leaving the system mired in politics.

While the graphical representations presented in the two figures are not sufficient to establish a causal relationship, they are illustrative of the theoretical contribution of this dissertation (the theory and hypotheses are outlined in the next section). Social and racial policies are thoroughly enmeshed in the competition for government funds. Multiple layering in administration of these funds further complicates the social milieu under which they are distributed. This important insight acts as the rationale for including implementation as the major theoretical base in studying the impact of government policy.

1.5. Theory and Data

An early version of my study revealed the following general patterns. Table 2 shows the correlations between, first, AIDS cases per capita and the urban black poverty rate and, second, AIDS cases per capita and the number of persons who report having had an HIV test. These data are state-level and culled from CDC studies of disease prevalence in 2007. The urban poverty rates are drawn from U.S. Census Bureau estimates over the same period and are state aggregated so that the unit of analysis is the same. The ANOVA correlations indicate that the measured relationships both reach conventional levels of significance. The strong correlation between HIV test rate and disease prevalence, however, leave lingering questions, which will be discussed below.

If $x$ and $y$ have the same assumptions but reach different conclusions, we must ask how this is possible. Patton (1990) insists that there is not a connection between knowing one’s HIV status and corresponding or subsequent behaviors. HIV testing, she claims, only serves to further marginalize by forcing sex and drugs “underground.” The data do seem to lend

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2 Note that the N is smaller for urban poverty rate correlation. This is due to the fact that state-level black urban poverty rates are unavailable for Hawaii, Idaho, Maine, Montana, New Mexico, New Hampshire, North Dakota, Oregon, South Dakota, Utah, and Vermont.
support to her contention about HIV testing. However, I will discuss later in Chapter Two, there is still a great deal of silence surrounding the disease and the disproportionate risks born by women and persons of color. Further, while testing and surveillance have little measurable effect on transmission rates, there is a high degree of variability in the degree to which tests are given in public health clinics administered by states and localities. This tendency is part of the puzzle, and is addressed at length in Chapter Six.

The theory guiding my study can be summarized as follows. Poverty is the missing link between minorities and disease risk. However, policy decisions miss this connection and focus instead on risk- and hazard-based models. This tendency causes local governments to focus disease prevention efforts primarily on mandatory testing of “at-risk” populations based on sexually transmitted epidemiological models. Thus, the disconnect between disease reality and perception is cemented in policy based on the following items:

Table 1.3. Areas of Policy to Risk Mismatch

<table>
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<th>Misperceptions of AIDS Risk</th>
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<td>Responses to the AIDS Epidemic Focus on Risk/Blame</td>
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<tr>
<td>Political Ideological Approach to Funding Decisions</td>
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<tr>
<td>Moneys are Funneled through State and Local Sub-Governments</td>
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<tr>
<td>States and Localities Focus on Mandatory Clinic Testing as a Means of Prevention</td>
</tr>
<tr>
<td>Result: Over-Diagnosis of HIV Cases and Decreased Funding for Those Who are Actually Sick</td>
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</table>
Simply stated, the central theory guiding my project is that the phenomena outlined above are not unconnected. Rather, they are mutually reinforcing. The same forces of racial stratification apparent in the cross-national studies (Lieberman 2009) are present in microcosm here in the United States. This marginalization through social and economic forces has enabled further marginalization through policy change. Policies which do not aim at prevention and do not address the underlying issues reflexively feed the problem they seek to address. Funding for U.S. AIDS programs is emblematic of the problems associated with this approach.

Disproportionate infection rates in minority groups are a function of:

(i) Not simply minority status, but a nexus of poverty, urban status, and socioeconomic inequalities (Chapter Four).

(ii) Urban contexts, which contribute to vulnerability through limited access to services; income inequality; fragmented and ineffective prevention efforts out-sourced onto private non-governmental providers (Chapters Four and Six).

(iii) Inaccurate opinions about disease susceptibility which leads to poor representation on the part of legislators which has led to inequities in federal HIV/AIDS funding efforts by the U.S. Congress (Chapter Five).

(iv) Poor policy implementation: a fundamental mismatch between federal funding under the Ryan White CARE/HOPWA Act and vulnerable communities (Chapter Six).
The data collection section below details the variables used for the regression and proportional hazard models in Chapters Three and Four and the state/local typology in Chapter Six. Building up to these central analyses, updated data are presented that highlight the central importance of income inequality culled from the primary theoretical literature (Brady 2009, Kelly 2009, Stillwaggon 1998, Lieberman 2009) and the data on retrenchment (Pierson 1997, Hacker 2004, Hacker 2002, Skocpol 2000). These data, however, are straightforward and can be presented descriptively through tables and graphs. As such, they are not described in detail here.

The rationale for my study comes from a household impact study conducted in South Africa in 2002 (Booysen & Bachmann 2002). The explanatory variables are taken from the Census Bureau’s Small Area Health Insurance Estimates (SAHIE) for Counties and States. The county-level data correspond to the geographic areas covered in the CDC urban area study. Each urban area is given a unique FIPS code by the Census Bureau which is easily matched up with the CDC infection data. The CDC data are from 2007 and the SAHIE data are from 2006, but there does not seem to be any serious disjunction between these estimates.

Descriptive statistics from CDC, IASP, and the Census Bureau are presented throughout the opening chapters. National and state data on poverty and HIV rates are presented culminating in an urban multi-state combined model. The centerpiece of the first four chapters is an OLS regression of urban infection rates conditioned on poverty and racial stratification variables. The dependent variable is operationalized as percent infected in 102 major metro areas, as determined by the CDC. Since the variable is continuous, OLS is the appropriate methodology. The list of the 102 urban areas to be included in the study, and their corresponding percentage HIV infection rates are listed in Appendix A, Table 0.1.
1.6. Plan of the Study

In this chapter, I have outlined the theory to be employed. Following the three major sets of theories in order, the following chapters will test each set of assumptions roughly in the order that they are presented in the literature review in Chapter Two. Chapters Three and Six are roughly similar in that they test the connections between poverty, or material disadvantage, and HIV and AIDS rates. The intervening Chapters Four and Five are more political, and explore the links between public opinion, legislative action, and the ways in which funding decisions have become vested in policy. Throughout the first three chapters, descriptive statistics from census tract data, the Institute on Assets and Social Policy, OECD and CDC will be presented in tables and figures. A summary of the chapters presented below outlines the major literature, but is not exhaustive.

Chapter Two assesses the theoretical explanations, culled from the literature, for the disproportionate infection rates among minority groups. Following the basic outline below, I draw on the literature to establish the tie between poverty and HIV infection among U.S. ethnic minorities who are far more likely to live in conditions of poverty. As Stillwaggon argues, transmission and spread of HIV “are not simply mathematical functions of sexual behavior” (Stillwaggon 2006, 5). The economically disadvantaged have less access to basic healthcare leading to a greater incidence of a variety of diseases which can also be exacerbated by poor nutrition and vitamin deficiencies. Further, social and political marginalization based upon behavioral explanations of HIV transmission has important implications in the provision of public health services.

The literature outlined highlights the contention that it is not merely poverty, which alone contributes to poor health outcomes, but also the unequal distribution of wealth and resources that leads to disempowerment and systematic marginalization. Stillwaggon argues that “structural adjustment policies” enacted by most developed economies throughout
the 1980’s and 90’s, which reduced public health services, served to increase the “cycle of poverty” based in uneven income distribution (1998). As an empirical demonstration, Chapter Two culminates with data obtained from the Institute on Assets and Social Policy (IASP) at Brandeis University, demonstrating that the racial wealth gap has increased four hundred percent since 1984. These data are compared to the increase in minority infection rates to show that the two phenomena have risen linearly and tend to mirror each other over the years since 1990.

Following the theoretical base presented in Chapters One and Two, a study of HIV prevalence in Texas is the subject of Chapter Three. As Texas is a unique mix of three major racial groups with three very different HIV epidemiological profiles, it provides a unique opportunity to study the interaction of race, poverty, and social context. In addition, variables for levels of racial and economic stratification, or bifurcation, are constructed based upon the theoretical guidance of Lieberman (2009) and Hero and Tolbert (1996). These data also come from the CDC and the Texas State Department of Health Services.

Additionally, a proportional hazard model for progression of HIV infection to AIDS with similar independent variables is also presented with the “failure” parameter defined as the contraction of full-blown AIDS (Box-Steffensmeier & Jones 1997). The second analysis is equally important in light of the fact that with the current economic recession, the government program that provides antiretroviral drugs has been “ravaged” (Sack 2010). The number of people on the waiting list to receive government subsidized drugs has increased each year since 2008.

Chapter Four takes the findings from the social/structural data analysis and compares them to public attitudes about HIV/AIDS from major national survey data. How do policy inputs and the perception of social groups effected by HIV/AIDS impact how AIDS funding efforts are administered? Answering this question is the focus of the chapter. Analyses of two major national surveys are presented, showing important differences in levels of support
for specific types of policy. The results also show that public perceptions are driven more by ideology than they are by social and religious attributes among individuals surveyed.

Chapter Five follows the theoretical guidance (Hacker 2004) of Chapter Four and examines funding decisions in the U.S. Congress. Much of the current debate surrounding public policy focuses on the assessment of risk (Beck 1992). Under this auspice, and given the poor media coverage of the changing face of AIDS noted in the previous chapter, it is easy for lawmakers who oppose state policies that address a problem caused by perceived immorality to make subtle changes to undesirable services. As Hacker notes, policymakers do not need to enact major policy reforms but merely need to make incremental changes that move provisions away from the groups that most need assistance (Hacker 2004, Thelen 2003, Adema & Einerhand 1998). This gradual “drift” produces the exact types of policy-to-risk mismatches seen in U.S. AIDS policy. These subtle policy changes began with the Reagan Administration and continued under Clinton (Skocpol 1996).

While the arguments presented by Pierson (1997) and Hacker (2002) greatly advance our understanding of how policy changes are often highly politicized and, as such, fail to respond to changing circumstances, there is much more work to be done. Hacker insists that these changes are often “subterranean” and not apparent to the general public (Hacker 2004). As such, he recommends looking at individual areas of social policy since the subtle reconfigurations that occur are not uniform across all policy domains. Aggregate analysis cannot tap the ways in which efforts are targeted in specific communities. Much of the federal money allotted under Ryan White are given as grants to community outreach centers and clinics. It is likely that a high degree of variability exists in the relative success rates of these centers as a result of social dynamics and simple know-how. Using aggregate CDC prevention numbers as a proxy is very unlikely to adequately measure these important differences.
An early paper on federal funding of AIDS initiatives found that at its inception funding decisions were hampered by poor communication between scientists and the federal government (Stoto et al. 1988). Whether this is still the case is an open question. The CDC and NIH have a hand in the budget process as they send their budget requests to the OMB and subsequently to Congress every two years. The President then writes his budget based in part on executive and independent agency requests. His budget goes through the mark-up process in congressional subcommittees who recommend funding levels for the Department of Health and Human Services who is tasked with administering the CDC and NIH. So the process is circular and fed by funding requests, many of a contradictory nature, at various points in the process. In this atmosphere, it is hardly surprising that politics is thoroughly enmeshed in the process of budgeting deliberations.

Chapter Six examines the question of risk perceptions further. The main finding is that using aggregate measures leads to misunderstanding of disease risk. Mandatory testing is primarily used as a means of prevention in some localities funded under federal programs. However, there exists a lack of concrete testing and reporting standards so it is entirely possible that HIV prevalence numbers are artificially inflated. Further, under the current competitive funding system, there is little incentive to make sure that the numbers are completely accurate.

Given the obvious dearth of accurate information on all sides, it is necessary to compile numbers in order to ascertain how these labyrinthine processes have impacted AIDS policy. The most obvious point of analysis is the policy output - how is the money spent at the state and local level? This is the rationale for the community social service study outlined in the Data Collection Section. It is important to ask why changes in the provision of care under the Ryan White/HOPWA Act recommended by the Government Accountability Office, meant to improve the program’s effectiveness for the most vulnerable groups, were not enacted (GAO 2006). The data will show that there is an important tie to the public
opinion results in Chapter Four, present among legislators, driving decision making. These perceptions have left the program too fragmented to fully address the HIV/AIDS epidemic.

A restatement of the problem and a review of the findings are presented in the concluding Chapter Eight. Does being politically disadvantaged or simply being economically disadvantaged contribute more to the disproportionate HIV/AIDS infection rate in the black community? Is the problem of minority infection exacerbated by poor or misplaced prevention efforts or more a result of structural inequalities within communities? These questions are reassessed in light of the principal findings. Finally, I will try to provide a more satisfying answer to the question of how to amend public policy going forward.

Finally, a brief diversion is made which explores how the provisions of the new national health care bill, most of which are scheduled to take effect in 2014, serve to ameliorate the disproportionate effect of HIV/AIDS on minority groups. It is unclear whether specific provisions aimed at alleviating the gaps in coverage by collectivizing risk actually help those at the bottom of the socioeconomic ladder. The bill was criticized for allowing too many concessions to private insurance companies in order to obtain passage. With this in mind, a good deal of doubt remains. As this study seeks to address the broader impact of poor provision of health benefits in the context of long-term AIDS care, this is an important final question to be considered.
CHAPTER 2

HIV, AIDS AND MINORITIES: THEORETICAL EXPLANATIONS

As Chapter One detailed, African American communities in the U.S. are disproportionately afflicted with HIV and AIDS. Attempts to explain this phenomenon among social scientists typically rely upon some variation of marginalization or vulnerability theories. Marginalization, as defined by Cohen (1999), refers primarily to the social and power structures inherent in American society. As described below, the black community has been historically disenfranchised and short of political clout, thus the poor, addicted, and homosexual sub-groups within a racially marginalized minority are at even greater risk of being socially disadvantaged. This can lead, in turn, to high risk behaviors.

Vulnerability, on the other hand, focuses more on material disparities. Further, the economically disadvantaged have less access to basic health care leading to a greater incidence of a variety of diseases which can also be exacerbated by poor nutrition and vitamin deficiencies. The World Health Organization (WHO) is taking seriously the possibility that poverty and lack of access to health resources are cofactors in increasing AIDS susceptibility. Their 2008 report on urbanization and disease argues that rapid urban growth accompanied by social and economic inequality produce “hidden cities” (CSDH 2008, xiii). In these contexts, inhabitants are more prone to the “triple threat” of chronic diseases like diabetes or cardiovascular disease, injuries and violence, and infectious diseases like AIDS and tuberculosis. This combination creates “vulnerable places” in which people become generally more susceptible to disease.
Both theories offer powerful insights into the roots of the connections that tie poverty and HIV infection among U.S. ethnic minorities who are far more likely to live in conditions of poverty. Stillwaggon argues that transmission and spread of HIV “are not simply mathematical functions of sexual behavior” (Stillwaggon 2006, 5). Further, social and political marginalization based upon behavioral explanations of HIV transmission has important implications in the provision of public health services.

The burden of caring for the infected in U.S. communities increasingly falls upon private providers such as charitable organizations and church and religious groups. In this context, it is important to review the literature on religion and homophobia, and social responses to the AIDS crisis. In what follows, I will examine each of these explanations in turn. First, the primary social explanations are laid out. These studies insist that societal marginalization of minorities, especially gay minorities, is the driving force contributing to risk. Secondly, I will look at research which argues for a more socioeconomically-driven model of disease prevalence. The guidance of these studies provides the rationale for the statistical models in the following chapters.

2.1. Socio-Behavioral Explanations

The foundations of my study owe to the ground-breaking work of Cathy Cohen. She argues that the structural inequalities run deeper than simply those that are socioeconomically derived. The African American middle class grew after the 1960s. Loath to endanger these gains, the mainstream black community became less likely to acknowledge the presence of homosexuals and the drug addicted - the groups most likely to contract HIV. Essentially, Cohen argues, the vulnerable parts of the community are “secondarily marginalized” and less likely to feel accepted, reifying social boundaries. Doubly marginalized individuals are thus more likely to engage in risk behavior (Cohen 1999).
Patton argues that AIDS has “rearticulated” social identities in terms of “homophobia and racism, sexism and classism” in ways that “prevent natural allies from forming coalitions in order to address problems raised by the HIV epidemic” (Patton 1990, 6). The conflicting and “multiple” social identities, i.e. black or gay, created a culture wherein gay black men became a minority within a minority. Patton points out that the mainstream predominantly white gay groups attempted to reach out to black gays by encouraging use of terminology which referred to “gay communities” rather than the seemingly monolithic term “gay community.” But gay black men, even in circumstances where stigmatization is not a prominent social issue, often find it difficult to form coalitions since their positions are embedded within the politics of race and class.

An initial look at the state data, however (Figure 2), reveals that states with high minority infection rates (expressed as a percentage of the total number infected of all racial groups) are more likely to be southern and to have moralistic political cultures. Under the classification of cultures popularized by Elazar (Hero & Tolbert 1996, Elazar 1994), moralist dominant states are more likely to view social problems in terms of religious values. As shown below, this type of culture has likely served to further stigmatize at-risk minorities.

Perhaps the most important point to be gleaned from Figure 2 is the correspondence between those states in the upper parts of the graph - those having high black poverty and infection rates - most are racially bifurcated (Hero & Tolbert 1996). Racial homogeneity, as opposed to bifurcation will be discussed in depth and operationalized in the data analysis section of Chapter Three. For the purpose of my study, bifurcation is defined as states with large minority populations and large majority (white) populations. According to Hero and Tolbert, this type of racial composition is more likely to place ethnic groups in competition or conflict with one another. Of the states in the upper quadrant of Figure 2, Alabama, Georgia, Louisiana, North Carolina, South Carolina, and Mississippi all have high levels of racial bifurcation under Hero and Tolbert’s classification. These states also have some of the
highest rates of HIV (CDC 2010b). By this rationale, it is necessary to determine the exact relationship between community racial composition and HIV infection.

Also important is the level of social service expenditures in the states listed above. Most of the states scattered in the upper part of the graph are classified as low service providers. An emphasis on moralism and individualism characterizes the response to social problems in these states which, in turn, conditions a smaller governmental commitment. This tendency has important implications: if homophobia and blame characterize moralistic responses to the crisis, it may lead to less active attempts to curtail the epidemic. Further, blame can lead to inaccurate perceptions of the social forces driving disease prevalence.

\[\text{For more on this, census data and information on social service provision can be found in the Civil Rights Leadership Conference reports on federal, state, and local services at: http://www.civilrights.org/census/your-community/funding.html}\]
As Epstein notes (1996), the activism surrounding AIDS must present a challenge to the politics of power in order to gain credibility. In short, the victims of AIDS must be empowered to help themselves. The gay community has been able to push back, to a certain extent, against the conventional wisdom that has sought to marginalize them by raising awareness and demanding a place in the political discourse. The fact that the black gay community has not been able to make similar gains owes largely to the factors discussed above and to the fact that gay black men often hide their sexual preferences so as to avoid having a “stigmatized identity” (Cohen 1999).

Survey data has provided researchers with a rationale for understanding the reason why HIV transmission among gay men has decreased while concomitantly increasing among heterosexuals. The studies (Bloor 1995, Winkelstein, Wiley & Padian 1988) show that gay men are more likely to have engaged in monogamous relationships. When sex does occur outside of a relationship, they are far more likely to use precautionary measures like condom usage. Another study (Doll, Peterson & White 1992), however, notes that deception is not uncommon. Even among men who self-reported as heterosexual, several also admitted to having intercourse with another male. Further, those who reported as heterosexual were far less likely than self-identified gay or bisexual men to have used safe-sex precautions.

Importantly, there are cultural differences in self-reported sexual orientation between the races. Gay African-American men are more likely to report that they are bisexual, yet Latinos who have sex with men are much more likely to identify as heterosexual (Doll, Peterson & White 1992). Douglas characterizes these differences in terms of an individual’s adherence to “bounded” cultural groups and norms. Among those who strongly conform to a group norm, they may either blame others for risks or hazards, or fatalistically accept risk as inherent in their social circumstance. Some may even perceive a social benefit to be had in engaging in risky behavior. In any of these cases, individuals are resistant to changing their behaviors (Douglas 1985, Bloor 1995).
2.1.1. Social Status, Religious Orientation, and Stigma

A great deal of variation exists among religious communities in their response to the AIDS crisis. Messer (2004), for one, urges a collective effort among churches and congregations in overcoming the stigma against the lifestyles that may have contributed to AIDS prevalence worldwide. Only when churches band together to promote an attitude of caring for all persons equally can the underlying message of prophylaxis be conveyed. Similarly, Wood (1999) addresses the role of religious organizations and their response to poverty and argues that congregations must focus on the demographic and social circumstances in which they are embedded. As important generators of culture and cultural symbology, Wood insists, churches must recognize their place in shaping social and political action.

Other scholars focus on the homophobia and social stigma attached to religious response to AIDS. A recent article (Katzenstein, Ibrahim & Rubin 2010) asserts that the liberal-republican tradition in the United States has a “dark side” of ascription which has served to justify the exclusion of certain groups. When AIDS is framed in a moral language, the victims are often blamed for their disease, and the perceived “causality” of their illness (Kayal 1985, 218). Similarly, “social constructions” of a disease can lead to serious misconceptions about the causes of, and risk factors associated, with transmission. Mistaken beliefs among heterosexuals about the nature of HIV/AIDS are found to be related to lower levels of education, income, and religiosity (Herek, Widaman & Capitano 2005).

A growing body of psychology literature focuses on the effect of mental health on disease progression. A meta-analysis of this research (Logie & Gadalla 2009) shows that while there is a high degree of variation in approaches, the studies show a correlation between mental health and perceived levels of stigma and discrimination which lead to lower rates of disease progression. They also report that stigma is associated with low levels of income, social support, and physical health.

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There is certainly some evidence that homophobia is increased by religious adherence. For instance, Ward’s (2005) study shows that hypermasculinity in black churches, driven by the “anti-homosexual rhetoric of black nationalism” has driven homophobic attitudes. This, in turn, has forced gay and bisexual men to hide their identities, thereby worsening HIV/AIDS prevalence. Increased prevalence, consequently, is likely to have increased homophobic attitudes in areas where HIV rates are high (Ruel & Campbell 2006). These paired phenomena may be important because they are likely to be self-reinforcing, and largely driven by public misperception. Similarly, religious views, according to Burton and Bosek, are often formulated as “principles or narratives” which are then expressed as social claims. These claims can “confound the ethical process” as they influence people’s perceptions of the role of religion in health and disease.

Yet, we should be careful in assessing the role of religious values in the face of the AIDS epidemic. Kowalewski (1990) shows that the responses to AIDS among religious communities are highly variable. They range from blame, to compassion, to blaming the lifestyle, rather than the victim. Even among orthodox Christian groups, belief can be expressed in many different ways as relates to attitudes about homosexuality and AIDS (Ford, Brignall, VanValley & Macaluso 2009). Another study finds that volunteerism among inner-city congregations led away from attitudes that the gay community had to be “controlled” toward acceptance and compassion (Kayal 1992). All of this evidence points to the fact that religious responses to AIDS are not monolithic or uniform; they run the gamut and reflect the variations of the social context in which they are embedded. Later in my dissertation I will examine this phenomenon and show how attitudes relate more to ideology and economic situation, as well.

I will return to the question of religion and public attitudes in Chapter Five. I show that the lines between degree of response to the epidemic are not as clear as the literature indicates when one looks at specific policy indices as measured by survey data. This indicates that levels of support hinge upon individual perceptions of factors driving disease prevalence as well
as ideology and feelings about the proper role of government. However, the data also show that correcting misperceptions about the epidemic could have serious policy implications.

2.2. The Socioeconomic Explanation: Structural Inequality

As outlined above, ethnic boundaries, or strong racial divisions in societies account for differences in infection rates in developing countries (Lieberman 2009). Understated by prior studies is the fact that in every model in which economic indicators are included, measures like GDP per capita are highly significant. This tendency is noted by scholars, examined below, who link disease prevalence to material disadvantage more so than social marginalization. While social stratification may reinforce income inequality, it is material inequities which place societal sub-groups in living conditions more conducive to the spread of disease.

It is not merely poverty, which alone contributes to poor health outcomes, but also the unequal distribution of wealth and resources that leads to disempowerment and the systematic marginalization described in Chapter Two. As Stillwaggon argues (1998) using Argentina as a case study, “structural adjustment policies” enacted by most developed economies throughout the 1980’s and 90’s, which reduced public health services, served to increase the “cycle of poverty” based in uneven income distribution. This income disparity resulted in the compounding of biological risk of all sorts of infection, especially HIV.

What is more, the often neglected aspect of HIV/AIDS is that the impact of the disease cuts both ways. AIDS may actually “impede efforts to reduce poverty” (Piot, Greener & Russell 2007, 314). Piot and co-authors refer to this as the “downstream” effect of HIV/AIDS: HIV decreases productivity, increases healthcare costs, and causes the infected to have to rely on public services. They further argue that in order to effectively fight the epidemic, HIV prevention must be coupled with targeted programs that fight poverty, seek to remove stigma, and promote testing and surveillance. Failing to do so further widens income
gaps and may lead to greater risk of infection. This process of exacerbated income disparity is the subject of Chapter Three.

The role of economic disparity is well-documented in the literature on the politics of race and ethnicity. Claudine Gay, for instance, notes that economic disparity, defined as the distribution of resources, is a determinant of between group racial animosities (Gay 2006). The literature from American studies of racial politics is also highlighted (Branton & Jones 2005, Bledsoe, Welch, Sigelman & Combs 1995). Primary theoretical guidance for this chapter also comes from Kelly (2009) and Brady (2009) whose recent works argue in similar ways that politicizing poverty and reducing redistributive policies have only served to worsen economic disparities.

Data from the Institute on Assets and Social Policy (IASP) at Brandeis University demonstrates that the racial wealth gap has increased four hundred percent since 1984. The median wealth holding for whites, which includes household wealth and assets, rose from 20,000 to 75,000 dollars between 1984 and 2007. During this same period, median wealth among African Americans has remained flat at approximately $18,000 (IASP 2010). The steepest increases in wealth disparity occurred after 1994 at the same time that HIV infection and other poor health care outcomes began to disproportionately affect African Americans.

This tendency has been noted in the popular media. Johnston writes that even as the economy grew in the 1990s, the gains went mainly to the top ten percent, while real income actually fell for the other ninety percent (Johnston 2007). As an illustration, I have collected Gini coefficients for all 50 states and compared them to cumulative AIDS percentages per state. The results are revealing. A one-way Scheffe test, which measures differences between pairs of means in a multiple level analysis of variance, results in an F-statistic of 2.16 ($p > F = 0.0327$). Further, a Bartlett’s test for equal variance yields a chi-square of 18.1407 ($p > chi^2 = 0.078$). This demonstrates, at the least, that these two trends are similar in magnitude.
As previously noted, minority women are infected at rates that outpace men. I can use state data from the U.S. Census Bureau to illustrate how income inequities between genders are related to cumulative HIV infection rates. The 2007 American Community Survey breaks down income disparities between men and women per state by expressing income estimates for female earners as a ratio of male income. For instance, Vermont has the highest ratio of female-to-male earnings, where women earn 84.1% of what men earn per year. Variance testing between cumulative HIV rate and income ratio yields an F-statistic of 4.02 ($p > F = 0.0156$), indicating a significant relationship between the two.

2.2.1. The Nexus of Inequality and Poor Health Outcomes

The aforementioned literature on marginalization and social/economic disempowerment makes important observations about the social dynamics that shape racial and blame-related responses to the AIDS crisis. But how does this link to poverty and susceptibility? Prior research notes that vulnerability to environmental hazard manifests itself more prominently in areas with greater population, less healthful built environments, and complex social structures. This risk can be much greater in federally assisted housing, a context which is disproportionately greater for minorities (Borden, Schmidtlein, Emrich, Piegorsch & Cutter 2007, Cutter, Hodgson & Dow 2001). A similar literature on place vulnerability emphasizes that poverty
and hazard can lead to greater incidence of disease (CSDH 2008). Several major international organizations, as outlined below, have begun to recognize the importance of poverty in explaining epidemics.

What makes the above discussion important is the growing awareness that disease susceptibility hinges upon a combination of factors; an interplay between behavior and environment. Oppong and Harold contend that a combination of bad policies and economics combine to deprive much of the world’s population of a chance at living healthy lives (Oppong & Harold 2010, CSDH 2008). Further, they argue that it is social class and not merely race that defines this relationship. The secondary marginalization described can give us a rough contour of this problem’s dimensions. Simply stated, the marginalized are more likely than those in other classes of society to live in conditions of poverty and unhealthful conditions.

We can usefully define a “health inequity” by looking at the World Health Organization’s description of this concept. They describe it as a condition in which “inequalities are systematic, socially produced (and therefore modifiable) and unfair (CSDH 2008, xiii). When growth occurs in large urban areas, the report continues, the inner city populations are largely ignored by public health officials, creating “hidden cities” which are more susceptible to a “triple threat” of long-term non-communicable diseases like heart disease, injuries and violence, and infectious diseases like HIV and tuberculosis.

Gender also plays a significant role in reinforcing these inequities. Women, especially those in minority groups, are far more likely to become HIV-infected than men. The WHO report recognizes that socially constructed gender institutions are key in cementing inequalities that “systematically empower” men to the detriment of women in urban settings (CSDH 2008, 23). Among the consequences of these systemic barriers are: less access to education and employment opportunities, less control over their own lives and decisions, and, importantly, diminished control over their sexual and reproductive health.
Epstein (2007) argues convincingly that the problems of HIV/AIDS infection in the developing world are inherently political and tied not just to poverty, but linked to income inequalities. Women in countries characterized by large disparities in wealth distribution become marginalized and disempowered. In these situations, women are more likely to engage in risky sexual behavior. This finding squares with the results from the Malawi study (Dugger 2010). According to these findings, providing a stipend to young women in deprived contexts reduces their dependence on traditional sex roles which subsequently lead to higher infection rates.

2.3. Other Explanations: The AIDS “Dissenters”

There are other explanations for the prevalence of HIV among minorities and these are inherently political. There is a large, and growing, literature on the problems with HIV-AIDS theory (Deusberg 1996, Culshaw 2007, Bauer 2007, Epstein 1996, Maggiore 2000, Patton 1990, Root-Bernstein 1993, Mullis 1998, Farber 2006, Lauritsen 1993). These voices are led by Peter Deusberg, one of the pioneers of retrovirus research in the 1970s, and Kary Mullis the chemist who won the Nobel Prize for inventing the Polymerase Chain Reaction (PCR) method of detecting HIV particles. This list includes scientists, mathematicians, medical doctors, sociologists, community organizers. Yet, few Americans have ever heard of their arguments. In particular, Peter Deusberg, who once held a $100,000 per year Distinguished Investigator Grant from the National Institutes of Health (NIH), had his funding cut off after pointing out some of the problems with HIV-AIDS theory (Epstein 1996). As Culshaw notes, the decision to silence dissent was made in a highly politicized environment wherein any deviation from the accepted paradigm is deemed odd or even dangerous (Culshaw 2007).

In Chapters Five and Six, I will discuss some of the poor correlations between HIV and AIDS rates. Bauer (2007), while not the first to demonstrate that the prevalence of HIV does not correlate to the rates of AIDS seroconversion and death rates, makes a strong case that the two phenomena are not related. Simply stated, if AIDS is an incurable disease and
there is a lag between HIV infection and onset of AIDS defining illness, or seroconversion, of roughly eight to ten years, then we should see between 3 and 10% of HIV positives developing symptoms and dying per year. This estimate takes into account the presence of antiretroviral treatments, since access to medications varies between 40 to 64% by state (Kahn, Zhang, Cross, Palacio, Birkhead & Morin 2002). The actual state-level correlations, however, are between 0 and 40, not 3 and 10. Rebecca Culshaw, a mathematician, also notes this phenomenon and points out that it is the HIV rate which appears to have increased while AIDS deaths have remained remarkably constant since 1985. These differences are partly explained by the uneven access to antiretroviral medications, but may also be due to the inequalities inherent in U.S. subpopulations. CDC has also begun to take note of this rather surprising trend (Culshaw 2007).

Some of the correlations between poverty and AIDS are likely to come from drug use. Deusberg has argued to no avail that drug use, and not just intravenous drug use, is correlated with AIDS rates (Deusberg 1996). In Chapters Seven and Eight, I will look at these claims. However, there is evidence that some of what Deusberg is saying may be correct. For instance, a study of prostitutes showed that AIDS was 3.5 times higher among those who also use drugs (Bauer 2007). Additionally, while prevailing views of HIV transmission insist that the virus is only spread by needle sharing, separate studies have shown that HIV was higher among those who did not share needles (Bruneau, Lamothe, Franco & Lachance 1997, Kreuger, Wood, Dier & Maxwell 1990). Another study finds that risk is highest among those who use crack cocaine, which is seldom injected (Moss, Vranizen, Gorter, Baccheti, Watters &

\[I\] will contend in Chapter Six that the addition of poverty causes some of the numbers to make sense. In non-urban settings and low poverty areas, the numbers conform to the tendencies noted by Professors Culshaw and Bauer: they simply do not match up. Among high poverty urban clusters, on the other hand, the correlations between the HIV and AIDS rates are quite high, close to 90%. Bauer and Culshaw are right, but one must disaggregate the numbers before the phenomenon they note is explicable. Doing so, in turn, lends additional support to the idea that poor health and urbanization may be driving the epidemic in minority populations.
Osmond 1994). Certainly the explosion of cocaine usage mirrored the onset of the AIDS epidemic in the 1980s and the movement of the epidemic into minority populations happened after the 1990s when crack cocaine became the underground economy of poverty-stricken black neighborhoods (Deusberg 1996).

These findings, among others, have led many researchers to conclude that AIDS as we know it is not a single disease. It is possible that the early New York and San Francisco cases of AIDS which involved Kaposi’s sarcoma (KS) may be quite different from current cases. It is generally agreed that KS is not “caused” by HIV (Beral, Peterman, Berkelman & Jaffe 1990, Weiss & Jaffe 1990, Miles, Martinez-Mata, Rezai & Magpantay 1992, Matsuyama, Kobayashi & Yamamoto 1991). In fact, research shows that KS responds better to traditional cancer treatments than it does to HIV treatment with antiretrovirals. The KS prevalence of the 1980s was likely due to use of “hot lubricants” during the explosion of the gay party scene in the 80s (Deusberg 1996). Similarly, some of the leukemia co-infections found in AIDS cases respond better when treated with conventional chemotherapy (Clerici 2010). So, AIDS in gays may be quite different than AIDS in drug users, and entirely different from AIDS in Africa which involves wasting diseases and food- and water-borne afflictions. Doctors and politicians may be remiss in treating all of these as instances of the same thing.

Similarly, Root-Bernstien (1993) was one of the first to advance the idea that AIDS prevalence is more akin to an autoimmune disorder than to a sexually transmitted disease. With STDs, rates go up and down and vary greatly over time. With AIDS, however, although HIV rates have increased over the years, AIDS rates and deaths have stabilized and remained surprisingly constant. Further, AIDS deaths declined prior to the widespread use of antiretroviral medications like AZT (Bauer 2007). There is a large and growing body of literature which recognizes this fact. While these epidemiological studies exceed the scope and aims of this project, they are briefly sketched in the final chapter. As mentioned above,
one of the stated goals of this study is to erode the barriers between the social and biological sciences, and the final chapters are a step in this direction.

2.4. Conclusion

This chapter has looked at the literature on marginalization and vulnerability. Through these insights, we see that it is necessary to shed more light on the plight of marginalized groups who are most susceptible to infection. As Boler and Archer argue, the “battle” for prevention needs to be politicized because the prejudice surrounding HIV/AIDS is “bound up with wider discrimination” (Boler & Archer 2008, 75). Effort is needed to break through the multiple layers of stigma that characterize responses to the crisis. Breaking these patterns of discrimination, however, requires groups claiming their place in the discussion and a public discussion of the attitudes that shape social and political responses to the epidemic. This will also require looking at the political consequences of inadequate representation in terms of AIDS policy implementation.

If, on the other hand, the socioeconomic explanations are found to be better at accounting for prevalence disparities among the races, then we would be advised to heed the advice of Stillwaggon (2006) that structural adjustment is needed. The implication is that little can be done about the epidemic’s toll on minorities until efforts are taken to ameliorate the burden of poverty. If HIV is shown to be driven largely by inequities, then the admonitions of the World Health Organization and UNAIDS that vulnerability is driven by urbanization and access can be applied to rate disparities in the United States. This requires abandoning the idea that the U.S. is unique and, as a rich democracy, not subject to the same forces driving disease in other parts of the world. Doing so will acknowledge that the lessons learned by other governments may be effective here as well.

The following chapters will take steps in this direction. The next chapter compares measures of racial and economic marginalization to prevalence as a means of assessing their
explanatory value. While, certainly, both social and economic elements are present as causes of AIDS and its disproportionate effect on minorities, it is important to find out which of these are most primarily responsible. Then, questions of access to health care are coupled with socioeconomic indicators to examine their relations to HIV rates. Subsequently, the policy implications are explored. The central question is explored: how does policy become more responsive to the actual conditions? Chapter Six will look at this issue; if AIDS is more likely tied to poverty and drug usage, are we effectively aiming our efforts at the wrong targets by not seriously engaging the correlates of the epidemic?

The final chapters will also examine some of the claims of the so-called “AIDS dissenters” using data from the CDC and U.S. Census Bureau. In particular, we will look at the poor correlations between HIV and AIDS through the lens of new research on poverty and disease. The seeds of a new understanding of AIDS are already in place, it merely takes recognition and synthesis to sharpen the focus of these efforts.
CHAPTER 3

HIV, AIDS AND POVERTY: A TEXAS STUDY

Are disproportionate minority HIV infection rates in the United States driven more by social factors like secondary marginalization of gay minorities, or by socioeconomic inequality? Attempts by scholars to find out why African Americans and Latinos are increasingly the victims of the AIDS epidemic, relative to Anglos, rely on these two predominant explanations. No study, however, has attempted to reconcile these two theories. Using Texas as a case study, this chapter looks at relative levels of minority integration as opposed to varying levels of income inequities in order to determine their explanatory power. A hierarchical linear model of HIV rates by Texas zip code and county, conditioned on racial and socioeconomic indicators, is presented. The results show that base income and income inequality perform far better than racial bifurcation measures in predicting HIV rates at multiple levels of analysis. However, there are important differences between racial groups which may explain some of the variance.

In previous chapters, we examined the literature positing that poverty, access, and racial marginalization are main factors driving the epidemic’s disproportionate effect on minorities. We will now operationalize these variables. In the two studies which follow, one for Texas at the zip-code level and one for 117 major metropolitan areas in Chapter Four, the theoretical literature of Chapters One and Two are tested with demographic data.

3.1. HIV Rates in Texas

Texas provides a unique platform for studying the impact of HIV/AIDS on communities of color and the contexts of political and economic inequality. With relatively large Latino and African American populations, the state is highly bifurcated in terms of the size of the
minority groups in relation to the Anglo population (Hero & Tolbert 1996). As we will see, HIV/AIDS prevalence in Texas disproportionately burdens the minority population in a manner which is broadly reflective of the unique racial and socioeconomic composition of the state.

The U.S. Census Bureau measures income inequality per state by calculating Gini coefficients by state and major metropolitan area. In 2007, Texas was fifth highest among all U.S. states with a Gini of .4738. Only Louisiana (.4768), Connecticut (.4809), Rhode Island (.5234), and New York (.5831) had higher Ginis in 2007. Of these five states, four (Texas, Louisiana, Connecticut, and New York) are in the top quartile of U.S. states in AIDS cases per capita. Texas is also, as of 2006, fifth in the nation in terms of statewide rates of HIV transmission (CDC 2007).

According to the Texas Department of State Health Services (TDSHS), between 2002 and 2008, the number of persons living with HIV/AIDS increased from 44,508 to 63,019 (TDSHS 2010). When we look at the racial breakdown the patterns mentioned throughout this study are evident, if not more pronounced. Statewide, in 2008, Anglos account for a rate 196.8 per 100,000 of persons living with HIV/AIDS. By contrast, African Americans have a rate of 850.4 per capita—more than 4 times the rate of whites. Over the same time frame, Latinos rates have risen from 138.0 in 2002 to 174.2 in 2008.

Statewide, the highest rates of infection are in Houston, Austin, Dallas-Fort Worth, San Antonio, and the Rio Grande valley bordering Mexico (TDSHS 2010). In all cases, African American rates of infection outpace Anglo rates at a nearly four-to-one ratio. In El Paso and San Antonio where Latinos are the dominant minority group, however, Latino rates are higher than whites and challenge those of African Americans. This could be evidence that either socio-behavioral or socioeconomic factors are driving the differences. The model below will attempt to parse this out.
Table 3.1. HIV, Race, and Urban Context

<table>
<thead>
<tr>
<th>City</th>
<th>Anglo</th>
<th>African American</th>
<th>Latino</th>
</tr>
</thead>
<tbody>
<tr>
<td>Austin</td>
<td>182.2</td>
<td>747.7</td>
<td>176.7</td>
</tr>
<tr>
<td>Dallas</td>
<td>243.5</td>
<td>631.5</td>
<td>158.6</td>
</tr>
<tr>
<td>Ft. Worth</td>
<td>111.4</td>
<td>483.5</td>
<td>98.9</td>
</tr>
<tr>
<td>Houston</td>
<td>241.7</td>
<td>926.5</td>
<td>188.0</td>
</tr>
<tr>
<td>El Paso</td>
<td>93.2</td>
<td>217.4</td>
<td>154.2</td>
</tr>
<tr>
<td>San Antonio</td>
<td>131.9</td>
<td>365.9</td>
<td>175.8</td>
</tr>
</tbody>
</table>

Cell entries are rates per Capita

3.2. Data and Methods

The data on HIV rates are from the Texas State Department of Health Services. There are a total of 1257 zip codes for which complete data on HIV and AIDS rates as well as census data on socioeconomic indicators are available. Additionally, each zip code is matched up to its corresponding county. This yields a two-level data set with 1257 zip code tabulation areas (ZCTA) nested within 222 counties. The HIV/AIDS rate data are from 2008, while the socioeconomic numbers are from the 2000 census. This lag makes sense in light of the fact that AIDS is a disease which can remain latent for years. Similarly, “late-testers,” or those for whom HIV status is not determined until years after infection, can carry the virus for some time before becoming symptomatic (Castilla, Sobrino, de la Fuente, Noguer, Guerra & Parras 2002).

1There are 20 counties, all small rural counties, left out of the analysis due to the fact that complete census data and/or HIV rates are unavailable. The large number of observations in the sample, however, assures that the sample is normally distributed. What is more, the dependent variable has a high degree of variation; it is bounded by zero on the low end (135 ZCTAs report 0 HIV rate), and 7,000 on the top end (a ZCTA in Harris County).
3.2.1. Dependent Variable

The dependent variable is HIV rate per capita by Texas zip code tabulation area. Rate calculations are a standard measure in epidemiologic studies (Szklo & Nieto 2001). These numbers, used in the descriptive statistics above, normalize incidence to population and express results as whole numbers amenable to statistical analysis. The rate calculation is straightforward, as demonstrated by the following example (PLWHA is a common abbreviation for persons living with HIV/AIDS):

\[
Rate = \frac{PLWHA}{Population} \times 10^6
\]

For Example:

Rate of PLWHA for a zip code =

\[
= \frac{23 \text{ PLWHA}}{7,187 \text{ Total Population}} \times 100,000
\]

\[= 320.02/100,000 \text{ (Rate per capita)}\]

Additionally, using a rate calculation makes the dependent variable conform better to the sub-level variables listed below. The census variables used to measure socioeconomic status are expressed as percentages. To improve model performance all measures are specified as continuous whole numbers so that equivalent units appear throughout the analysis.

3.2.2. Explanatory Variables

If disease susceptibility is conditioned by unhealthy living conditions poor access to health care, we should expect to see higher infection rates in concentrated urban environments, as the WHO posits (CSDH 2008). To measure this, a simple percentage of the population living within urban clusters is calculated for each ZCTA. Additionally, since people in urban contexts are more likely to live in poverty, the first SES indicator is simply household median
income - a blunt measure of economic capacity. Use of this variable is well-established in the literature (Kalipeni & Ghosh 2007, Masanjala 2007).

The second set of variables is straightforward. The overall percentage of minority population, percentage with less than high school education, and the percent unemployed are calculated for each ZCTA. These are obtained simply by dividing the raw numbers from the census data for each SES indicator by total population per ZCTA and converting to percentage. These are more or less standard SES indicators. It is expected that education will have a strong effect as those from low-education backgrounds are more likely to harbor inaccurate perceptions of disease risk (Bowleg, Belgrave & Reisen 2000, Boler & Archer 2008).

Finally, two variables are added to capture the effects of bifurcation on HIV rates. Theoretical guidance for constructing these indicators comes from Hero and Tolbert (1996) and the literature on GINI indices (Firebaugh 1999). The racial bifurcation variable is expressed as a ratio of the minority population in relation to the total white population for each ZCTA. In a similar manner, the income bifurcation measure is a ratio of the number living below poverty compared to those above. Of note, in minority - majority ZCTAs, the income bifurcation ratio may be greater than one.

3.2.3. Method of Analysis

The rationale for using hierarchical linear modeling lies in its ability to analyze variance between subjects while simultaneously considering information from higher levels of analysis (Steenbergen & Jones 2002). The parameters are not fixed, as they are in a simple linear regression, but allowed also to vary across the level-2 unit clusters. These variable coefficients enable the model to identify fixed versus random effects. Additionally, by considering nested levels, the model more generally conforms to the assumption of uncorrelated errors, reducing the possibility of Type I error (Luke 2004).
It could be argued that aggregating up to the zip code level as a method of inferring individual-level behaviors is an example of the classical “ecological fallacy” (Lancaster & Green 2002). In other words group characteristics do not necessarily describe the risk behaviors of individuals within that group. However, Pearce (2000) points out that several epidemiological studies recommend using multilevel regression as a means of overcoming any sort of systematic bias since variance at more than one level of analysis is compared. Further, while individual level statistics are collected by CDC and Texas State Department of Health Services, these data are not generally considered suitable for analysis of this sort since doing so could conceivably compromise patient confidentiality. Thus, while these models are admittedly coarse, we can at least gain some insight on the social characteristics which may increase HIV susceptibility among individuals living in these areas.

3.3. Results

The results of the hierarchical linear model are presented in Table 2:

An LR test of the model’s log-likelihood reveals that the model performs significantly better than the straight linear model without nested levels (Luke 2004). As an additional robustness check, I reran the zip code level data as an OLS with clustered standard errors. The standard errors were essentially the same. Since the main purpose of the hierarchical model is to test the variance between subjects and between groups, looking at the errors provides the critical test of the model’s fit (Steenbergen & Jones 2002). The χ² statistic is also highly significant (p>0.001), which indicates that the model performs well in explaining the variance between subjects and levels.

The most significant effect in the model is household median income, followed by the urban context and education variables. This provides support for the theory that socioeconomic status coupled with urbanization are associated with high-risk behaviors. Similarly, a higher number of persons with low educational attainment correlates to an increased HIV rate.
### Table 3.2. HLM of HIV Rates and SES Indicators

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Std. Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent Urban</td>
<td>.669**</td>
<td>.302</td>
</tr>
<tr>
<td>Household Median Income</td>
<td>.005***</td>
<td>.001</td>
</tr>
<tr>
<td>Percent African American</td>
<td>5.198***</td>
<td>.784</td>
</tr>
<tr>
<td>Percent Latino</td>
<td>-.908</td>
<td>.655</td>
</tr>
<tr>
<td>Percent High School or Less</td>
<td>10.519***</td>
<td>2.303</td>
</tr>
<tr>
<td>Percent Unemployment</td>
<td>-.334</td>
<td>1.038</td>
</tr>
</tbody>
</table>

*Bifurcation Measures*

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>Std. Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Income Bifurcation</td>
<td>1.784**</td>
<td>.715</td>
</tr>
<tr>
<td>Minority Bifurcation</td>
<td>-.016*</td>
<td>.008</td>
</tr>
<tr>
<td>Constant</td>
<td>386.834***</td>
<td>59.552</td>
</tr>
</tbody>
</table>

Significance levels: * p<.10, ** p<.05, *** p<.001

N = 1257; Number of groups = 222; Avg. per group = 5.7

The insignificant and negative effect of unemployment is curious but theoretically-grounded. Studies have shown that some level of economic livelihood is necessary for disease transmission since it leads to an increase in social relations (Masanjala 2007).

Minority status is marginally significant but it is clearly overwhelmed by poverty. This suggests that while minority status is predictive, being urban and poor is much more important. This contention is reflected more clearly in the bifurcation measures. Being income bifurcated is significantly and positively related to HIV rates, lending support to the hypothesis that income inequality - being a member of a poor subgroup of a wealthier general population - feeds infection rates. As mentioned above, however, the effect is not as strong as base-level income. Interestingly, minority bifurcation is insignificant and inversely related to HIV rates,

We cannot tell from the model, however, the interactive effects of race, poverty and urban context. If we split up racial groups and rerun the analysis for the percentage population represented by Anglos, African Americans, and Latinos in each zip, we can better see the effect of SES on each individual grouping. In the following analysis, the dependent variable for HIV rate from the previous model is used as an independent variable and the SES variables are regressed hierarchically on the percentage population of each racial category in zip codes and counties.

What is particularly interesting about the results in Table 3.4 is the direction of the coefficients. We must keep in mind that we are conditioning on percentage of each racial group, not on HIV rate, so the coefficients indicate the differential effect of race on HIV and each of the SES indicators by each ZCTA and county. The results indicate there are very large differences between the races and suggest that these account for the variable rates detailed in this study. Further, the results point to a highly interactive relationship between race and context.

What the results show is that being an African American greatly increases the probability that one will live in an urban context and have a markedly lower household income despite the fact that educational attainment is significant and inverse. Blacks are also much more likely to live in contexts of both income and racial bifurcation. In fact, the highly significant \( (p > .001) \) effect of all of the variables except unemployment (which is in the expected negative direction, however) indicate that African Americans are far more likely to live in highly unequal circumstances. This, in turn, can explain the aforementioned fact that blacks are diagnosed with HIV/AIDS at four times the rate of whites.

While Latinos are more likely to be urban, they are far less likely to be unemployed and have low levels of household income. Further, they are less likely to be at risk of infection
Table 3.3. HLM of Racial Group and SES Indicators

<table>
<thead>
<tr>
<th>Variable</th>
<th>Black</th>
<th>Latino</th>
<th>Anglo</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIV Rate</td>
<td>.007***</td>
<td>-.004***</td>
<td>-.001</td>
</tr>
<tr>
<td></td>
<td>(.001)</td>
<td>(.001)</td>
<td>(.001)</td>
</tr>
<tr>
<td>Percent Urban</td>
<td>.034***</td>
<td>.115***</td>
<td>-.192***</td>
</tr>
<tr>
<td></td>
<td>(.011)</td>
<td>(.010)</td>
<td>(.012)</td>
</tr>
<tr>
<td>Household Median Income</td>
<td>-.000***</td>
<td>.000</td>
<td>.000***</td>
</tr>
<tr>
<td></td>
<td>(.000)</td>
<td>(.000)</td>
<td>(.000)</td>
</tr>
<tr>
<td>Percent High School or Less</td>
<td>-.307***</td>
<td>1.930***</td>
<td>-1.460***</td>
</tr>
<tr>
<td></td>
<td>(.074)</td>
<td>(.061)</td>
<td>(.079)</td>
</tr>
<tr>
<td>Percent Unemployment</td>
<td>-.050</td>
<td>-.200***</td>
<td>-.082</td>
</tr>
<tr>
<td></td>
<td>(.047)</td>
<td>(.049)</td>
<td>(.057)</td>
</tr>
</tbody>
</table>

**Bifurcation Measures**

<table>
<thead>
<tr>
<th></th>
<th>Black</th>
<th>Latino</th>
<th>Anglo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Income Bifurcation</td>
<td>.065***</td>
<td>-.081***</td>
<td>-.052*</td>
</tr>
<tr>
<td></td>
<td>(.026)</td>
<td>(.022)</td>
<td>(.030)</td>
</tr>
<tr>
<td>Minority Bifurcation</td>
<td>.003***</td>
<td>-.001***</td>
<td>-.002***</td>
</tr>
<tr>
<td></td>
<td>(.000)</td>
<td>(.000)</td>
<td>(.000)</td>
</tr>
<tr>
<td>Constant</td>
<td>20.894***</td>
<td>-11.484***</td>
<td>91.769***</td>
</tr>
<tr>
<td></td>
<td>(2.265)</td>
<td>(2.185)</td>
<td>(2.509)</td>
</tr>
</tbody>
</table>

Significance levels: * p<.10, ** p<.05, *** p<.001

Standard errors in parantheses

despite strikingly lower levels of educational attainment. The bifurcation measures suggest that they are more likely to be integrated with other racial groups, and the other indicators point to the conclusion that they are closer to whites socioeconomically than they are to
blacks. The striking similarities between Anglos and Latinos provides overall support for the idea that a social and economic context much closer or even better than Anglos in relative terms places rates of HIV infection much closer to whites except in areas where Latinos are severely disadvantaged.

This contention can be brought into sharper relief. Dividing up urban and rural contexts shows how race and socioeconomics interact with living conditions to create vulnerable environments. The next analysis uses the same dependent variable but filters the counties by percentage of the populations that live in rural versus urban settings. The first column of coefficients is regressed on HIV rates in counties that are more than eighty percent rural; the second column coefficients are for counties where the population is more than eighty percent urban.

Table 4 illustrates racial differences in context and HIV rates by racial group in Texas. The table shows that in rural environments, only the African American variable is strongly predictive of HIV prevalence. In urban settings, however, income and percent African American, as well as education level, are strongly predictive of prevalence. The Latino variable is insignificant and negative, suggesting that Latinos are more economically mobile than blacks, even in urban environments.

Further, the bifurcation measures add some weight to the contention that economics are more important than simply being a minority. The minority bifurcation measure is significant and negative, suggesting that mixed high minority is inversely related to rates. The income bifurcation measure, however, is highly predictive. We can more simply illustrate these differences with the descriptive data in Table 5. The highest rates of relative poverty growth are among Latinos. Similarly, the highest relative rates of HIV prevalence increase are among high-poverty Hispanics (TDSHS 2010). According to the hierarchical regressions, we should expect to see the percentage of HIV cases per year increasing as a function of relative poverty. This is exactly what we see happening in the Latino population.
Table 3.4. HLM of HIV and SES Indicators, by Urban/Rural Status

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rural $&gt;80$</th>
<th>Urban $&gt;80$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Household Median Income</td>
<td>.001</td>
<td>.008***</td>
</tr>
<tr>
<td></td>
<td>(.001)</td>
<td>(.001)</td>
</tr>
<tr>
<td>Percent African American</td>
<td>1.941**</td>
<td>7.012***</td>
</tr>
<tr>
<td></td>
<td>(.861)</td>
<td>(1.243)</td>
</tr>
<tr>
<td>Percent Latino</td>
<td>.282</td>
<td>-1.315</td>
</tr>
<tr>
<td></td>
<td>(.551)</td>
<td>(1.234)</td>
</tr>
<tr>
<td>Percent High School or Less</td>
<td>-1.139</td>
<td>16.830***</td>
</tr>
<tr>
<td></td>
<td>(2.330)</td>
<td>(3.864)</td>
</tr>
<tr>
<td>Percent Unemployment</td>
<td>5.385</td>
<td>-1.116</td>
</tr>
<tr>
<td></td>
<td>(7.196)</td>
<td>(1.375)</td>
</tr>
</tbody>
</table>

*Bifurcation Measures*

| Income Bifurcation                | .827        | 1.510*      |
|                                  | (1.131)     | (1.012)     |
| Minority Bifurcation              | -.005       | -.024**     |
|                                  | (.020)      | (.012)      |
| Constant                          | 12.225      | -470.408*** |
|                                  | (72.009)    | (91.454)    |

Number of Observations             | 649         | 608         |

Significance levels: * $p<.10$, ** $p<.05$, *** $p<.001$

Standard errors in parantheses

The results of the models presented above and the descriptive statistics presented in Table 4 suggest that if relative poverty levels among Latinos stay constant or worsen, so too are HIV rates likely to rise. This trend will likely accelerate if the Hispanic population moves
Table 3.5. Texas HIV and Poverty Rates

<table>
<thead>
<tr>
<th>Indicator</th>
<th>White</th>
<th>Black</th>
<th>Latino</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent Poverty</td>
<td>11%</td>
<td>30%</td>
<td>34%</td>
</tr>
<tr>
<td>Percent of HIV Cases</td>
<td>33.3%</td>
<td>42.6%</td>
<td>21.4%</td>
</tr>
</tbody>
</table>

increasingly into the cities, and into conditions of concentrated urban poverty. Systematic disempowerment, accompanied by decreased social and economic mobility, these results suggest can lead to a more casual acceptance of high-risk behaviors.

3.4. Conclusion

The findings provide overall support for the theory that socioeconomic factors more directly condition minority infection rates. As we have seen, however, there are important inherent racial differences. It appears that African Americans are the out-group in terms of economic and social resources. To address these differences, future AIDS policy and prevention efforts will need to take demographics into account in designing ameliorative programs. The results suggest, additionally, that it may not be possible to decrease infection rates without corresponding efforts to reduce poverty and structural inequalities.

The question of access is one of the more significant problems in American health care policy. Cochran, et al, summarize the problem by insisting that American health care is “maldistributed” (Cochran, Mayer, Carr & Cayer 2009, 270). Medical service providers tend to gravitate to the areas in which the profit potential is greatest, which are typically the more affluent urban and suburban areas. As a result, the populations most in need of medical services, the impoverished inner-city denizens, are usually the most under-served. Numbers for percentage insured and access to public health clinics are available for major metropolitan areas through the Census Bureau. They are not available for all Texas ZCTAs. Additionally,
the differences in amount and type of access points between the urban and rural areas that characterize Texas demographics would impose serious modeling limitations.

This study has obvious limitations. This study does not model for access. Therefore, no attempt is made to break down infection rates by mode of infection (e.g. male-to-male sexual contact, intravenous drug use, or heterosexual contact). This information would be useful, as one would expect women in contexts of poverty to be especially susceptible to heterosexual transmission. Similarly, males coming from prison would be more likely to be infected through same-sex modes. Case level data, however, are protected by strict patient confidentiality rules and are unavailable for this type of analysis. Also, mode of transmission, at least in the Texas case, has changed very little between 2002 and 2008 (TDSHS 2010). Thus, it is unlikely that social factors affecting differential infection have changed substantially in the last decade.

However, this study has taken a preliminary step in sorting out the factors contributing to disproportionate minority HIV rates. This chapter has examined the relationship between minority HIV infection and socio-behavioral versus socioeconomic explanations. The results suggest that African American disparities are likely to be largely a result of structural differences between the races. Iterations of this research will look at the implications of SES as a major predictive factor. The conclusions suggest that future policy will need to take these links seriously in order to address the problem of AIDS and minorities.
CHAPTER 4

PUBLIC SUPPORT FOR HIV/AIDS FUNDING

Political attitudes are an important linkage between lawmakers and constituencies. Some research indicates that the correlation between attitudes and policy areas does not yield a “neat correspondence” (Peffley, Hurwitz & Sniderman 1997). Others find that “data driven” models perform well in predicting when stereotypes relate to specific policy opinions (Bobo & Kluegel 1993, Terkildsen 1993). AIDS policy is an area in which opinion is largely driven by the way individuals feel about homosexuals and the perceived rights of persons living with HIV/AIDS (Herek & Glunt 1991). Although AIDS is an acknowledged public health risk, information about the ties between poverty and risk are not generally to be found in the media. Thus, attitudes are likely to be conditioned by sexual or racial stereotyping.

In this chapter, I look at two national opinion studies. The first, the General Social Survey (GSS) asked questions in its 1988-1991 panels about support for government programs to help persons with HIV/AIDS. Unfortunately, the GSS stopped using these questions after the 1991 study. To supplement this study, the American National Election Studies (ANES) panel from 2004 is used. The models presented below measure support for AIDS programs conditioned on demographic, ideological, and religious variables. The results show that attitudes toward AIDS programs, and specifically negative feelings about how we should fund programs, are largely conditioned by how individuals feel about homosexuals. Yet, we see evidence in Chapters Two and Three, that AIDS is driven largely by poverty, inequality, and urbanization. This is important for understanding how public opinion maps onto the changing reality of AIDS, and we will subsequently look at how this translates to policy in Chapters Five and Six.
The results of the two studies reveal that attitudes toward funding HIV and AIDS initiatives are largely conditioned by ideology and individuals’ feelings about the rightness or wrongness of homosexuality. Contrary to much of the literature outlined in Chapter Two, the way people feel about different types of AIDS programs is not primarily conditioned by levels of religiosity. Rather political leanings and the degree to which respondents believe that AIDS is a product of improper social behavior more directly condition attitudes. These studies provide a link to the next chapter in which we will examine the ways ideology conditions the actions of lawmakers in shaping federal policy.

4.1. Political Representation and the Disadvantaged

Social and racial politics are thoroughly enmeshed in the competition for government AIDS funds. Where strong ethnic boundaries exist, policies are more likely to involve blame and stigmatization (Lieberman 2009). This tendency is exacerbated where informal social institutions serve to divide rather than unite community groups. The decline in HIV infections in the white gay community was due in large part to community-led responses aimed at fighting high-risk sexual behavior (Martin 1987, CDC 1987). Where these community responses are lacking, however, governments must often step in to spur responses. And this requires accurate information flows to government from the communities themselves.

The response to the AIDS epidemic among the gay community, however, was not apparent in black communities. No organized lobbies sprang up and no black congressional leaders took the lead in proposing ameliorative legislation to curb the disease’s disproportionate effect on communities of color (Cohen 1999). Rather, what followed was a process of “secondary marginalization.” As Cohen documents, black leaders and the burgeoning black middle class through the 80’s and 90’s were gaining political clout. Loathe to compromise these gains, the concerns of the black gay community were downplayed or ignored by black leaders. There was a push to separate the “respectable” from the “deficient” parts of the black culture. Further,
we found in Chapter Three, that minority infection rates are more directly conditioned by material disadvantage which places the marginalized at an even lower level of political access since they lack not only the clout, but also the resources, to make their voices heard. In this political environment, the impetus did not exist to protect secondary minority groups.

Political access is essential, especially among the under-represented, in obtaining access and policy objectives for their respective groups (Pantoja 2005, Swain 1993). This knowledge, it has been pointed out, is essential both for expressing their interests to elected leaders and for holding them accountable (Delli Carpini & Keeter 1993). Yet, minority groups are historically short of this important political resource (Swain 1993). This inability to express interests to elected leaders can lead to crucial inequities in the competition for government funded programs. We will see below that minorities are substantially more likely to support programs to assist those living with HIV or AIDS, however, there still appears to be a fundamental disconnect between intention and outcome.

Some research indicates that white representatives do just as well at representing the interests of minority groups as do those of minority status. Swain finds that white Democrats do well at supporting black interests and do so upward of 90 percent of the time as opposed to Republicans who only do so at a rate close to 35 percent (Swain 1993). Her indices, however, are built around civil rights and redistributive issues. This contention is challenged in the face of an issue which pits secondary minority groups against not only a dominant majority, but against other minority groups as well. As we will see in the next section, the gay as well as the poor and drug addicted in the black community are further disadvantaged not only by the dominant political system, but by their own communities as well.

While political knowledge is not directly modeled in the analysis which follows (my intention is to look at policy rather than community dynamics), this discussion is nonetheless crucial to our understanding. I will argue below that the disproportionate infection rate among minorities is related to fundamental misunderstandings of disease risk among information-poor groups.
And, as I will argue in the next section, black leaders, enmeshed in the politics of power, have done little to change perceptions of the disease’s impact on these sub-communities. Moreover, if government efforts are aimed more at disease control than prevention, then this tendency acts to further disadvantage under-informed groups who are most at risk. To be effective, obviously, initiatives must target those communities which need them most.

Moreover, Mansbridge argues (Mansbridge 1999) that in “contexts of mistrust” lack of descriptive representation may actually hamper the promotion of substantive interests. In these contexts, especially when accompanied by a history of “political subordination,” the specific knowledge of under-advantaged groups held by descriptive representatives can lead to greater recognition of “uncrystallized interests.” To accomplish this, leaders of under-represented groups must create an environment wherein these groups feel empowered to rule. Similarly, Dovi (2002) contends that in order for descriptive to translate into substantive representation, leaders must recognize and sympathize with dispossessed groups.

Strolovitch finds that interest groups do a poor job of representing disadvantaged sub-groups within their own membership (Strolovitch 2006). It is likely that interest groups in the AIDS community are emblematic of this phenomenon. In their study of AIDS activism, Jennings and Andersen (2003) find that of those who join in the cause, almost half (48 percent) are gay men, most are well-educated and have middle-to-upper class incomes. Only 7% have education levels of high-school or less and only 16 percent have incomes below $21,000. Also, among AIDS activists, 60 percent report following public affairs “most of the time.” Since AIDS, in its current context, is a disease which disproportionately afflicts the poor and politically disadvantaged, these trends are extremely important.

In terms of the findings on AIDS activism, a notable consequence is that interest groups tend to neglect “policy issues that affect low-income people in favor of those that are of interest to middle-class members” (Strolovitch 2006, 908). If interest groups do not adequately or accurately represent the needs of their sub-groups, it enables lawmakers to make cuts to...
social programs, such as the provision of health services, and to force them onto individuals and private institutions. This is the subtle, and often hidden, process of retrenchment which is discussed at greater length in the next chapter.

What is more, research shows that in states with high income inequality the dynamics of opinion on AIDS funding are markedly different. In states with large income differences, the poor are more likely than the economically well-off to perceive that inequality is a problem (Xu & Garand 2010, Bartels 2008). This can lead to important political consequences. Prevailing state economic conditions and ideology are the primary determinants of levels of support for AIDS programs (Ohsfeldt & Gohman 1992). These factors relate not only to perceptions of risk and the efficacy of education programs, but also to levels of support for coercive measures against persons living with HIV/AIDS.

Of particular importance is Gilens’ contention that heuristics and media influence shape Americans’ responses to public expenditures for social services (Gilens 1999). Further, inaccurate media depictions of policy-specific ignorance can lead to variations in political attitudes even among the most politically informed (Gilens 2001). These findings reinforce the previously noted contention (Cohen 1999) that the media have not adequately portrayed the changing face of AIDS. This is directly related to the contention raised above that accurate political knowledge is essential to expressing the needs and interests of social sub-groups. The national news outlets have consistently focused overwhelmingly on the gay face of AIDS. Even the black media focus on the more “established” parts of the black community and tend to express a marked “discomfort” with the “immoral” lifestyles of those at risk of infection.

Certainly, the way the issue is framed can lead to inaccurate perceptions about effected populations and disease prevalence. A “specific issue frame” that “links government activities with targets in society” can have a powerful influence on public opinion (Jacoby 2000, 751). AIDS funding by the federal government is an issue related to a certain target group, mostly the gay community, leading to its association with individual evaluations on the rightness or
wrongness of homosexuality. In the models that follow, I will condition support for various types of AIDS funding on other social attitudes and socioeconomic indicators.

4.2. Two Studies: GSS and ANES

Since the 1980’s, the General Social Survey has asked questions of respondents concerning their individual level of support for government sponsored AIDS programs. The General Social Survey (GSS) is conducted nationally, every two years, by the Social Science Research Center at the University of Chicago. The data program focuses its questions on attitudinal and demographic variables aimed at measuring social change in the United States. Their responses on AIDS-related subject areas indicate mixed levels of social support for various governmental AIDS policies. Table 1 shows the 1988-2006 average support levels for some key policy areas.

Table 4.1. American Attitudes toward AIDS Policies

<table>
<thead>
<tr>
<th>Policy Area</th>
<th>Percent in Favor</th>
<th>Percent Opposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Government should provide disability benefits for PLWHA</td>
<td>57.9</td>
<td>42.1</td>
</tr>
<tr>
<td>Government should pay health care costs of PLWHA</td>
<td>31.9</td>
<td>68.1</td>
</tr>
<tr>
<td>There should be a public info program on safe sex practices</td>
<td>86.1</td>
<td>13.9</td>
</tr>
<tr>
<td>Schools should teach safe sex education</td>
<td>86.8</td>
<td>13.2</td>
</tr>
</tbody>
</table>

Source: General Social Survey, Aggregated Data 1988-2006

There is significant variation between different types of policies aimed at curbing the impact of AIDS on society. Over the period surveyed, Americans tended to overwhelmingly support public information initiatives to teach society in general about safe sex practices. However, they are almost evenly split on providing disability benefits, and largely opposed to government programs to pay the health care costs of persons living with HIV and/or AIDS. As
we will see below, much of this adverse opinion stems from the fact that Americans consider AIDS risk to be a product of immoral behavior. Resistance among the general public to providing care is largely predicated upon peoples’ perceptions that homosexuality is morally objectionable.

Two early studies focused on moral/religious values in America and their relations to attitudes about AIDS. Cochran, Will, and Garner (1994) find mixed support among the religious for ameliorative social policy. Greeley, using a similar framework, also finds a fair amount of ambiguity. Some commonly used variables like church attendance, religious intensity, denomination, and socioeconomic indicators such as age, race, and income, have been culled from the General Social Survey (GSS) to try and explain variation in levels of support. Unfortunately, however, the GSS stopped asking AIDS-specific questions after 1991. To update and refine the GSS measures, a separate model based on the American National Election Survey is presented below.

The following table shows probit estimates on three different indicators taken from the GSS 1991 panel. The dependent variables for the three models gauge two different types of proposed policy. The first two are more authoritarian in nature: requiring AIDS tests and requiring persons living with AIDS to wear identification are meant to tap the degree to which individuals perceive AIDS to be a public threat from which society must be protected. The third is a measure of support for giving benefits to persons living with AIDS. Differences in the number of observations reflect the dropping of observations for persons expressing no opinion to the survey item.

Some of the explanatory variables are included in order to replicate and compliment the previous study (Cochran, Will & Garner 1994). For instance, in addition to the variable asking if homosexuality is “always wrong”, an indicator for social cohesion - the “Rotten Apple” variable - is added. This question asks if a person of perceived poor character can influence society to become less moral as a whole. Other variables are added which gauge
the socializing effect of having known someone with HIV or AIDS, or having received some sort of AIDS education. As with the Cochran study, I found no evidence of endogeneity in the model due to the inclusion of variables.

Table 4.2. Probit: GSS Social Acceptance Indicators

<table>
<thead>
<tr>
<th>Variable</th>
<th>AIDS Test</th>
<th>AIDS IDs</th>
<th>AIDS Benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Political Views</td>
<td>-.203 (.069)**</td>
<td>-.028 (.051)</td>
<td>.096 (.054)*</td>
</tr>
<tr>
<td>Size of Community</td>
<td>.001 (.001)</td>
<td>-.001 (.001)</td>
<td>-.001 (.001)</td>
</tr>
<tr>
<td>Know PLWHA</td>
<td>.002 (.104)</td>
<td>.006 (.095)</td>
<td>-.273 (.180)</td>
</tr>
<tr>
<td>AIDS Education</td>
<td>.460 (.289)*</td>
<td>.138 (.212)</td>
<td>.485 (.214)**</td>
</tr>
<tr>
<td>Church Attendance</td>
<td>.066 (.038)*</td>
<td>.021 (.028)</td>
<td>-.013 (.028)</td>
</tr>
<tr>
<td>Fundamentalism</td>
<td>.075 (.138)</td>
<td>.158 (.098)*</td>
<td>-.003 (.100)</td>
</tr>
<tr>
<td>Homosexuality Wrong</td>
<td>.305 (.080)***</td>
<td>.177 (.067)**</td>
<td>-.353 (.080)***</td>
</tr>
<tr>
<td>Rotten Apple</td>
<td>.038 (.091)</td>
<td>.124 (.067)*</td>
<td>.052 (.068)</td>
</tr>
<tr>
<td>Perceived Class</td>
<td>.032 (.186)</td>
<td>.081 (.129)</td>
<td>.161 (.136)</td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>.002 (.040)</td>
<td>-.009 (.028)</td>
<td>.046 (.029)</td>
</tr>
<tr>
<td>Education</td>
<td>.027 (.036)</td>
<td>.049 (.027)*</td>
<td>.009 (.028)</td>
</tr>
<tr>
<td>Race</td>
<td>-.050 (.217)</td>
<td>-.137 (.162)</td>
<td>-.049 (.157)</td>
</tr>
<tr>
<td>Gender</td>
<td>-.416 (.193)**</td>
<td>-.015 (.140)</td>
<td>-.160 (.143)</td>
</tr>
<tr>
<td>Age</td>
<td>-.005 (.006)</td>
<td>.002 (.004)</td>
<td>-.001 (.004)</td>
</tr>
</tbody>
</table>

Observations: 407 391 382
Pseudo $R^2$: .169 .067 .115

The most obvious conclusion to be drawn from the models in Table 2 is that the strongest effects come from the perception that homosexuality is wrong. By contrast, the other social variables are seen to be relatively weak. The size of one’s community, knowing a person with HIV or AIDS, or having had AIDS education exert little influence on attitudes. Importantly, in light of the discussions in Chapter Three, for the individuals in this survey, class, race, and income do not seem to be associated to levels of support for social programs. The socioeconomic indicators driving risk have little connection to public opinion.
The overall findings are generally supportive of previous studies. In particular, Cochran, Will, and Garner (1994) find a relatively, and surprisingly weak overall effect for the religious variables. Church attendance is marginally significant in the model of support for AIDS testing, but does not exert a strong effect elsewhere. As we will discuss below in the concluding comments, there is simply too much variation in religious responses to the AIDS crisis to make aggregate generalizations from religion variables.

We can graphically represent the interaction between support for mandatory AIDS testing and ideology. The following figure shows the mean predicted probability of support for AIDS testing by political views from very liberal to very conservative. This trend will become much more important as we progress through discussions of how attitudes translate into policy implementation in Chapters Five and Six. As we will see, in general the more conservative members of society are more likely to support mandatory HIV testing. Chapter Six will discuss this finding in more detail.

![Graph of Support for AIDS Testing and Ideology](image)

Figure 4.1. Graph of Support for AIDS Testing and Ideology

To expand the analysis, a general support measure is added from the 2004 National Election Study. This variable is more broad in nature and asks about the respondents’ overall
level of approval for AIDS spending rather than individual types of initiatives. The ideology measure from the models above is replaced by party ID and the AIDS education variable is represented by a more general political information indicator. Support for welfare spending and laws to protect homosexuals gauge individual tolerance and receptivity for ameliorative domestic policy. Importantly, perceptions that income differential, or relative poverty, is a problem in society are measured by the “perceived change in income inequality” variable. The standard socioeconomic controls are also included.

The different measures in Table 3 reflect some similarities to the GSS measures, but reveal important differences as well. In particular, the party preference and homosexuality variables exert the strongest effects. More liberal political preferences strongly condition support for AIDS programs. Not surprisingly, those in favor of laws to protect homosexuals are also more likely to favor AIDS programs. This is an inversion of the result above that “dislike for homosexuals” is more likely to increase support for mandatory testing, but additional evidence for the fact that attitudes are strongly conditioned by perceptions about homosexuality. As in the previous models, religion has little sway on overall attitudes.

Race/ethnicity, income, and level of political information have predictable effects. The fact that racial minorities and those of upper income are more likely to support social programs echoes Gilens’ (1999) contention that these socioeconomic characteristics often lead to greater levels of approval. Similarly, political information is inversely associated with attitudes, but there is a negligible effect for the perceived inequality variable. This finding echoes the contention (Cohen 1999) that there are still huge gaps between public perception and media coverage and the true contours of this epidemic.

The Census Region variable comes close to conventional statistical significance (p= .114). A stronger effect was expected for this variable. Some of the highest rates of infection are in the south, as well as some of the most conservative ideology. It is, however, likely that the
party preference variable is washing out some of the significance and that misperceptions of disease risk are more pervasive. I will reexamine this trend in Chapters Five and Six.

4.3. Discussion

In the previous section, we saw that attitudes toward AIDS programs vary largely by ideology and party preference and attitudes toward homosexuality. However, as we saw in
Chapter Two, AIDS rates vary by region with the highest levels being found in some areas of the south where individuals are more likely to express moralistic attitudes. The literature notes that in the southern region of the U.S., both whites and African Americans are likely to exhibit traditional male-dominant gender roles reinforced by the influence of conservative religious attitudes (Brown 2006, Ellison & Sherkat 1995, Brown 1994). The analyses above showed some evidence of regional variations in attitudes toward social programs. However, it is more likely that these are conditioned more generally by ideology and the perception that AIDS is primarily associated with immoral behaviors.

The literature is mixed on the role of religion in shaping attitudes toward morals and sexuality. Ellison and Sherkat (1993) argue that conservative Protestants are more likely to endorse authoritarian stances on family, gender roles, and sexual conduct and norms. Their results, however, also reveal significant effects of education, income, and being Southern and rural. This echoes another study which finds that sexual risk-aversion is influenced less by religiosity than it is by healthful behaviors exhibited by the parents. Levels of parental income are also highly significant in the empirical model (Baldwin & Baldwin 1988). A similar study shows that persons with no religious orientation are more likely to perceive themselves to be at risk for infection, to be risk-averse, and more likely to practice safe sex (Prohaska, Albrecht, Levy, Sugrue & Kim 1990). The null findings with regards to religion and support for AIDS programs in this work, leave that relationship murky.

Recent research highlights the importance of religion and its interaction with “relative power” theory. The recognition among the wealthier segments of society that there are high levels of inequality within their communities fosters increased religiosity. The authors do not directly model for political ideology, but they do find that those who do not favor income redistribution are more likely to have a social dominance orientation and see religion as a means to shape the attitudes of the less well-off (Solt, Habel & Grant 2011). Other recent work (Hetherington & Weiler 2009) argues that racism and levels of income inequality are
greatest in the south where authoritarianism is high. These tendencies interact with ideology and produce an atmosphere more hostile to behaviors perceived as immoral.

Clearly, as faith-based communities and service providers are called upon to provide for those with HIV and AIDS it bears looking at the social dynamics that shape their approaches to the disease. In areas where religiously oriented social organizations operate under the auspices and purview of a more authoritarian ideology, it is to be expected that their views of service provision will be directed more toward means of controlling or containing the disease as opposed to treatment. In this environment, providers will be more likely to insist on directing funds to mandatory testing as a means of prevention. This will tendency is the focus of Chapter Six, to follow.

In the final analysis, the main effects of attitudes toward AIDS and funding are indirect and suggest a misunderstanding of the underlying causes. They reflect the fact that most people still view AIDS as a “gay” disease. Thus, support is predicated on the perception that homosexuality is always wrong or immoral. If, however, poverty and inequality are pushing disease prevalence, then the moral interpretation may be misdirecting many of our efforts. The degree to which these factors are conditioning congressional responses to funding decisions is the topic of the next chapter.
CHAPTER 5

FEDERAL AIDS FUNDING UNDER RYAN WHITE

Up to this point, we have examined the links between poverty and HIV prevalence and public attitudes toward funding ameliorative measures. Chapter Four provided evidence that perceptions of HIV risk are conditioned by ideology and attitudes toward homosexuals. While the CDC, WHO, Texas State Department of Health Services and various other national and international organizations are beginning to recognize the link between concentrated poverty and disease susceptibility, this link is not recognized by the general public. Instead, general perceptions are constrained largely by how individuals feel about the acceptability of homosexuality.

The question at hand is how these public perceptions become reified in public policy. The analysis below will show that members of Congress are also highly constrained by ideology, which has a tangible effect on their funding decisions. Further, funding considerations are hampered by competition for funds, which makes members loath to accept reductions in funding even though their districts may have experienced decreased HIV prevalence which would seem to necessitate shunting money away from that district to a higher-need area. We are left then with a funding system unreflective of the changing face of AIDS, impervious to change, and characterized by a highly competitive fight for funds.

To make matters worse, funds are layered through multiple levels of state and local bureaucracies via the formula grant. This allows state and municipal authorities much of the authority to decide how funds will be administered, thereby introducing another level of ideological funding decisions. CDC and the Health Resources and Services Administration (HRSA) internal policies forbid them from issuing any sort of directive to local governments as
to how HIV prevention funds can be used and there is no single CDC validation requirement on the prevalence numbers being presented by states and localities for the purposes of funding apportionment. Sub-governments thereby become embroiled in the competition for funds. This has important consequences which will be examined in this chapter and in Chapter Six.

It remains to be seen that the funding provided by government grants fails to address the real problem of disproportionate infection rates among minorities. Finally, a look at the 2009 federal outlays by state will show that the funding gaps are likely to be even wider under the current award formula. The new “Minority AIDS Initiative Provisions” of Ryan White Parts A and B are unlikely to be effective as they fail to address the underlying inequalities which drive the epidemic and continue to fragment care through multiple providers at various levels of government. The data from GAO will show why these initiatives are not likely to improve infection rates among the most susceptible.

5.1. The Ryan White Act

As mentioned in Chapter One, when the AIDS epidemic started in the early 1980s, the governmental response was slow. Early responses involved a fair amount of political posturing. The first budget proposals from the Reagan administration recommended a zero dollar budget increase to the Centers for Disease Control to fight the burgeoning epidemic (GAO 2006). Congress, however, did push back and ultimately passed a new budget which gave more money to both the CDC and the National Institutes of Health. Yet, it was not until 1990 that Congress passed a comprehensive AIDS program, The Ryan White CARE Act. Public awareness was raised when Ryan White, a hemophiliac, died of AIDS-related illness after, it is believed, that he was infected with HIV by a tainted blood transfusion. Since it was passed in 1990, the Ryan White Act provides funding primarily to state and local programs and over

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1Personal communication, CDC, June 7, 2011.
2,000 charity organizations to provide treatment to those living with HIV or AIDS. Specific provisions of the Act are discussed below.

However, the program must be re-authorized every session of Congress. The budget battles over the years have become increasingly politicized, and the votes for and against funding increases divided almost exclusively along party lines (GAO 2006). The 2006 re-authorization was especially convulsive with large disagreements between the parties as to appropriate funding levels. To make matters more complicated, the contentious debates in the House Health Subcommittee over HR 6143, the 2006 re-authorization, and the subsequent bill mark-up were conducted by a committee membership with only moderate constituent interest in HIV/AIDS funding. The members were: Steve Barton, Texas 6th District; Nathan Deal, Georgia 10th; Sue Myrick, North Carolina 9th; Joe Pitts, Pennsylvania 6th; Lee Terry, Nebraska 2nd; Steve Buyer, Indiana 4th; Paul Gillmor, Ohio 5th; Charles Norwood, Georgia 9th; George Radanovich, California 14th; and Fred Upton, Michigan 6th. Only one of these members, Charles Norwood, comes from an area, Augusta, Georgia, with a moderately high HIV disease burden: 14.6 cases per 100,000 population. The rest of the subcommittee membership came from largely rural districts with low HIV prevalence (see Appendix A).

5.2. Appropriations and the Funding Formula

This chapter follows the theoretical guidance (Hacker 2004) of Chapter Two and looks at state and local AIDS service providers. Much of the current debate surrounding public policy focuses on the assessment of risk (Beck 1992). Under this auspice, and given the poor media coverage of the changing face of AIDS noted in the previous chapter, it is easy for lawmakers who oppose state policies that address a problem caused by perceived immorality to make subtle changes to undesirable services. As Hacker notes, policymakers do not need to enact major policy reforms but merely need to make incremental changes that move provisions

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2Information on specific provisions can be found at [http://hab.hrsa.gov/](http://hab.hrsa.gov/), the website for the U.S. Health and Human Services Administration.
away from targeted outputs (Hacker 2004, Thelen 2003, Adema & Einerhand 1998). This gradual “drift” produces the exact types of policy-to-risk mismatches seen in U.S. AIDS policy. These subtle policy changes began with the Reagan Administration and continued under Clinton (Skocpol 1996).

The paired problems of classism and inequality have political implications. A corollary issue is whether AIDS funds most adequately meet the need to which they are allotted. Since the process of budgeting money to government agencies is largely political, we are well-advised to look at the dynamics involved in this process. The budget process depends on information and involves numerous inputs. Lawmakers, in allocating funds, depend on advice from executive agencies, the president, and interest groups, among others. They also depend upon an information environment that accurately reflects needs. If members of Congress are unaware of the needs of the communities they represent, it is less likely that they will act in their behalf.

In an atmosphere of ethnic stratification, poor or incomplete information on the part of lawmakers, and political wrangling, policy can be constrained in important ways. As a result of the aforementioned process of marginalization, lawmakers find it easier to “scale back” or “reconfigure” social policy in ways that mask and decentralize distribution of social welfare programs (Hacker 2004). This process of “retrenchment” has been used since the Reagan Administration to gradually dismantle the welfare state (Pierson 1997). As a result, the burdens once eased by social programs have increasingly been privatized onto individuals who can least afford them, which further widens the income gap between the poor and well-off. And, it is precisely poor people of color who are most at risk for HIV infection.

The problem of representation and its interface with policy is even more pronounced when populations are bifurcated (discussed in Chapter Three). Because lawmakers will potentially listen more to the concerns of the affluent portion of their constituency, this may more acutely disadvantage marginalized sub-groups. The affluent group is more likely to possess political
resources, access, and clout. Through this process, lawmakers may perceive that they are effectively serving their constituency interests when they are, in fact, only propagating the problems of the under-served.

While the arguments presented by Pierson (1997) and Hacker (2002) greatly advance our understanding of how policy changes are often highly politicized and, as such, fail to respond to changing circumstances, there is much more work to be done. Hacker insists that these changes are often "subterranean" and not apparent to the general public (Hacker 2004). As such, he recommends looking at individual areas of social policy since the subtle reconfigurations that occur are not uniform across all policy domains. Aggregate analysis cannot tap the ways in which efforts are targeted in specific communities. Much of the federal money allotted under Ryan White are given as grants to community outreach centers and clinics. It is likely that a high degree of variability exists in the relative success rates of these centers as a result of social dynamics and simple know-how. Using aggregate CDC prevention numbers as a proxy is very unlikely to adequately measure these important differences.

An early paper on federal funding of AIDS initiatives found that at its inception funding decisions were hampered by poor communication between scientists and the federal government (Stoto et al. 1988). Whether this is still the case is an open question. The CDC and NIH have a hand in the budget process as they send their budget requests to the OMB and subsequently to Congress every two years. The President then writes his budget based in part on executive and independent agency requests. This budget goes through the mark-up process in congressional subcommittees who recommend funding levels for the Department of Health and Human Services who is tasked with administering the CDC and NIH. So the process is circular and fed by inputs and requests, many of a contradictory nature, at various points in the process.
To make matters more complicated, Congress never legislates how federal outlays are ultimately spent by NIH, the agency which receives the bulk of money spent on AIDS initiatives; it merely allocates a lump sum (Stoto et al. 1988). In their paper, Stoto and his colleagues recommended that an advisory committee be set up to facilitate better communication between Congress, health service agencies, and biomedical researchers. Absent some systematic effort to provide feedback to Congress, it is unlikely that federal funding efforts will be nimble enough to respond to changing conditions on the ground.

Given this obvious dearth of accurate information on all sides, it is necessary to compile numbers in order to ascertain how these labyrinthine processes have impacted AIDS policy. The most obvious point of analysis is the policy output - how is the money spent at the state and local level? As such, we must look first at the budgetary outlays and then look at the ways in which having multiple budgetary processes, political wrangling, and grant layering effect the expenditure of allocated funds.

To vastly simplify matters, we can narrow our budgetary analysis down by looking at how much federal money spent by CDC and NIH specifically target prevention and treatment efforts. Throughout the 1980’s virtually all CDC attention was focused on gay men (Oppenheimer 1988). This trend began to change, however, with the passage of the Ryan White CARE Act in 1990. Title IV of the act is aimed at providing grants to health clinics and treatment centers in areas hardest hit by AIDS and at providing grants to fund care for women, infants, and children, and AIDS education training centers. The numbers and data presented below are taken from the Government Accountability Office (GAO) whose 2006 report to Congress highlighted important holes in congressional and CDC oversight. Important data are quite simply lacking. Absent any concrete CDC guidelines on HIV prevention and oversight on how these funds are ultimately spent by sub-governments, it is hard to ascertain how efficacious local-level efforts are. However, we can begin by looking at aggregate numbers.
Table 5.1. Ryan White Funding 2006-2009

<table>
<thead>
<tr>
<th>Year</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emergency Care</td>
<td>611,581</td>
<td>603,993</td>
<td>627,149</td>
<td>663,082</td>
</tr>
<tr>
<td>HIV Care</td>
<td>1,134,596</td>
<td>1,195,500</td>
<td>1,195,248</td>
<td>1,223,791</td>
</tr>
<tr>
<td>Early Intervention</td>
<td>196,054</td>
<td>193,721</td>
<td>198,754</td>
<td>201,877</td>
</tr>
<tr>
<td>Women, Infants, Children</td>
<td>72,960</td>
<td>71,794</td>
<td>73,690</td>
<td>76,845</td>
</tr>
<tr>
<td>AIDS Ed. Training Ctrs.</td>
<td>34,700</td>
<td>34,701</td>
<td>34,034</td>
<td>34,397</td>
</tr>
<tr>
<td>Total Ryan White Funding</td>
<td>2,062,713</td>
<td>2,112,795</td>
<td>2,141,792</td>
<td>2,238,412</td>
</tr>
</tbody>
</table>

All numbers are in thousands of dollars

Table 1 shows federal budget outlays to the various programs funded under the Ryan White Act from 2006 through 2009. The most apparent trend is that funding for long-term HIV care which funds clinics and provides access to anti-retroviral therapy has increased while AIDS education training has decreased slightly. Similarly, care for women, infants, and children has remained relatively flat. This despite the fact, mentioned above, that black women are infected at a rate some 20 times that of white women. Of course, it is difficult to tell from these data how many of the grants specifically target minority communities. In order to disentangle this, a typology is required that exceeds the scope and aims of this study. However, Table 2 provides a basic typology of the various types of programs funded under Ryan White.

Title I of the Act contains the parts A and B provisions most important to the current discussion. Part A provides emergency grants to metropolitan areas hardest hit by the epidemic. To qualify an area must have more than 50,000 total population and over 2,000 AIDS cases reported over the previous five year period. Part B provides across-the-board supplemental grants to all fifty states. The Minority AIDS Initiative, discussed below, was later added as a
Table 5.2. 2008 Federal HIV/AIDS Appropriation

<table>
<thead>
<tr>
<th>Program</th>
<th>Appropriation</th>
<th>Change</th>
<th>President’s Request</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ryan White/CARE Total</td>
<td>2.113 billion</td>
<td>+75.8 million</td>
<td>+21 million</td>
</tr>
<tr>
<td>Title I</td>
<td>604 million</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Title II: Care</td>
<td>406 million</td>
<td>+75.8 million</td>
<td>-5.02 million</td>
</tr>
<tr>
<td>Title II: ADAP</td>
<td>789.5 million</td>
<td>0</td>
<td>+25.4 million</td>
</tr>
<tr>
<td>Title III</td>
<td>193.7 million</td>
<td>0</td>
<td>+6.32 million</td>
</tr>
<tr>
<td>Title IV</td>
<td>71.8 million</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Part E: AETCs</td>
<td>34.7 million</td>
<td>0</td>
<td>-6 million</td>
</tr>
<tr>
<td>Part F: Dental</td>
<td>13.1 million</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>HRSA Community Health Centers</td>
<td>1.988 billion</td>
<td>+206.9 million</td>
<td>0</td>
</tr>
<tr>
<td>HRSA: Title X</td>
<td>283 million</td>
<td>-.04 million</td>
<td>0</td>
</tr>
<tr>
<td>HIV Prevention and Surveillance</td>
<td>698.1 million</td>
<td>+46.4 million</td>
<td>+47 million</td>
</tr>
</tbody>
</table>

supplement to Parts A and B to deal with the recognized prevalence increases in communities of color in major metro areas.

Of Title II funding, about 71% are provided for ADAP and non-ADAP medications. ADAP is the acronym for the AIDS Drug Assistance Program. This program provides eligible recipients with anti-retroviral medications meant to delay the onset of full blown AIDS, as well as non-ADAP medicines like antifungals and antibiotics to control secondary infections or co-morbidities. The ADAP programs contained in the various programs all together constitute about one-third of all AIDS program funding. The remainder of programs provide outpatient, palliative care, and even housing assistance for AIDS patients. Part E includes AIDS Education and Training Centers (AETCs) and a number of smaller programs like the dental supplement aimed at improving overall health outcomes.
These trends in funding beg the question of representation detailed above. The Ryan White Act, like much legislation passed by Congress, is not perpetual but must be renewed periodically. The last such effort occurred in 2006 with the passage of HR 6143, the Ryan White Reauthorization Act. The budget numbers listed in Table 2 are the result of congressional and presidential bargaining over budget requests by the CDC and NIH as detailed above. The bill was marked up in the House by the Energy and Commerce Committee, Subcommittee on Health. It moved from the subcommittee to the floor in one day without hearings. Of the bill’s sponsors, none were African-American, and only one (Charles Norwood, R, GA-10) represented a district with an urban area hard hit by AIDS: Augusta, Georgia at 14.6 cases per capita.

Of particular note, the area of Ryan White funding which received the largest funding increase is prevention and surveillance. This is the policy area aimed at funding HIV tests in public clinics and health provider settings. Recall from Chapter Four that more draconian measures are likely to be endorsed by those favoring an “authoritarian” approach to the epidemic (Hetherington & Weiler 2009). We will take this matter up again below and return to it as the focus of Chapter Six.

5.3. The 2006 GAO Report and Ryan White Reauthorization

During the Ryan White reauthorization, a community group called AIDS Alliance actively attempted to lobby Congress on the part of women, children, and minorities. They wrote a policy paper decrying the fact that Title IV funding aimed at these under-represented during the 1990’s and 2000’s decreased or remained flat. The paper concludes that AIDS prevention efforts “have never been adequate” to meet the needs of women and low-income Americans.

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As a result of efforts like these, in 2005, Congress tasked the Government Accountability Office (GAO) to compile a report assessing the efficiency of federal funding for AIDS initiatives under the Ryan White/HOPWA Act, the primary vehicle of national funding efforts. Despite the report’s findings that the formula grant was malapportioned, the 2005 reauthorization continued most funding at or lower than 2004 levels. The last chapter detailed how public opinion reflects misapprehension of the changing face of AIDS in the U.S. In terms of policy, the same misunderstanding of factors affecting AIDS prevalence may be present among members of Congress. An in-depth look at the reauthorization reveals consistencies in congressional voting.

When GAO issued its report in 2006, it recommended changes in the funding formula and a more competitive application for emergency funds based on severe need. They argued that CARE and HOPWA funds were not being meted out based on the current distribution of the disease. Further, changes enacted in 2004 left funding malapportioned because “hold harmless” provisions of the previous authorization kept grantees from losing funding if their case load drops (GAO 2006, 16). Resistance to changing the funding formula on the part of lawmakers resulted not in a more competitive application process, but in a compromise which added yet another level of funding to the mix. Rather than change existing programs, Congress voted to implement the Minority AIDS Initiative (MAI) to supplement Ryan White and provide an additional $6.7 million in new grants to states and $42 million in new grants to major metropolitan areas. Yet, as with Ryan White, the new MAI came without clearly defined goals and specifications from the CDC as to how the program should be enacted.

Consequently, despite the new initiative, subsequent GAO reports conclude that both programs, the original Ryan White Act as well as the Minority AIDS Initiative, are not fully proportional to the number of persons living with AIDS (GAO 2009). The GAO recommends revising the funding formula counts to numbers of living AIDS cases and not including HIV counts, or numbers of deceased cases in the counts. As the 2009 GAO report highlights,
there is intense disagreement between members of Congress as to how to count cases in order to apportion funding. This is further complicated, as described below, by the fact that CDC has no single, set validation requirement for case reporting from states and localities.

The roots of these disparities are arguably ideological in nature and represent partisan wrangling on the part of legislators. The committee report accompanying the 2006 reauthorization of the Ryan White Act (HR 6143) reveals that while members of the Health Subcommittee of the Committee on Energy and Commerce were very aware of the disproportionate toll of AIDS on communities of color, the Committee of the Whole voted almost exclusively along party lines to strike proposed changes to the funding formula. In an effort to overcome resistance to altering grants, Frank Pallone (D-NJ) offered an amendment to the reauthorization bill that would have increased overall Parts A and B funding for the years 2007-2011, effectively enlarging grant eligibility to affected communities. This provision was struck down by the Committee of the Whole in a very partisan vote. All Democrats voted for the provision and all but two Republicans voted against it.

A second proposed amendment would have provided for a one-year $30 million increase for states with “unmet need” or non-eligibility for Part A funding. This provision was similarly struck down in a partisan manner. All Democrats voted ‘Yea’, but all but one Republican voted ‘Nay’. The final bill also left in place the five-year “hold-harmless” mechanism which precluded any alteration of funding amount if prevalence rates in a geographic locale decreased. These types of compromises highlight the subterranean political processes at work in the appropriation system.

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5.4. Problems with Ryan White and MAI

There are two important consequences of note to emerge from the latest round of funding. The first is that there are lingering problems of community access, or bars to participation in community-based programs experienced specifically by persons in high-poverty areas. These barriers leave a large segment of the most vulnerable populations untreated despite the new initiatives. The second, and perhaps more serious, problem stems from the fact that there is very little client-based feedback coming from communities as to the efficacy of these programs. This is paired to the growing awareness that CDC validation requirements are imprecise and not nationally standardized. This can lead to not only a lack of efficacy information but also poor reporting of accurate HIV prevalence numbers within communities.

In their dissenting views statement added to HR 6143, Frank Pallone, Eliot Engel, Harry Waxman, et al, note that flat funding of Ryan White would unduly disadvantage states with the highest prevalence. They note that over one-half of all persons living with HIV/AIDS reside in just five states: New York, New Jersey, California, Texas, and Florida. Not increasing Parts A and B funding as requested under the Pallone Amendment would result in large cuts in areas with largest unmet needs in ways that the hold-harmless provisions would be unable to prevent. Simply stated, without at least a 3.7% overall increase in funding would result in redistribution away from hard-hit areas. Ultimately, if the pie stays the same size, but more individuals are claiming a share, the individual share becomes smaller.

Interestingly, two years later, the GAO still reports that Ryan White and MAI are seriously malapportioned resulting in barriers to minority access. The newest GAO report on the Minority AIDS Initiative reports that the three major barriers to care are poverty, housing issues, and “co-morbidities” such as mental illness and hypertension that keep those who would be eligible for Parts A and B grants from obtaining care. Simply stated, these inherent conditions can “prevent minorities with HIV/AIDS from accessing healthcare and adhering
to complex HIV/AIDS treatments because they must attend to more immediate needs, such as obtaining food and shelter” (GAO 2009, 25). Out of the 56 grantees of the MAI, 8 out of 10 reported lingering problems of access; 54 and 55 of the grantees respectively reported that poverty and housing issues are major barriers to care.

The second source of dissent to the ultimately reported version of HR 6143 concerns HIV reporting standards among states and localities. Recall from the discussion above that GAO recommended using AIDS counts and not HIV and AIDS counts together for determining formula eligibility. There is a basic rationale for this standard: counting both parameters introduces the serious possibility of double counting noted by GAO in their 2006 report. The Pallone dissent in particular highlighted the fact that there is no way with the current states’ HIV surveillance programs, which vary widely, to verify that reporting is accurately tracking the epidemic.

The GAO echoes Pallone’s contention. Their 2009 report notes that while several states use confidential name-based reporting, several more use a complicated code-based system which can contain duplication rates in excess of five percent. A national code-based validation system is being generated which will have rigorous standards intended to eliminate double-counts and over-reporting. Such a system, however, is as much as ten years from being fully implemented.

5.5. Conclusion

Ultimately, Ryan White and the MAI do not adequately meet the needs of the most vulnerable because they do not deal with the underlying issues. In Chapter Three, we saw the links between poverty and HIV prevalence in the Texas data. And we know that Texas is in the top five among states in both poverty rates and HIV rates. Federal programs are intended to fill this void and compensate for needs that are not being met by state insurance
and/or Medicaid-type programs. This being the case, it is important that those most at risk for HIV and AIDS are also most likely to be left behind by ameliorative programs.

We noted above that the largest program increases under Ryan White after 2006 were for testing and surveillance despite the fact that there are no uniform CDC surveillance standards. Similarly, grants to community health clinics, already funded at two billion dollars per annum jumped four million dollars over previous levels. These community health centers are often private providers, over-stretched in terms of resources, and struggling to meet demand. There may be a tendency among these clinics to misreport or duplicate cases, especially if their government funding levels are dependent on case-load.

The CDC also notes, in addition to a lack of national reporting standards, that there is a fundamental mismatch between HIV and AIDS prevalence rates between various regions of the U.S. It is actually the case that HIV and AIDS rates are relatively uncorrelated. This has caused concern among CDC officials about HIV testing in public clinics. This will be the topic of the next chapter; in it we will see that the correlations between HIV and AIDS rates are highest among those living in poverty. This contention will bring the need to overhaul AIDS program provision into sharper relief.
Chapter Three detailed the link between urban poverty and HIV prevalence. Subsequently, we explored the ways in which public opinion and policy are conditioned by partisanship and attitudes toward homosexuality. However, we have not shown that having a policy focused on risk and blame is causing a shift away from those who are actually sick and creating gaps in prevalence between those who test positive for HIV and those who are actually developing full-blown AIDS. As this chapter will show, there are huge disparities between HIV and AIDS that are being explored in the literature and are a current topic of debate at the CDC. We will expand this conversation with another look at the Texas data and extending the analysis to broader U.S. urban data.

Extending the Texas study to American Communities, with a broader measure of Census variables, will show the interaction between place and access. The 2007 American Community Survey, published by the U.S. Census Bureau, contains data directly relating to the question at hand. The data show, as we saw in Chapter Three, that across the United States, African Americans have median household income levels $6,000 below Hispanics and over $16,000 less than whites. Further, the highest levels of income inequality are found in the deep south, New York, and California (USCB 2008). These areas correspond to the highest levels of HIV prevalence, as we have seen in previous chapters.

We should expect to see these disparities reflected in terms of access to health care and healthful living conditions. The aforementioned literature details the ties between poverty and HIV prevalence. In addition, where there are large numbers of concentrated poor, the prospects of gaining access to medical care and information can be limited (WHO 2010).
This tendency can be even greater for persons living with HIV/AIDS. Sack (2010) explains that financial crises like those experienced after 2008 can cause states to close enrollment for federal programs that provide drugs and clinical care for AIDS patients. This directly impacts those populations most vulnerable to infection.

In the previous analysis, we discussed the socioeconomic factors driving infection rates in Texas. The findings indicate that it is not merely race, but the nexus of race, poverty, and urban contexts which are related to disproportionate infection rates among minority groups. Next, we turn to broader measures of health inequities within large urban areas based on CDC data. The model above measured urban socioeconomic indicators against the same indicators in suburban and rural settings. The model presented here focuses on the dynamics within urban and major metropolitan areas. A different set of metrics is employed which will show the combined effects of both SES variables and health care access on HIV rates for 117 major metropolitan areas.

The comprehensive focus of this chapter is establishing the fact that HIV and AIDS numbers must be disaggregated in order to be meaningful. Many scholars (Bauer 2007, Culshaw 2007) note the poor correlations between HIV and AIDS rates. This poor correspondence has not gone unnoticed by the CDC. It has become customary to refer to individual cases as “PLWHA,” meaning Persons Living with HIV and AIDS, lumping them together as if they are all the same thing. However, as noted in the case of Texas in Chapter Three, treating HIV and AIDS as the same phenomenon hides important information that can be gleaned by looking at these cases separately. As we will see in the course of the following sets of statistics, we can make more sense of the data by looking at the rates of AIDS progression separately as a function of poverty.

This chapter concurs with an assessment of a CDC official, described below, that discrepancies between the rates of HIV and seroconversion to AIDS are likely an artifact of increased
testing. This is a consequence of having a policy based upon risk and blame and a prevention program largely focused on increased testing as a means for controlling infection rates. However, Chapters Four and Five showed, decisions about how best to control the epidemic are still based on mischaracterizations of disease risk based on stigmatization of gays. Thus, the dependence on increased testing may be leading to false positives while simultaneously masking the possibility that what may actually be killing the victims of AIDS are diseases more closely linked to poverty.

6.1. HIV and Poverty

Chapter Two detailed the findings of the Institute on Assets and Social Policy on income equality in the U.S. The Census Bureau’s American Community Survey in 2007 came to similar conclusions. According to this study, nationwide median income for whites is $50,740, for Hispanics $40,766, and for African Americans $34,001. Further, as a percentage living in poverty, minority groups bear a disproportionate burden. In 2007, only 13 percent of whites are living below the poverty line as opposed to 20.7 percent of Hispanics and 24.7 percent of blacks. Nationwide income estimates reveal that African Americans are almost twice as likely to be poor (USCB 2008). ¹

These disparities have important implications in terms of HIV prevalence. In 2010 the CDC completed a major study of over 9,000 individuals not considered to be at high risk of HIV infection. Their results conclude that being urban and poor double one’s chance of contracting the virus. Among the findings, CDC notes that among the infected persons included in the study 55% were female and 75% live below the poverty line (CDC 2010c). The study authors offer that socioeconomic deprivation lowers access to health care and testing, thereby increasing prevalence. Despite the fact that CDC is touting this study as a

¹The poverty estimates are based on a new matrix which compensates for state and local differences in cost of living and housing. The report on geographic differences in poverty thresholds is available at: www.census.gov/hhes/www/povmeas/topicpg3.html.
first step in identifying a solid link between poverty and AIDS, no attempt is made to identify community factors that tie these phenomena together.

In the previous chapter, we saw important differences in urban versus rural contexts in Texas. The results for urban indicators were not as strong as those for raw household income and income inequality. Some research indicates that, while poverty is important for urban contexts, income disparity has also moved into the suburbs (Murphy & Wallace 2010). Given this situation, it is more useful to measure neighborhood access to social goods in terms of “organizational deprivation,” expressed as services oriented toward the poor. Rothwell and Massey (2010) find similar patterns. Importantly, their results show that in metro areas with higher levels of income segregation, racial differences in access are exacerbated. Additionally, these two studies may explain why HIV/AIDS has moved to the suburbs at a rate that mirrors the movement of poverty to the cities since the 1980s.

A recent study from the Centers for Disease Control (CDC) indicates that risk of genital herpes infection is much higher for African Americans (CDC 2010a) and that these same risk groups are two to three times more likely to contract HIV. A similar report found that rates of sexually transmitted diseases are higher across the board for African Americans (CDC 2010b). This summary of current research notes that blacks, especially women, account for almost half of all chlamydia and syphilis cases and more than 70 percent of gonorrhea infections. Among possible reasons for these disparities, the report lists stigma and misinformation about STD transmission, discomfort among doctors in talking to their patients about sex, racial discrimination, poverty, and poor access to health care among minorities. It is also worth noting that much of the burden of HIV/AIDS in this country also falls disproportionately upon women and infants vertically infected in utero.

As Boler and Archer point out, since 2000, the federal funding for abstinence-only sex education has risen precipitously. In 1997, funding for these types of initiatives was around $9 million, but had increased to over $176 million by 2007 (Boler & Archer 2008). Results of
these educational changes, however, have been ambiguous with students receiving conflicting information about the efficacy of condoms and susceptibility to sexually transmitted diseases. There is conflicting evidence that sexually transmitted diseases actually go up in areas with abstinence-only educational programs.

Yet, while these studies are helpful in defining the scope of the problem, they do not answer the question of why African Americans in particular are more susceptible to many of these diseases and poor outcomes. As Patton notes (1990), and this study highlights in Chapter One, there is evidence that HIV testing actually increases rather than decreases HIV prevalence. Similarly, a 2003 CDC study found no evidence whatsoever that African Americans are substantially more sexually promiscuous than any other race. The same CDC study reports that it takes on average 1,000 heterosexual contacts with an infected partner to transmit the virus just once (CDC 2003). For a certain group to be more susceptible to a disease as we see among African Americans - recall that African American women are infected at 20 times the rates of Anglos (CDC 2007) - there would have to be an exceptionally high rate of promiscuity among black women or an unusual degree of susceptibility among same. The absurd alternative would be that the virus is somehow able to discriminate among races (Bauer 2007, Deusberg 1996). There must be some other reason for this disparity in HIV infection rates.

6.2. HIV and AIDS Correlations

Consistent with the analysis in the paragraph above, there are conflicting patterns apparent in the prevalence trends, especially when one compares rates of HIV and progression to AIDS. This has not escaped notice. Culshaw compared mathematical models for HIV and AIDS and realized that there is little correspondence between the two. In fact, AIDS does not obey any conventional epidemiological models like Farr’s Law for spread and prevalence of epidemics. We should see an initial spike in disease rates with a corresponding decrease,
and then a stochastic function thereafter, with rates increasing and decreasing randomly (Culshaw 2007). Instead, rates have remained surprisingly constant with HIV increasing at comparable relative rates year after year and AIDS rates flat or declining.

In fact, in between 1995 and 2005 AIDS deaths among African Americans have remained constant, even though HIV prevalence has increased.² This trend occurred despite the fact that care and services like access to antiretroviral medications is spotty among blacks, as mentioned in Chapter Five. A major project called the HIV Cost and Utilization Study, surveyed and compiled data on 26 smaller state and local HIV service providers. They find that averaged over the aggregate, African Americans are 2.16 times less likely, and Latinos 1.67 times less likely, to have access to AIDS medications due to de facto barriers to access (Kahn et al. 2002). Given this trend, it should be likely that AIDS deaths among blacks would have increased over the same period. This is, however, not the case.

Based on the data from the CDC, I have constructed this graph of U.S. HIV and AIDS prevalence. It is virtually identical to the ones presented by Bauer (2007) and Culshaw (2007), as well as that of CDC’s Brooks and Hall (Hall, Ruiguang, Rhodes, Prejean, Qian, Lee, Karon, Brookmeyer, Kaplan & McKenna 2008). From the graph, we see that while HIV rates have increased over time, AIDS deaths have leveled off and actually declined since 1995. Further, this occurred despite the fact that use of highly active antiretroviral therapy (HAART) did not become widespread until 1996, after the rate stabilization.

From the aggregate data, we can discern the following trends concerning AIDS deaths in the United States³:

**U.S. AIDS Deaths Since 1987**

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²Data from CDC, taken from 37 states and 5 dependent areas with confidential name-based reporting of HIV infection rates since at least 2005. In some cases, data have been estimated through back-calculation to compensate for late-testing and reporting delays or incomplete reporting.

³Source: (CDC 2009; CDC 2010)
Figure 6.1. U.S. HIV Rate Estimates and AIDS Deaths


- Fell to 31,130 in 1996 and again to 16,685 in 1997.

- Have stayed steady at 16,500 to 18,000 since 1998.

Returning to the Texas data yields some interesting findings. The same data which were modeled in Chapter Three for socioeconomic variables, can be compared to AIDS rates in an identical hierarchical linear model. The sample size is the same and includes all Texas zip codes, nested within counties, for which we have a full set of AIDS rate and SES indicators.
The following table (Table 1) presents a side-by-side of HIV rates and AIDS rates conditioned on the same variables. For comparison, all independent variables are completely identical.

<table>
<thead>
<tr>
<th>Variable</th>
<th>HIV Rates</th>
<th>AIDS Rates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent Urban</td>
<td>.748**</td>
<td>.061</td>
</tr>
<tr>
<td></td>
<td>(.301)</td>
<td>(.160)</td>
</tr>
<tr>
<td>Household Median Income</td>
<td>.005***</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>(.001)</td>
<td>(.000)</td>
</tr>
<tr>
<td>Percent African American</td>
<td>5.147***</td>
<td>2.744***</td>
</tr>
<tr>
<td></td>
<td>(.758)</td>
<td>(.396)</td>
</tr>
<tr>
<td>Percent Latino</td>
<td>-.864</td>
<td>-.077</td>
</tr>
<tr>
<td></td>
<td>(.635)</td>
<td>(.350)</td>
</tr>
<tr>
<td>Percent High School or Less</td>
<td>11.705***</td>
<td>1.937*</td>
</tr>
<tr>
<td></td>
<td>(2.222)</td>
<td>(1.159)</td>
</tr>
<tr>
<td>Percent Unemployment</td>
<td>-.213</td>
<td>.448</td>
</tr>
<tr>
<td></td>
<td>(1.022)</td>
<td>(.551)</td>
</tr>
<tr>
<td>Constant</td>
<td>-363.834***</td>
<td>31.769</td>
</tr>
<tr>
<td></td>
<td>(58.800)</td>
<td>(30.635)</td>
</tr>
</tbody>
</table>

Significance levels: * p<.10, ** p<.05, *** p<.001

N = 1257; Number of groups = 222; Avg. per group = 5.7

In Chapter Three, we were concerned with HIV rates only. When we display the numbers of HIV conditioned on SES indicators next to the AIDS rates, most of the significant correlations fall away. From the rate comparison, the urban and income variables fall to insignificance. The household income variable comes close to conventional levels of significance (p<.175).
The percentage African American variable is the only one with a comparable effect between the two models.

This measure is admittedly crude. The time from HIV is simply the date of diagnosis, so late-testers—persons who carry the virus for some time before diagnosis—are present in the sample. However, with 1257 zip codes, we can expect variation among observations to be random. The means per zip code are based upon, in some instances, hundreds of individual cases, so there are tens of thousands of total cases represented in the sample. The independent variables are expressed in the same manner as the above HLM models. Based on the findings above, African Americans are isolated in the analysis to measure the effect of deprivation on access to health services post-infection and how these influence disease progression.

It remains to see how disadvantage relates to the progression from HIV to full-blown AIDS. Recalling the report from Chapter Two that funding to treat persons living with HIV/AIDS is affected by prevailing economic circumstances (Sack 2010), we should expect to see that in areas with high relative poverty, survival rates will be decreased. This is due to the fact that the poor must rely on federal and state assistance in order to obtain antiretroviral medications that prolong life. Yet, in adverse financial situations, waiting lists for this type of assistance are seen to increase greatly.

The data on progression from HIV to AIDS by zip code are available from the TSDSH. Table 4 presents the results of a Cox proportional hazards model. This non-parameterized method of gauging event histories regresses the independent variables as a function of the time it takes to reach a “failure” event as the dependent variable (Box-Steffensmeier & Jones 1997). The failure parameter, is the mean time, per zip code, it takes for a patients to progress from an initial positive HIV test to the onset of active AIDS infection as defined by the presence of an opportunistic infection (Fan, Connor & Villarreal 1991). As the dependent
variable, this measure operationalizes time to the failure events, HIV to AIDS and AIDS to death respectively, in weeks.

Table 6.2. Cox Model of HIV to AIDS Progression

<table>
<thead>
<tr>
<th>Variable</th>
<th>HIV to AIDS</th>
<th>AIDS to Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Household Median Income</td>
<td>-.000</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>(.000)</td>
<td>(.000)</td>
</tr>
<tr>
<td>Percent African American</td>
<td>.002</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>(.005)</td>
<td>(.005)</td>
</tr>
<tr>
<td>Percent Latino</td>
<td>.002**</td>
<td>.024**</td>
</tr>
<tr>
<td></td>
<td>(.009)</td>
<td>(.010)</td>
</tr>
<tr>
<td>Percent High School or Less</td>
<td>-.040***</td>
<td>-.023*</td>
</tr>
<tr>
<td></td>
<td>(.012)</td>
<td>(.012)</td>
</tr>
</tbody>
</table>

Significance levels: * p<.10, ** p<.05, *** p<.001

N = 211

The results reveal many of the same hazard variations as the HLM models in Tables 2 and 3. Urbanization, low income, living in a largely African American community, and having low education all decrease the time from HIV diagnosis to AIDS onset. Living as an economically depressed minority within a more prosperous base culture also seriously decreases survival time. Social dispersion, however, as in the HLM models, while negative is not found to be a significant predictor. This lends further support to the contention that economic factors are the more dominant drivers. Intuitively, persons with HIV/AIDS in vulnerable contexts have less access to life-extending services, despite the presence of public health clinics in inner city areas.

Looking at the border counties in relation to each other and to the rates of infection in Mexico raises some serious questions. Why would two contiguous counties with similar
immigrant populations and demographic characteristics have vastly different prevalence rates? Further, why do counties bordering Mexico on the South have such a vastly different infection rates than Mexico, the country of origin for most of the immigrants? Does the key lie in HIV testing rates between counties and between the U.S. and Mexico? Mexico has a very low HIV rate. So, we are led by theory to conclude that immigrants from Mexico come here and, within a generation, utterly change their behaviors and contract HIV. And we must conclude that HIV rates are confined to certain borders. This certainly strains credibility.

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4UNAIDS lists the rate for Mexico at less than 0.3 while the comparable U.S. rate is more than double that, at 0.7.
We can look at this a different way. The next figure is a Kaplan Meier survival graph of disease progression. The four lines on the graph are income broken down into quartiles. The survival estimates reveal that there is little absolute difference between income levels and progression to AIDS, despite the fact that the poor are more frequently infected with HIV. Table 3 shows that the hazard ratios are actually smallest among those with the lowest income levels. The hazard rates are comparable but increase slightly as personal and household wealth increases. This is certainly puzzling in light of the fact that 26 separate studies found that the poor are far less likely to have access to HIV medications (Kahn et al. 2002) and that programs to provide these drugs are frequently ravaged by the economy (Sack 2010).

Table 6.3. Hazard Ratios: HIV to AIDS Progression

<table>
<thead>
<tr>
<th>Income Level</th>
<th>Hazard Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Low Income</td>
<td>.0307282</td>
</tr>
<tr>
<td>2 Moderate Income</td>
<td>.0378092</td>
</tr>
<tr>
<td>3 High Income</td>
<td>.0321955</td>
</tr>
<tr>
<td>4 Household Income</td>
<td>.0413076</td>
</tr>
</tbody>
</table>

Texas statistics reveal a serious disjunction in the correlations between HIV and AIDS rates. Much of the problem comes from the fact that the survival estimates can only capture those who have progressed to the “failure” parameter. Looking at the raw data gives some insight into the exact scope of this problem. The data set from Texas State Department of Health Services contains 32,786 observations, defined as individuals who have been diagnosed with HIV between 1999 and 2008. Of these, only 46.48% have progressed to active AIDS and only 12.06% have died. The correlations for over half of the cases cannot be assessed if we only define progression by the failure event. A table of the pairwise correlations between HIV and AIDS rates for each of the 1257 zip codes in the Texas data highlights the demographic disparities that make the correlations difficult.
Table 6.4. HIV and AIDS Rate Correlations, by Demographic Category

<table>
<thead>
<tr>
<th>Percent Rural &gt;80</th>
<th>Percent Urban &gt;80</th>
</tr>
</thead>
<tbody>
<tr>
<td>.1457</td>
<td>.7995</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Percent Anglo &lt;80</th>
<th>Percent Anglo &gt;80</th>
</tr>
</thead>
<tbody>
<tr>
<td>.9060</td>
<td>.0151</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Percent Poverty &lt;10</th>
<th>Percent Poverty &gt;10</th>
</tr>
</thead>
<tbody>
<tr>
<td>.3548</td>
<td>.8933</td>
</tr>
</tbody>
</table>

Considering all of the cases yields an impressive finding. Living in an urban environment means that an individual is more than five times more likely to progress to full-blown AIDS. Living in poverty makes one 2.17 times more likely to progress from latent infection to disease. This fact highlights the central contention upon which this entire dissertation is predicated. Something about being in an urban poverty circumstance makes individuals much more likely to develop the illness. This suggests indirectly that poverty is the actual force behind the development of AIDS.

6.3. Why are African Americans More Susceptible?

This type of study has obvious limitations. In order to obtain the data, I had to agree not to use the individual-level data for purposes of maintaining patient confidentiality. For this reason, the units of analysis are aggregated up. However, aggregation can show us something about how the social dynamics of the larger community interact to affect individuals. One of the puzzling results to come out of these studies is why being an African Americans is so much more likely to show up as primary risk factor in all cases.

The simple answer is: we just don’t know why. The percent Anglo indicator in Table 4 highlights the huge race gaps in disease progression. In communities where minorities make up more than 20% of the total population, the chance of developing AIDS symptoms is 60
times higher. This is a staggering result. Is there something about being black, perhaps some genetic predisposition, which is driving this race disparity? or is it driven more by income disparity? We do know from previous research that here are large race gaps in HIV prevalence among intravenous drug users. Rates of infection are found to be highest among African Americans and Hispanics (Hahn, Onorato, Jones & Dougherty 1989). What is more, Hahn et al find that blacks are 5 to 7 times more likely to develop AIDS than are whites in a matched male cohort of drug users.

Why is this the case? We are unlikely to find the answers to these questions as long as we continue to view AIDS primarily in terms of sexual risk-taking behavior. There is some rationale for looking at this differently. As Bauer notes, being HIV positive can be seen as a “non-specific” sign of some more general health challenge (Bauer 2007). Living in densely populated areas places one at risk for an even greater variety of health threats, including infectious disease and even less obvious environmental hazards like exposure to toxic air (Borden et al. 2007). The results from Chapter Three provide some support for this contention. One of the most consistent indicators of HIV prevalence is urban minority status, especially in high-poverty contexts. The place and social vulnerability arguments of Oppong and Harold (2010) can provide insight. However, the question of how vulnerability translates to poor health outcomes by some biological mechanism remains unanswered. This question will occupy much of the following chapter.

What we can say for sure at this point is that there appear to be major health gaps in this country that unfairly disadvantage the black population. We can highlight these gaps in various ways. The analyses below look at poverty and access at state and major metropolitan areas. The data are from U.S. Census Bureau’s 2007 poverty and health insurance estimates and from the National HIV Prevention Inventory compiled by the Kaiser Family Foundation in 2009. Since funding for AIDS preventative and treatment services are administered at
multiple levels, we will look briefly at the state and local community programs. Relative gender differences are also important in assessing the disease’s disproportionate effect. Gender “underpins” much of the inequality present in both developed and underdeveloped countries (Dowsett 2003, 22). Women are more likely to bear the brunt of unequal conditions since women are primarily responsible for providing for their children, and therefore more likely to suffer poverty in single parent situations.

Table 5 shows some of the curious correlations at the state level. As several scholars point out (Patton 1990, Culshaw 2007, Bauer 2007) state level funding and test percent do not make HIV and AIDS rates go down. Based on the pairwise Pearson correlations, we cannot say that funding is having any tangible positive effect. All the results can tell us is that rates are very well matched to all four major indicators. Whether or not funding actually works is still an open question. However, it is apparent that rates are strongly associated with the poverty indicators. State GINI ratings, which we recall are measures of income inequality, are also seen to significantly influence rates. Furthermore, bolstering various arguments (Deusberg 1996, Culshaw 2007), media campaigns and needle exchange programs have a negligible 

\textit{positive} effect. There are no negative, or inverse, correlations among any of the Pearson coefficients reported.

At the local level, in 117 major metropolitan areas, all classified as heavily impacted areas by the CDC, we see the same sorts of correlations. Table 6 shows that rates per capita increase with testing, poverty and inequality based on pairwise Pearson coefficients. With these correlations, we also see that in major metro areas funding per capita is positively and significantly associated with high rates. It is not clear that what we are doing is having any tangible effect, and may actually be exacerbating the problem.

Why would testing seem to be associated with increased rates? Some evidence can be found from both the CDC and some of the literature. A recent presentation by John Brooks, M.D. of the CDC, asks if the prevalence of HIV may not be an artifact of increased testing.
We have seen that increased testing at several levels is correlated with HIV rates. This problem can arise from the fact, highlighted in Chapter Five, that there are no solid national verification standards to ensure that the reported numbers are accurate. However, there is much evidence (Deusberg 1996, Culshaw 2007, Bauer 2007, Epstein 1996) that HIV tests are notoriously inaccurate. In fact, the FDA warning on the box for the ELISA test, the primary test for HIV antibodies, states that the kit should not be used as a confirmatory test for HIV (Bauer 2007).
If Bauer is correct, the number of ELISA false positives could actually be higher than the number of diagnosed cases. The argument runs as follows: according to the FDA, the ELISA test yields false positives more than 10% of the time. The antibody test is known to cross-react with various other antigens and antibodies, also with various forms of cancer, and even leprosy (Bauer 2007). Most importantly, HIV antibody tests can cross-react with tuberculosis. Since, AIDS and tuberculosis are thought to be intimately related, this could be of large consequence in assessing the effectiveness of the tests used to detect HIV. This will be addressed at greater length in the next chapter.

6.4. Conclusion

The results echo other studies which show that public health and media indicators are largely irrelevant in predicting HIV prevalence (McIntosh & Thomas 2004). None of the media and needle exchange variables are significant. Perhaps, in the final analysis, we do not know enough about how AIDS services are rendered to make a meaningful appraisal of the efficacy of prevention efforts. A study of AIDS services in New York state, largely administered with state funding by private, faith-based providers, reveals a lack of basic information about how funds are spent (Tesoriero, Parisi, Sampson, Foster, Klein & Ellemberg 2000). The authors sent a questionnaire to congregational providers which yielded such a poor response rate that a follow-up phone survey had to be utilized to obtain useable results. Even with the dearth of information, they find that the congregations only provided preventative efforts in 16.7 percent of cases, citing “resource related” reasons as well as lack of knowledge. This may be a large part of the problem: we are relying on community-based organizations, who are poor in terms of resources and know-how to provision a very fragmented prevention system.

Perhaps more disturbingly, responses to the epidemic are likely still conditioned, as we have seen by blame and stigma. This can accompany and reinforce the lack of knowledge noted above. Locally administered programs are free to focus their efforts on testing as a
means to “control” the spread of HIV while ignoring the true correlates of the disease. In the effort to diagnose, it is likely, that those who are truly ill are falling through the cracks. In the next chapter, we will explore some alternate explanations of disease prevalence, and challenge some of the assumptions about HIV and AIDS.
CONCLUSION

There is much anecdotal evidence on both sides of the HIV/AIDS debate. Gregg Bordowitz was diagnosed with AIDS in the late 1980s at the age of 23. He describes his feelings at the time of diagnosis as having received a death sentence (Bordowitz 2004). As of this writing, however, he is still alive and well and working as an art professor at the University of Chicago. He credits his long survival to a positive attitude and a continuing regimen of antiretroviral medications. He, like Earvin “Magic” Johnson, is one of countless thousands who have remained HIV-positive but healthy for over twenty and even thirty years. In fact, there is a whole sub-population of so-called “HIV elite controllers” (Yang, Al-Mozaini, Buzon & Beamon 2012) who maintain states of good health and fully functional immune systems without taking antiretroviral medications ¹.

Christine Maggiore, however, presents an interesting counter-example. In 1992, during a routine medical screening, she was diagnosed HIV-positive. After some research, she concluded that HIV was not the cause of AIDS and subsequently wrote a book, What if Everything You Thought You Knew about AIDS was Wrong?, which made her one of the more notable AIDS “dissidents” (Maggiore 2000). Even after her diagnosis, she married and had two children. In 2004, she contracted pneumocystis pneumonia, an AIDS-defining disease, and died. Two years earlier, her daughter had died of the same illness. Her husband and son, however, remain to this day healthy and HIV-negative. Notably, her husband remains healthy despite years of “latex-free sex” with his infected wife.

¹There is an elite controller national study and corresponding website which can be accessed at: www.hivcontrollers.org
Another anomaly arises when one looks at the nature of the HIV test itself. The ELISA test, the primary and standard test for HIV, detects antibodies to the virus. Presence of an antibody is typically taken as evidence that the body has mounted a successful response to a disease antigen. HIV is the only infection in which presence of an antibody is taken as a sign of illness (Deusberg 1996). Take make matters worse, the test is erratic and inaccurate, as described in Chapter Six (Bauer 2007). ELISA testing is odd in that samples are diluted “four hundred fold with a diluting agent provided by the test manufacturer” making it the only antibody test which relies on this procedure; hepatitis B tests are run on undiluted blood serum (Culshaw 2007). A medical doctor at Cornell University, Roberto Giraldo, ran ELISA tests on undiluted serum and found that 100% of the samples tested positive (Giraldo 1998).

These two anecdotal examples above point to the very confusing nature of this disease. Why do some people remain healthy despite engaging in high risk behavior? The inconsistencies inherent in the transmission and progression of HIV and AIDS have arguably provided the entre for those examining other explanatory frameworks for understanding this disease. The human immune system is a complex of interactive components which, like other biological systems, appears to depend upon balance among its constituent parts. The assumption that HIV alone is capable of crippling the immune system and inducing the complex of diseases which define advanced stages of AIDS infection has been largely abandoned by the biomedical community; see, for instance: (Dingle 1997, Shearer & Levy 1987, Des Jarlais, Friedman, Marmor & Cohen 1987). Increased susceptibility to HIV can be facilitated by non-infectious agents like drug and alcohol use, the presence of other diseases or antigens, or simply by lack of natural resistance. Additionally, researchers recognize that cofactors may be present in the progression from HIV infection to AIDS. These can be environmental, not only biological or behavioral.

Chapter Six of this study also points out many of the confusing and controversial aspects of the progression of this disease. The correlations between HIV and onset of AIDS defining
disease are highest among the poor and those living in concentrated urban environments. This finding, however, is inconvenient and easily overlooked by those who view AIDS strictly in behavioral terms. If poverty is a major contributing factor, this may explain why AIDS is so prevalent in poor areas of the developing world. Similarly, in the United States, minority populations are infected at eight times the rates of Anglos (CDC 2007). This is likely partially due to the fact that minorities are over four times more likely to live in conditions of poverty (IASP 2010). What the literature detailed above points toward is a biological mechanism driving this phenomenon. As poverty is a “social disease,” social science should not abrogate its important role in bringing about recognition of this important link.

The exists the very real possibility that HIV is not the “cause” *per se* of AIDS. The presence of elevated HIV titers in the blood may, instead, be a sign of an immune system which has gone seriously awry. The causes for this immune system dysfunction are more likely to be a product of environment if they can be observed with or without the presence of HIV. Further, HIV antibodies can be detected in a great many persons without disease onset even in persons not taking antiretroviral medications. These are very serious questions which deserve investigation. Instead of addressing these questions, the biomedical community continues to come up with more and more complicated explanations for how a mostly harmless virus is able to single-handedly cripple the immune system (Epstein 1996, Deusberg 1996).

However, it is unlikely that many of the desirable answers to these questions will be obtained so long as this disease is heavily politicized. As long as perceptions of AIDS are heavily influenced by blame, risk, and stigma, there is less broad support for examining some of the underlying questions. We are still a long way from a full understanding of this disease. Until other factors like poverty and urbanization are taken into account, we may not be able to see the larger picture. This study is an attempt to bring these other factors into the discussion. Similarly, this study is an attempt to bring integration of knowledge in an area where fragmentation has become the norm. Social science does not interface with biological
science, neither fully interact with policy, and medical practitioners and sufferers are left with only partial understanding and the potential for incomplete service.

To sum up: minorities are at risk due to the confluence of misunderstanding and practice. Minorities are more than four times more likely to live in poverty (IASP 2010). They are also much more likely to be prey to all sorts of healthcare disparities, including the extremes of obesity and malnutrition, diabetes, liver problems, drug abuse (Kreuger et al. 1990). In a political environment which does not take seriously these disparities and is likely to tie problems to risk, blame, and stigma, it is highly likely that health outcomes will be tied to behavioral ascription (Patton 1990). But are we effectively blaming the victim?

7.1. A Tie Between AIDS and Poverty?

Can unequal contexts actually lead to poor biological health? A recent series of PBS documentaries called *Unnatural Causes: Is Inequality Making Us Sick* posits this very connection, and cites Harvard epidemiologists and public health findings showing that bad health outcomes predominate among America’s poor. Similarly, new research from the social sciences (Bellatorre, Finch, Do & Bird 2011) finds that racial segregation, unequal contexts, and marginalization can lead to inflammatory responses, immune system problems, and various instances of metabolic disorders. Social, “economic or policy specific exposures,” incarceration, housing status (Milloy, Marshall, Kerr & Buxton 2012) and even mental health disorders (Nurutdinova, Crusciel, Zeringue & Scherer 2012) can lead to accelerated disease progression in HIV infection and more frequent opportunistic infections.

It remains to see what possible links may exist between poverty, malnutrition, and AIDS. The roots of this connection come from an unexpected source. While rheumatoid arthritis is in no way thought to be connected to AIDS, its source is thought to lie in an over-activity of the immune system. Controlling this immune activity entails suppressing the immune system so that it does not attack one’s own tissues. Unexpectedly, and largely overlooked in the
literature, this research might enhance our understanding of the immune system suppression inherent in AIDS infections. Unfortunately, however, when AIDS is looked at in strictly behavioral terms, the tie between poverty and poor biological health is largely ignored.

Immune reactions are controlled by cytokines, which are chemical messengers that spur the production of antibodies, signal the increased production of immune cells, and mediate a whole host of inflammatory responses during disease states (Brunton, Chabner & Knollman 2010). They include interleukins and tumor necrosis factors and are highly active during the healing stage of injury or inflammation. When over-produced, however, they can be pathogenic and injurious (Tracey & Cerami 1993). Similarly, research on the precise connections between AIDS disease and the chemical messengers which mediate cellular responses to pathogens including HIV is still in its developing stages. As late as 2001, the ties linking specific cytokine responses, cell-mediated immunity, and the actions of antiretroviral therapy remain “controversial” (Bocchino, Ledru, Debord & Gougeon 2001, 1213). In recent years, however, more research has been done on the role of cytokines in the onset of acute AIDS and the various types of diseases observed therein. So important is this vein of research that some researchers have even asked if AIDS is, in fact, a “tumor necrosis factor disease” (Matsuyama, Kobayashi & Yamamoto 1991).

One of the more puzzling aspects of HIV/AIDS is the constellation of diseases which characterize the acute phase of opportunistic infection. If it were merely the case that HIV is able to establish itself as a chronic pathogen and totally incapacitate the entire immune system, then plausibly the host would be susceptible to any generalized infection, such as flu or cold viruses. Instead, AIDS is characterized by a very distinct group of opportunistic infections, many of which were rare before the AIDS-era. Among the most common infections are: tuberculosis, candidiasis, histoplasmosis, coccidiomycosis, pneumocystis and mycobacterial pneumonia, wasting syndrome, and nervous system disorders. Viral infections including hepatitis and herpes also regularly occur, and secondary cancers such as lymphoma and Kaposi’s
sarcoma skin lesions are common. And, there is some evidence that the prevalence of certain types of infections has changed since the advent of antiretroviral therapy (Ives, Gazzard & Easterbrook 2001, Brodt, Kamps, Gute & Knupp 1997).

As early as 1991, several researchers were looking at the possibility of a direct link between cytokines and AIDS diseases. Matsuyama, Koboyashi, and Yamamoto (1991) point out that TNF-α and TNF-β both enhance HIV replication based upon observations in a number of studies (Matsuyama, Yoshiyama & Hammamoto 1989, Unanue & Allen 1987, Harada, Koyanagi & Yamamoto 1985, Matsuyama, Hammamoto & Yoshida 1988). They conclude that, while other cells like macrophages and monocytes are also targeted by HIV, it is CD4+ lymphocytes that are decimated in AIDS. And, it appears that TNF-α increases susceptibility to HIV infection in these cells, resulting in higher levels of HIV replication. They further find that different types of cytokines have differential effects on various cell lines. Cytokines appear to represent a “network” of mediators which have regulatory effects not only on cells, but on other cytokines, as well. Their up- or down-regulation can induce a feedback loop which serves to increase immune activity. For this reason, the authors argue that AIDS “represents a cytokine or TNF disease” (Matsuyama, Kobayashi & Yamamoto 1991, 1410).

A cohort analysis of persons showing symptoms of active AIDS infection, as opposed to asymptomatic HIV carriers, showed a much higher production of TNF-α in the AIDS group (Hober, Haque, Wattre & Beaucaire 1989). They note that increased levels of TNF-α and IL-1 may have the twin effects of suppressing T-cell populations and facilitating the onset of opportunistic infections. For instance, increased levels of both cytokines are seen in Kaposi’s sarcoma and in AIDS-related neural lesions. A separate study finds that certain genotypes may be predisposed to increased tuberculosis and mycobacterial risk through the overproduction of TNF-α (Stein, Zalwango, Chiunda & Millard 2007). The authors find a strong link between TB and TNF receptor genes and, similar to the aforementioned study,
find cytokine production to be associated with the active state of TB disease and not its latent phase. Interestingly, this effect is noted in both HIV- and non-HIV-infected individuals.

Other research backs up these contentions. Increased production of gp120 HIV envelope protein is noted to correlate with increased levels of TNF-α, IL-6, and IL-10 in early phases of HIV infection. Further, these cytokines seem to inhibit T-cell production under the same circumstances (Rychert, Strick, Bazner & Robinson 2010). Another study finds that increased secretion of TNF-α both inhibits cellular response and increases the replication of HIV (Munoz-Fernandez, Navarro, Gracia & Punzon 1997). These articles point toward the conclusion that it may be the cytokines themselves spurring these reactions more so than the cells.

Bocchino, Ledru, Debord, and Gougeon (2001) find that the actions of certain cells, CD64 monocytes/macrophages, are differently affected by the actions of certain cytokines. They propose that the action of antiretroviral medications in increasing T cell populations lies in their ability to modulate IL-12 and TNF-α. In this vein, the actions of antiretroviral medications used to treat AIDS are, perhaps, more comprehensible. Despite the noted success of combination antiretroviral therapy (ART) in prolonging the lives of persons living with HIV/AIDS, there is little consensus as to the exact pharmacological actions of these medications. Recent articles point out that ART therapy may play an important role in moderating the dysregulated production of interleukin-12. The immune abnormalities that characterize HIV/AIDS are complicated, and often multi-focal. However, it is the regulation of this important cytokine, by ART, that appears to balance relative levels of CD4+, CD8+, and natural killer (NK) cells in the early stages of HIV infection (Trinchieri 1998). As one report notes, “IL-12 plays a central role in host resistance in models of infection with numerous pathogens that cause opportunistic infection in HIV/AIDS” (Byrnes, Harris, Atabani & Sabundayo 2008, 1448).
However, there are notable exceptions to the efficacy of these medications. For example, ART appears to decrease the risk of, and slow the progression to, Kaposi’s sarcoma. In cases of non-Hodgkin’s lymphoma and central nervous system lymphoma, on the other hand, ART does not have the same effect, and cancer treatment is more efficacious (Pipkin, Scheer, Okeigwe & Schwartz 2011). A separate study notes that while ART can normalize many cytokines, but leaves interleukin-6 and C-reactive protein unaffected. It is these cytokines that regulate B-cell activity, so ART’s lack of effectiveness in regulating them leaves patients susceptible to lymphomas even after prolonged antiretroviral therapy (Regidor, Detels, Breen & Widney 2011).

There are also noted instances in which ART fails to modify cell counts despite the fact that viral loads have dropped (Kaufmann, Pantaleo, Sudre & Telenti 1998, Florence, Lundgren, Dreezen & Fisher 2003). In many cases, cell counts have failed to increase even though HIV has been completely suppressed. A number of potential explanations have been proffered including viral replication continuing undetected by virtue of HIV hiding in lymphoid tissue. A more compelling explanation, however, hinges on knowledge of the various actions of antiretroviral agents, their relation to types of immune dysfunction, and their underlying effect on host-related factors like genetic predisposition (Aiuti & Mezzaroma 2006). These authors point out that, in some individuals, failure to modulate dysregulated levels of TNF, IL-2, IL-7, and IL-15 may be the “crucial” event in determining whether cell levels will stabilize during treatment.

All of these separate studies raise the important question: is the arrow of causation pointing in the correct direction? The connections between HIV and AIDS are clear and unequivocal. It is not clear, however, which event is more predictive of the development of AIDS-defining disease. For example, Glienke, Esser, von Briesen, Schuster, et al (1994) suggest that viral proteins from HIV-infected cells may have an indirect effect of inducing
cytokine release from both infected and uninfected cells. While they suspect that HIV infection may increase cytokine release and did note a substantially larger amount of IL-6 and TNF-α in an in vitro culture of macrophages, the differential expression of cytokines between infected and uninfected cells “is so small that it cannot be detected” (Glienke, R., von Briesen & Schuster 1994, 196). The conflicting evidence available does not allow us to definitively state that HIV alone is the cause of the collapse of the immune system; it may be time to fully explore all of the alternatives.

A 2004 report on the incidence and virulence of *Mycobacterium kansasii* provides an illustration of the connections. The mycobacterium class of bacteria, which also includes *M. avium* and *M. intracellulare*, produces tuberculosis-like illness. It, like many other AIDS-defining fungal infections, was rare in populations prior to the AIDS-era. The prevalence of *M. kansasii* has increased in both HIV-positive and negative persons. While co-infection with HIV does increase disease burden and raise the rate of hospitalization, the study authors find that poverty is a major factor in transmission of the organism. Lower socioeconomic status and unstable housing situations are found to be linked with mycobacterial infections (Bloch, Zwerling, Pletcher & Hahn 2004). This suggests that poverty, and increased susceptibility to all types of disease may act as cofactors contributing to the prevalence of AIDS-defining disorders.

Kaposi’s sarcoma (KS), a cancerous skin lesion connected with AIDS, is another case-in-point. KS is particularly prevalent among gay and bisexual men. Like histoplasmosis, coccidiomycosis, and other AIDS-related parasitic fungal infections, it was relatively rare before the 1980’s. However, KS was not unknown before the AIDS-era and was noted in immune-suppressed individuals, particularly organ transplant patients. A study published in 1990 proposed that the increase in KS in those without HIV is “probably due to an unknown sexually transmitted agent” (Beral et al. 1990, Weiss & Jaffe 1990, 660). Other researchers, however, note the connections between cytokines and the onset of KS skin
lesions. Proliferation of Oncostatin-M, which normally moderates the effects of IL-6 and TNF, is found to be strongly associated with the presence of KS-derived cells in endothelial and smooth muscle tissue. Thus, Oncostatin may be directly involved in the pathogenesis of KS (Miles et al. 1992). KS growth in epidermal cells further appears to be mediated by the activities of IL-6 and TNF-α (Matsuyama, Kobayashi & Yamamoto 1991).

This is precisely the point. There can be little doubt that HIV and AIDS are linked. Viral load of HIV is highly predictive of disease progression from chronic HIV to acute AIDS. There is good reason, however, to question the exact nature of this relationship. A good deal of literature is devoted to the effects of cytokines on the human immune system. We have seen that TNF enhances HIV replication, and that both TNF and interleukins can cause imbalances in cell populations with and without HIV. It may be the case that the central underlying mechanism of AIDS-defining disease is a state of systemic immune imbalance that accompanies, or even precedes, susceptibility to HIV. If this is the case, then it will prove much more fruitful in the future to heed Clerici’s advice and pursue a science of cytokinomics rather than a more-or-less single minded pursuit focused on HIV.

It is worth noting that the effect of some cytokines on cells in vitro mimics the onset of AIDS. In the early days of HIV testing, since the virus itself is seldom detected, the designers of the ELISA and Western Blot kits had to define the proteins which would used as the markers which yield a positive result. They did so by stimulating cells in culture with various cytokines. As Culshaw describes it:

“...cell cultures from AIDS patients were activated using powerful chemicals called mitogens and after this activation, about thirty proteins were found in this mixture, all of which gathered at a density characteristic of retroviruses. A subset of these was specifically attributed to HIV and nothing else, and ten of these are used to define reactivity on the ELISA and Western Blot HIV antibody tests” (Culshaw 2007, 38).
Further, the HIV antibody tests are unique in that they must be diluted four hundred times to keep from yielding “false positives.” The only other antibody test which is diluted at similar levels is the rheumatoid factor (RF) test which is used to diagnose some autoimmune disorders. RF must be diluted 1:50 in order to distinguish the normal spectrum of antibodies from those that are actually implicated in the autoimmune response. However, with generalized autoimmune disorders, unlike with AIDS, it is agreed that the cytokine imbalance precedes the antibody reactivity.

In the aforementioned PBS documentary *Unnatural Causes*, several researchers note that contexts of poverty can induce the increased production of the stress hormone cortisol. People with elevated cortisol are found to have poor health in general, including immune and metabolic disorders. Elevated cortisol is also associated with increased cytokine levels in autoimmune diseases (Neidhart 1996). Clerici and his team also find that a shift in cytokines is associated with elevated cortisol and/or cortisol/anticortisol imbalances characteristic of advanced stages of HIV infection (Clerici, Trabattoni, Piconi & Fusi 1997).

Poverty certainly plays a part in the prevalence of disease, perhaps even in the progression of AIDS. The World Health Organization (WHO) is taking seriously the possibility that poverty and lack of access to health resources are cofactors in increasing AIDS susceptibility. Their 2008 report on urbanization and disease argues that rapid urban growth accompanied by social and economic inequality produce “hidden cities” (CSDH 2008, xiii). In these contexts, inhabitants are more prone to the “triple threat” of chronic diseases like diabetes or cardiovascular disease, injuries and violence, and infectious diseases like AIDS and tuberculosis. This combination creates “vulnerable places” in which people become generally more susceptible to disease.

We would be unwise in neglecting the role of poverty and malnutrition in the development of AIDS-related disease. New research is beginning to look at the links between HIV infection, diabetes mellitus, and vitamin D deficiency (Gharakhanian & Kotler 2011, Zsoffia, Guaraldi,
Shah & LoRe 2010). Other research focuses on the fact that the hypothalamus, pituitary, and adrenal glands form a sort of “axis” which integrates the functions of the neuroendocrine and immune systems (Hanson, Padyukiv, B. & Wramner 2000). Given this integration, and that TNF is produced by fat cells, both extremes of malnutrition and obesity may be contributing to the under- and overproduction of cytokines like interleukins and TNFs.

Further, are some instances of AIDS diseases in the U.S. linked to poor nutrition? Another paper finds that blood levels of lipopolysaccharide, independent of CD4+ levels and HIV viral load, is a powerful predictor of disease progression (Marchetti, Cozzi-Lepri, Merlini & Bellistri 2011). The seroconversion from HIV to AIDS onset appears to be facilitated by poor nutrition apart from the influence of HIV. If this research is confirmed, it will provide evidence that perhaps we have the arrow of causation pointed in the wrong direction. We assume that viral activity is inducing poor health outcomes, when in fact the opposite may be true: poor health may be aiding the actions of the virus.

If poverty is a major contributing factor, this may explain why AIDS is so prevalent in poor areas of the developing world. Similarly, in the United States, minority populations are infected at eight times the rates of Anglos (CDC 2007). This is likely partially due to the fact that minorities are over four times more likely to live in conditions of poverty (?). What the literature detailed above points toward is a biological mechanism driving this phenomenon. As poverty is a “social disease,” social science should not abrogate its important role in bringing about recognition of this important link.

7.2. A Summary of the Major Findings

This dissertation examined the politics of AIDS funding through two lenses. The first looked at theoretical explanations for the disparities in infection rates among minorities, finding that SES indicators better explain prevalence among African Americans, who are more likely to live in contexts of concentrated urban poverty. The second found that funding
decisions are based in large part on fundamental misunderstandings, on the part of individuals and lawmakers alike, of the socioeconomic forces driving the epidemic’s disproportionate effect on minorities. It remains, also, to look at potential ways of improving prevention and the provision of health services for persons with AIDS.

Chapter Three looked at the effect of socioeconomic status on HIV risk in Texas counties and zip codes. The findings highlighted the differences between the races in terms of susceptibility. Being African American and living in an environment of concentrated urban poverty is the single largest predictor of HIV prevalence at the two levels tested in the hierarchical linear model. Latinos, it can be inferred, are more economically and socially mobile, thereby reducing their chances of being HIV positive. The models presented in Chapter Three, however, were only preliminary and did not compare HIV risk with the prognosis for progression to symptomatic AIDS. These findings, however, bolster much of the literature positing a direct link between material disadvantage and HIV prevalence.

The look at public attitudes towards AIDS funding efforts in Chapter Four highlighted some important linkages between public opinion and support for AIDS programs. While the face of AIDS has changed, perceptions generally have not. The majority of respondents in opinion surveys tend to view AIDS as a “gay disease.” The linkage that organized interests around the gay responses to the disease was not present for the poor and minorities. Predictably, at-risk minorities lost out in the process. As a result, perceptions are still largely structured around risk, blame, and stigmatization. Characterizations of the disease are largely conditioned by ideology/partisanship and feelings about homosexuality.

How these public attitudes become cemented in policy enactments was the subject of Chapter Five. While the links are indirect, it is apparent that partisanship is very much a part of the funding decisions. With funding effectively stagnant, more political players are competing for a piece of the pie, which may disadvantage the areas with genuinely greater need. To make things worse, CDC does not have the same set validation measures for all
states and localities which may increase the possibility of over- and double-counting cases to secure funding. What is more, the competition for funding which made the addition of the supplemental Minority AIDS Initiative necessary has not solved access disparities. A full 85% of all severe need municipalities still report barriers to care as a result of poverty, housing issues, and other health problems.

Finally, Chapter Six discussed the proposition that not only is poverty increasing susceptibility to HIV, evidence shows that it has a tangible effect on health outcomes. Those living in poverty are more likely to progress to full-blown AIDS and ultimately to death. The correlation between HIV and AIDS rates is very poor, as many have noted (Bauer 2007, Culshaw 2007, Root-Bernstein 1993). However, if we disaggregate the numbers and compare the progression rates to income levels, the pairwise correlations are strikingly high. This can help explain two major phenomena: the fact that the national AIDS statistics are often very difficult to explain, and part of the reason that AIDS programs don’t seem to work as well as we would like. These phenomena are linked through the intersection of poverty.

How can we begin to remedy these disparities? The aforementioned WHO Hidden Cities report (CSDH 2008) lists several suggestions to reduce health inequities. They call for a focused effort at “narrowing the health gap” by targeting the worst-off in urban areas and a broad-based approach which levels health inequities in the entire population. The plan insists that social determinants, including gender and class inequalities, must be addressed so that all persons have access to nutritious foods, clean water, and healthful living conditions. Unfortunately, the report does not include specific recommendations for implementing these proposed changes.

The section below gives some brief suggestions for improved provision of care based on the findings from Chapters Five and Six. However, it is likely the case that HIV and AIDS rates will not come down until there is a renewed focus on social programs aimed at ameliorating poverty. This dissertation is intended to call attention to that fact. Rather than addressing
the underlying causes of the epidemic and its progression, governmental programs are aimed at treating the symptoms. Similarly, the biomedical industry is focused on producing a vaccine or magic bullet treatment rather than ways to improve living conditions and basic medical services for the millions and billions of impoverished in America and across the globe.

The problems with HIV and AIDS policy are an example of what Greenberg terms the “ethical erosion” of science. Wherever government funding is involved, the central scientific actors become “ex officio” representatives of the political forces operating behind the scenes (Greenberg 2001, 19). Add to this the fact that much biomedical research is funded by profit-motivated pharmaceutical companies. The medical science surrounding AIDS is especially vulnerable to being manipulated by money and politics since it invokes political ideology aimed at behaviors some consider to be immoral and disproportionately afflicts the already disadvantaged. Additionally, the biomedical industry would stand to make billions from a potential vaccine or treatment. This echoes Dyson’s contention that science operates largely to the detriment of the poor because “…pure scientists have become more detached from the mundane needs of humanity, and the applied scientists have become more attached to immediate profitability” (Dyson 1997, 62).

Similarly, Pisani argues that the bureaucrats and business people turned AIDS into a disease of “wickedness.” They transformed it into a “development problem, a security problem” in which the resources of the rich had to be enlisted to solve the problems of the innocent victims of the epidemic, while selectively disenfranchising those exhibiting wicked sexual behavior (Pisani 2008, 316). However, her argument falls apart if it turns out that the problem is largely a result of poverty and not one of behavior.

Some of the suggestions for stopping the AIDS epidemic, while well-intentioned, may be more dangerous. For instance, Green and Ruark (2011) argue that the recipe is not likely to be technological or monetary but based instead on modifying behaviors toward risk avoidance. As an ameliorative, they suggest getting injection drug users to switch delivery methods to
“safer” methods like snorting or smoking. This is based on a perhaps mistaken assumption that it is the sharing of needles and not the drug itself which is causing immune system damage.

More importantly, thirty years have passed with little progress having been made in stopping global AIDS. The unpredictable spatial and demographic distributions of AIDS and its ties to other types of disease have stifled attempts to forecast patterns of spread and prevalence (Palloni 1996). It may be time to take more seriously the social and economic explanations. If we are serious about addressing this problem, we should be willing to consider any approach, even if it entails abandoning the idea that HIV is the sine qua non of AIDS-defining disease, and beginning to consider the possibility that cofactors like poverty and access may be spurring the epidemic. Perhaps it is time to pursue other avenues.

Social scientists should, and have, done their parts in exploring new solutions to the AIDS crisis. It is social science that has established the connections between poverty and AIDS (Poku 2005, Stillwaggon 2006). We are moving beyond socially loaded assumptions about race and sexual orientation as determinants of risk behavior toward a new paradigm which identifies environmental factors like poverty and poor living conditions as proximate causes of disease. Much, however, remains to be done; we are a long way from showing the direct connection between environment and biological effect. The links detailed above call upon all communities of knowledge to evaluate our current state of understanding. Only by so doing can we potentially adapt to a better, more integrated approach, enabling us to provide better answers.

7.3. The 2010 Health Care Bill

Does the Health Care Bill passed in 2010 begin to address these problems? This brief diversion will explore how the provisions of the new national health care bill, most of which are scheduled to take effect in 2014, serve to ameliorate the disproportionate effect of HIV/AIDS
on minority groups. It is unclear whether specific provisions aimed at alleviating the gaps in coverage by collectivizing risk actually help those at the bottom of the socioeconomic ladder. The bill was criticized for allowing too many concessions to private insurance companies in order to obtain passage. With this in mind, a good deal of doubt remains. As this study seeks to address the broader impact of poor provision of health benefits in the context of long-term AIDS care, this is an important final question to be considered.

According to a report from the Health Law and Policy Clinic of the Harvard Law School, the Patient Protection and Affordable Care Act will have a tangible effect on health care access for persons with HIV and AIDS.² One of the major improvements is expansion of existing HIV treatments under Medicaid. For instance, it eliminates the disability requirement and raises the maximum income requirements on claiming some Medicaid benefits, so that even persons up to 400% poverty may qualify for AIDS medications under public insurance. This effectively raises the poverty line under which persons can claim AIDS drugs under Part D requirements. This is important, as institute on Health Policy Studies at U.C. San Francisco shows that low-income persons living with HIV/AIDS more readily access medications via Medicaid than they do through public health clinics (Kahn et al. 2002).

The Health Care Reform Act also eliminates many of the pre-existing condition exclusions and provides broader access to health care. Lifetime limits on coverage will be reduced and patients will have access to increased palliative care, mental health services, substance abuse treatment, and management of other chronic diseases, or “co-morbidities.” As we pointed out in Chapter Five, many of the barriers to treatment among the impoverished are due to these contributing factors like mental health issues, drugs, or other health problems. Overall, the new law is intended to reduce some of the disparities in health access that particularly affect the poor.

²Information can be accessed from the website sponsored by the Harvard law school’s healthcare policy studies branch at http://www.taepusa.org/HealthCareReform.
This is even more important in states like Texas. Much of the data on HIV and AIDS used in this dissertation comes from Texas and, not surprisingly, it is becoming apparent that persons with HIV and AIDS in Texas will be even more disadvantaged with the passage of the 2011 state budget. It is estimated that the Texas HIV Medication Program (THMP), which is a joint state-federal ADAP provider, will be $20 million short of meeting demand for medications among the Texas poor. It is expected that the Health Care Reform Act will plug some of those holes once provisions are enacted.

7.4. Recommendations for Going Forward

How do we work to improve AIDS service provision? We need to start over and question the assumptions behind the HIV-AIDS theory. Researchers should not dismiss out-of-hand the objections of the so-called AIDS dissenters, but should bring them back into the discussion. This need not entail abandoning efforts at prophylaxis, which in themselves are valuable. However, we need to admit that we do not have the answer; much more research is needed, and a fresh approach, if we are going to solve the mysteries behind HIV and AIDS.

One immediate conclusion of this study is that little progress is likely to occur until there is a serious effort to ameliorate poverty. Work on this front is underway, but new studies are needed to enhance our knowledge of the ways chronic illnesses like AIDS are connected to long-term poverty. These studies serve two purposes: they raise our awareness of poverty in general, and they force us to look at the needs of the underserved. Shi and Stevens (2005) note that health outcomes across the board are improved when federal and community services aim at dealing not only with individual diseases, but also with general health inequities.

Second, CDC needs to establish clear national testing and reporting validation standards. Experts like Bauer (2007) point out that the ELISA test used for initial diagnosis of HIV is notoriously inaccurate. False positives can show up on the order of hundreds per ten
thousand and, as other researchers have noted, tests for HIV antibodies can cross-react with antibodies to other diseases like hemophilia, hepatitis, leprosy, certain cancers and, most notably, tuberculosis (Johnson 1996). Without clear standards mandating repeat and confirmatory tests like the Western Blot, there is no way to ensure that the numbers are accurate. And, with funding to states and localities depending as it does on numbers (see Chapter Five), there is less incentive to prove that the reported numbers are accurate.

Awareness must also be raised as to the questionable connections between HIV and AIDS. There is a huge body of literature, some of which is noted in this chapter, concerned with secondary explanations for the morbidity of AIDS-defining diseases. Yet, AIDS is so heavily politicized that few have even heard of these connections beyond the small group of specialists publishing in niche journals. Any attempt in the general literature to point out these pockets of literature is met with skepticism and even harsh criticism. Any attempt to depart from the accepted paradigm is called an “oddity,” as Weiss and Jaffe (1990) have characterized Professor Peter Deusberg and his well-publicized attack on the HIV/AIDS hypothesis. At worst, dissenters are called “AIDS denialists,” seemingly placing them on a par with holocaust deniers. Kary Mullis has been the subject of this type of derision. He is the Nobel Prize winning chemist who invented the polymerase chain reaction (PCR) technique used to detect HIV antibodies. Mullis is also skeptical of the idea that HIV is the sole causative agent of AIDS (Mullis 1998). This dissent has caused some to call Mullis a “pseudo-scientist” who espouses strange, and even dangerous, ideas (Nattrass 2007).

Ultimately, we owe it to the victims of AIDS. If, as the data tell us, there is little correlation between HIV and AIDS absent poverty, then people are being dealt a death sentence every day, and choices about their future are taken away from them in the name of public health. Further, these judgments about public health are likely based on assumptions not supported by the evidence. The focus needs to be shifted away from blame, risk, and fear, and back
toward good science and good policy. Some of the literature outlined briefly in the concluding remarks below suggests a direct biological link between poor health and AIDS.

7.5. Concluding Remarks

If theory predicts something, but we observe something else, we are left with an interesting puzzle. Is the theory wrong or have we left something out? There may be other explanations that are currently being overlooked because of the highly political nature of this disease. This is precisely the point. There can be little doubt that HIV and AIDS are linked. Viral load of HIV is highly predictive of disease progression from chronic HIV to acute AIDS. There is good reason, however, to question the exact nature of this relationship. A good deal of literature is devoted to the effects of chemical messengers called cytokines on the human immune system. Researchers have noted that a cytokine called tumor necrosis factor (TNF) enhances HIV replication, and that both TNF and interleukins can cause imbalances in cell populations with and without HIV (Matsuyama, Yoshiyama & Hammamoto 1989, Unanue & Allen 1987, Harada, Koyanagi & Yamamoto 1985, Matsuyama, Hammamoto & Yoshida 1988). Excessive production of TNF is known to cause auto-immunity and is the central cause of rheumatoid arthritis, an immune disorder. It may be the case that the central underlying mechanism of AIDS-defining disease is a state of systemic immune imbalance that accompanies, or even precedes, susceptibility to HIV. If this is the case, then it will prove much more fruitful in the future to heed Clerici’s advice and pursue a science of cytokinomics rather than a more-or-less single minded pursuit focused on HIV (Clerici 2010).

Several researchers including Kary Mullis, the inventor of the PCR technique used to measure viral load of HIV, have suggested that HIV is generally not deadly at all, but probably endogenous. According to Mullis, the human body is populated by an “uncountable” number of retroviruses. They are everywhere and inserted at various places in our genome. So, at various times, humans cells do produce their own retroviral particles, many of which do
resemble HIV (Mullis 1998, 178). There is little evidence, however, that they are deadly. Bauer (2007) and Deusberg (1996) make the same argument. Deusberg, one of the pioneers of retrovirology in the 1970’s and Bauer a prominent chemist, insist that cofactors must be present before the immune system can be crippled in ways characteristic of advanced AIDS disease. Certainly poverty, drug use, malnutrition, and poor living conditions can contribute to immune system disorder.

We are certainly not at a point where we can declare a direct relationship between the types of immune dysfunction in AIDS and generalized autoimmune disorders. However, the research highlighted above suggests a solid link between AIDS-defining disease and the cytokine disorders which characterize autoimmunity. It is also suggestive of a secondary or indirect role for HIV in the onset of AIDS-defining diseases. Yet, despite this impressive body of research, scientists do not, or cannot, speak openly about perceptions of the reduced capacity of HIV to cripple the immune system. Epstein (1996) argues that a power structure dominates discussions about the causative relationship between HIV and AIDS. Scientists have invested heavily, both psychologically and in terms of time and resources, in the dominant view of AIDS. Questioning the paradigm would require admitting to at least partially erroneous assumptions.

When a paradigm is questioned, scientists and social scientists alike frequently remember the arguments of Thomas S. Kuhn (1970). We must be cognizant, as Kuhn tells us, that science is a human enterprise and thus subject to a host of social constructions and assumptions. Among these is a tendency to establish specialized journals and societies that cement a single discipline’s hold over a technical and esoteric knowledge. Further, when the promise of scientific advance is coupled with the promise of a social good, Sarewitz (1996) argues, a mythology develops which places science in a position of considerable political advantage. This myth simplifies the complex and holds out scientific research as the vehicle for solving complex social problems. Stretching a scientific paradigm beyond its limitations, however,
only serves to reify the myth beyond what is tenable. And, as Epstein (1996) contends, a challenge to the politics of power surrounding the AIDS community is needed in order for a useful dialogue to be established.

AIDS is an example of such an area of expertise. It is, arguably, the world’s first geopolitical disease. As such, it is prone to the influence not only of scientists, but policymakers, international organizations, and national governments, as well. Yet, despite all of these efforts, AIDS continues to pose a significant threat, particularly in the developing world and among the poor (Poku 2005). Perhaps the unavoidable conclusion to be reached is that we simply do not know enough about AIDS or the human immune system itself to postulate a direct role for HIV in the complex of AIDS-related infections without other contributing factors. This study has argued that there is much work to be done.

Throughout this dissertation, I have reviewed the literature on poverty and disease and shown that the roots of a challenge to the paradigm are already in place. If the scientific community, however, is unwilling or unable to urge a reconsideration of the dominant paradigm, it is impinging upon social science to do so. This study has argued for an integrated approach to not only the study of social factors and their relation to immune disease, but also to the integration of knowledge that separates the biological and social sciences.
APPENDIX A

LIST OF METRO AREAS AND AIDS PREVALENCE
<table>
<thead>
<tr>
<th>Metro Area</th>
<th>AIDS per capita</th>
<th>Cumulative Cases</th>
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<td>Population</td>
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<td>Washington, DC-VA-MD-WV</td>
<td>30.5</td>
<td>32,809</td>
</tr>
<tr>
<td>Wichita, KS</td>
<td>5</td>
<td>882</td>
</tr>
<tr>
<td>Worcester, MA</td>
<td>7.9</td>
<td>1,861</td>
</tr>
<tr>
<td>Youngstown-Warren-Boardman, OH-PA</td>
<td>9.5</td>
<td>576</td>
</tr>
<tr>
<td>Metropolitan areas with 500,000 or more population</td>
<td>15.6</td>
<td>871,192</td>
</tr>
<tr>
<td>Metropolitan areas with 50,000 to 499,999 population</td>
<td>7.7</td>
<td>97,658</td>
</tr>
<tr>
<td>Nonmetropolitan areas</td>
<td>5.1</td>
<td>57,604</td>
</tr>
</tbody>
</table>

Total (including persons of unknown residence) 12.5 1,028,991
APPENDIX B

WEB ADDRESSES FOR DATA AND REPORTS
CDC Reports

“CDC Study Finds U.S. Herpes Rates Remain High”
http://www.cdc.gov/nchhstp/newsroom/hsv2pressrelease.html

“New Studies Explore Reasons for Heavy STD Burden Among Women and Minorities”
http://www.cdc.gov/nchhstp/newsroom/stddisparitiessummary.html

Census Bureau Reports

Civil Rights Leadership Conference Reports on Federal, State, and Local HIV/AIDS Services
http://www.civilrights.org/census/your-community/funding.html

Census Bureau’s Small Area Health Insurance Estimates (SAHIE) for Counties and States

Institute on Assets and Social Policy

“The Racial Wealth Gap Increases Fourfold”
“Diseases of Poverty and the 10/90 Gap”
http://www.fightingdiseases.org/pdf/

UN Habitat and World Health Organization Report

“Hidden Cities: Unmasking and Overcoming Health Inequities in Urban Settings”
http://www.hiddencities.org/report.html

U.S. Poverty Estimates by State and County
http://poverty.us/Texas-Poverty.html

World Health Organization HIV/AIDS Prevalence Estimates
www.globalhealthfacts.org
BIBLIOGRAPHY


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Hober, D., A. Haque, P. Wattre & G. Beaucaire. 1989. “Production of Tumor Necrosis Factor Alpha (TNF-α) and Interleukin 1 (IL-1) in Patients with AIDS. Enhanced level of TNF-α is Related to a Higher Cytotoxic Activity.” Clinical and Experimental Immunology 78(2):329–33.


